# Public Understanding of Science

The role of genes in talking about overweight: An analysis of discourse on genetics, overweight and health risks in relation to nutrigenomics Rixt Komduur and Hedwig te Molder Public Understanding of Science 2014 23: 886 originally published online 28 January 2013 DOI: 10.1177/0963662512472159

> The online version of this article can be found at: http://pus.sagepub.com/content/23/8/886

> > Published by: SAGE http://www.sagepublications.com

Additional services and information for Public Understanding of Science can be found at:

Email Alerts: http://pus.sagepub.com/cgi/alerts

Subscriptions: http://pus.sagepub.com/subscriptions

Reprints: http://www.sagepub.com/journalsReprints.nav

Permissions: http://www.sagepub.com/journalsPermissions.nav

Citations: http://pus.sagepub.com/content/23/8/886.refs.html

>> Version of Record - Oct 27, 2014

OnlineFirst Version of Record - Feb 1, 2013

OnlineFirst Version of Record - Jan 28, 2013

What is This?



Public Understanding of Science 2014, Vol. 23(8) 886–902 © The Author(s) 2013 Reprints and permissions: sagepub.co.uk/journalsPermissions.nav DOI: 10.1177/0963662512472159 pus.sagepub.com



# Rixt Komduur

Wageningen University, The Netherlands

Hedwig te Molder

Wageningen University, The Netherlands

#### Abstract

This study examines whether the assumptions embedded in nutrigenomics, especially the alleged relation between information about personal health risks and healthy behaviour, match how people account for the relation between food, health and genes in everyday life. We draw on discourse analysis to study accounts of overweight in six group interviews with people who are and who are not overweight. The results show potentially contradictory normative orientations towards behavioural explanations of (over)weight. *Overt* gene accounts are interactionally problematic (in contrast to more indirect accounts such as 'build'), indicating that participants treat 'behaviour' as the normatively appropriate explanation for overweight. At the same time, however, healthy behaviour is an *accountable* matter, i.e. it is dealt with in interaction as behaviour that is not self-evidently right but requires an explanation. It is discussed how bringing these interactional concerns to the surface is essential for understanding future users' response to nutrigenomics and emergent technologies more in general.

#### **Keywords**

discourse analysis, genetics, overweight, nutrigenomics, technology assessment

## I. Introduction

New technologies are envisioned and produced in ways that embed certain assumptions, for example with regard to future users' competencies and ideal usages of the proposed innovations (Akrich, 1992). To take the role of users into account, programmes from a constructive or real-time

**Corresponding author:** 

Rixt Komduur, Wageningen University, Hollandseweg 1, Wageningen, 6706 KN, The Netherlands. Email: Rixt.Komduur@gmail.com Technology Assessment perspective (Guston and Sarewitz, 2002; Oudshoorn et al., 2004) aim at integrating developments in natural science and engineering with insights from social science from the outset (Schot and Rip, 1997). The presumption is that early user-involvement in technology development furthers the domestication of technologies (Silverstone and Hirsch, 1992), i.e. the incorporation of technology into the everyday lives of users. Scenarios, questionnaires, qualitative interviews and observation methods provide for anticipated evaluations of a future technology or evaluations of already developed or partially developed technologies in different user contexts. But future technologies do not allow for easy assessment. Research into participants' needs, expectations, and desires regarding future products is hampered by the fact that these needs are often not clear-cut and readily available. Even the use of prototype applications of the technology starts from the assumption that an individual knows or is able to predict his or her needs and desires, thus requiring an imagination, commitment or investment that is often not there – at least not yet.

This article focuses on nutrigenomics-related technologies and therapies as a high-profile and rapidly developing research area on the relationship between health, food and genetic differences. Our presumption is that new technologies interact with everyday problems and concerns that, to a large extent, already exist but often have gone unnoticed, including by (future) users themselves. These concerns are normally not technology imbued or, rather, will predominantly not be constructed as such by users; but they may be a decisive factor in whether or not technologies in a particular environment will germinate. A major advantage of analysing current concerns is that they are built up from automatic and necessary involvement, and therefore they do not allow for the kind of detachment and aloofness that characterise the reflection on future worries and desires. Moreover, our approach enables us to map accounts and concerns that are typically so self-evident and implicit that they are overlooked.

The aim of this study is to help gain insight into the extent to which there is a match or clash between assumptions incorporated into the technology and socio-interactional practices embedded in everyday life. In the present context of nutrigenomics-related technologies, we focus on how participants account for problems in the domains that these technologies supposedly affect, namely issues of overweight, the role of genetic susceptibility therein, and participants' ways of coping with lifestyle-related health risks more generally. More specifically, we examine the different interactive goals that these accounts may work to accomplish, consciously or not, such as managing cause and responsibility regarding overweight and overweight-related disease, and establishing particular identities in relation to issues of health and health risks.

Bringing these broader interactional concerns to the surface is essential for understanding (potential) future users' response to new technologies (Veen et al., 2011a; cf. te Molder and Potter, 2005). The results may help to translate differences and similarities into relevant technology characteristics and also help users to articulate their needs and expectations towards future technological developments once their everyday concerns have been made explicit. Our starting point is that technologies need to find 'points of reference' in daily life if they are to be successful at all.

#### Nutrigenomics

Nutrigenomics studies the interaction between nutrients and genes and aims to develop diagnostic tools that can detect (pre-)diseases in a still reversible stage or reveal genetic susceptibilities for lifestyle-related diseases (Barraso, 2005; Dembinska-Kiec, 2008; Johnson et al., 2006; Ordovas and Corella, 2004). Developments in nutrigenomics parallel, and have stimulated the ambitions for, a so-called personalised diet, that is, the tailoring of nutrition to individual needs (Castle and Ries, 2009). Although these early aspirations have been tempered, for example in the direction of

group rather than individual profiles, the promise of a genetically based differential dietary advice remains. Developments in nutrigenomics go hand-in-hand with the increasing importance of health in modern society, the attribution of personal responsibility for one's health (cf. Crawford, 2006), and emergent preventative medicine (Harvey, 2009). We look at the emerging field of nutrigenomics as an example of how genomics may influence everyday life at the intersection of health, risk and genetics, for example through functional food products and personalised nutritional advice, e.g. on the basis of tests for the genetic susceptibility for overweight (Castle and Ries, 2009).

As with many emerging disciplines and technologies, nutrigenomics is controversial. Supporters of nutrigenomics presuppose a match between nutrigenomics applications and already existing ideas and use of knowledge about obesity, health, lifestyle and food in people's everyday lives. Critics, however, are not so certain that such a match really exists. Some argue that the presentation of eating and healthy living in terms of risk management and disease prevention that nutrigenomics presupposes may not be feasible or desirable; it may not square with notions of eating and healthy living that people have or aim for (Komduur et al., 2009). Others question the efficacy of nutrigenomics information for healthy behaviour. They wonder whether the idea of empowerment brought by providing genetic risk information entails too simplistic a notion of change in human behaviour (Bouwman et al., 2008).

The aim of this study is to find out whether the assumptions embedded in future nutrigenomics tools, especially the alleged relation between information about personal health risks and healthy behaviour, match with how people account for the relation between food, health and genetic differences in everyday life.

## Effects of genetic risk information on healthy behaviour and lifestyle

A handful of studies have been done on the effects of giving people genetic risk information on obesity or other lifestyle-related diseases. Although these studies suggest that people are better motivated to adopt a healthy lifestyle after receiving genetic information on increased risks for lifestyle-related diseases, no proof has been found that this information actually changes people's behaviour.

Regarding obesity, there are no indications to suggest that providing genetic risk information to obese people has psychological disadvantages (Harvey-Berino et al., 2001; Hilbert et al., 2009; Rief et al., 2007; Segal et al., 2007). Positive effects on behaviour could not be measured either (Hilbert et al., 2009; Rief et al., 2007), although there are signs that a higher genetic risk for obesity increased people's motivation to live healthily (Frosch et al., 2005).

Similar results have been found in research on the effects of genetic testing in relation to other lifestyle-related diseases. Genetic information about lung cancer or heart disease (Sanderson and Michie, 2007; Sanderson and Wardle, 2005) and colorectal cancer increased people's motivation to adopt a healthy behaviour (Lucke et al., 2008).

With respect to behavioural change however, the results are mixed. People who received genetic test results indicating a higher risk for thrombophilia, for example, reported that the genetic information had not changed their lifestyles (Saukko et al., 2006). Another study showed that, after a genetic test for lung cancer in both an increased and decreased risk group, both groups showed participants who quitted smoking (Sanderson et al., 2008). It seemed that it was not the genetic risk information itself but rather the procedure followed that was responsible for the reported effects.

These results show that the relation between genetic risk information and healthful behaviour is at best indirect. One of the questions that can be raised is the extent to which the information offered connects with people's everyday realities. A closer look at the understanding of genetic influence shows it to be a mixture of everyday knowledge about heredity and scientific insights. New scientific knowledge about genetics is integrated into already existing 'lay' knowledge and beliefs about inheritance (Emslie et al., 2003). Lock et al. (2006) showed a similar pattern for risk perceptions: relatives of people with Late-Onset Alzheimer's Disease (LOAD) picked out a relative who resembled the one with LOAD, e.g. in terms of looks or behaviour, as the one who was most likely to be at risk of the disease. The latter results indicate the importance of not restricting research to overt 'gene discourse' but also looking at accounts that are more indirectly linked to possible hereditary characteristics, such as participants' orientations to physical appearance.

#### Gene discourse

Discourse studies on generic genetic influences on health and illness show that the notion of genes is predominantly used to debate the moral responsibility for being ill (Crossley, 2002; cf. Van Exel et al., 2006). Patients with Family Hypercholesterolemia were found to draw upon gene talk so as to manage feelings of guilt and shame (Frich et al., 2007). Crossley's (2002) focus group study shows that participants use 'genetic predisposition' to highlight the ultimate lack of control over health when individual responsibility had been given much emphasis in the discussion. In her analysis of a Finnish internet discussion, Kokkonen (2009) points out, however, that genetic explanations are not automatically accepted. Forum participants tended to construct the parents of obese children as the primary cause of their child's fatness.

The same ambivalence is found in dealing with health risks more generally. Although selfresponsibility and discipline are deemed important, the importance of pleasure, and insurgence against control and regulation of health, are also emphasised (Crossley, 2002; Pajari et al., 2006). As Hodgetts et al. (2005) show, people relatively easily resist health messages in the media by constructing other notions of health, such as the need for a psychological balance in life through pleasurable unhealthy habits.

These studies demonstrate that people use different or even contradictory accounting practices when addressing the influence of genes in achieving health, and acting or not acting upon health risks more generally, but they pay hardly any attention to, or fail to address, the social actions that people establish in doing so. Therefore we propose to use the discourse analytic notion of an interpretative repertoire.

#### A discursive approach to health and genetic risks

The method adopted in this article is informed by a discourse analytic perspective as originally developed by Potter and Wetherell (1987). This perspective treats speech as constructed and constructive. Rather than simply reflecting the outside or mental world, talk is social practice, i.e. it is drawn upon to accomplish different social actions such as accusing, defending, building expertise, complaining and complimenting. The action-oriented approach to language makes this form of discourse analysis radically different from cognitivist traditions in psychology that treat mental states as the *source or cause* of what is being said (for a debate on the status of cognition in interaction analysis, see te Molder and Potter, 2005).

The perspective focuses on the ways speakers construct different versions of the world as they move across various interactional situations. The concept of *interpretative repertoire* is used to describe these situated descriptions. More specifically, it represents a vocabulary or register of terms and metaphors that people draw upon to characterise and evaluate actions and events (Lawes, 1999; Potter and Wetherell, 1987: 138).

On the basis of group interview data, we first show that both overweight and non-overweight participants display reluctance to use or accept overt genetic explanations for being overweight and rather attribute it to 'build'. Part of the interactional business performed with these accounts differs, however, for the overweight and non-overweight groups. Second, we show that participants orient to a healthy lifestyle as *accountable*, i.e. as behaviour that is not self-evidently right but requires an account such as family history or health tests.

The results thus reveal an interesting ambivalence in the talk: while participants treat behavioural (rather than genetic) explanations for overweight as normatively appropriate – for example by providing accounts once their talk diverts from a behavioural explanation – they strikingly attend to a healthy lifestyle (in terms of risk prevention) as accountable too.

# 2. Materials and method

We organised a total of six group discussions with people who were selected through a recruiting agency that informed them that the topic of discussion concerned overweight and that the study could help find new ways of giving advice on nutrition. Self-reported body mass index<sup>1</sup> (BMI) was a selection criterion. Because of possible differences in explaining one's own weight and the weight of others, three groups of people with a BMI above 28 and three groups with a BMI below 25 were selected. Second, to take into account possible genetic explanations that involved family members, both the overweight participants and the normal weight participants had at least one close relative who was overweight. Third, to explore the issue of parental responsibility for child health, people with children were recruited, leading to a total of 22 parents with children from 4 to 40 years of age. Finally, a sample with a wide age range (between 24 and 68 years old) and diverse socio-economic and ethnic backgrounds was collected and divided into groups with a mix of these characteristics. The participants were reimbursed for their travel expenses and received an hono-rarium of €35. To protect the identities of the participants, all names and other information were disguised.

First, the interviews were analysed by studying the role and purposes of accounts that suggested genetic susceptibility to being overweight, e.g. constructions that described or implied unalterable components in the body that make people vulnerable to overweight. Second, the role of health risks in relation to accounts of healthy eating, living, and overweight were studied by focusing on accounts in which risks, diseases, and disease-related complaints were presented. The interviews were organised in such a way as to allow informal conversational exchanges between participants. In the analysis, all contributions including those of the facilitator/interviewer were examined (cf. Lawes, 1999). The analytic levers to identify the repertoires were as follows:

- variability of descriptions: usage of different variants of the same concepts is known to signal different interactional goals;
- rhetorical character of the talk: reports from the participants automatically resist other possible constructions of the world. By reflecting on these other possibilities, the analyst gains insight into the conversational issues that are handled by the participants in present versions;
- participant uptake: looking at how the participants (including the facilitator) treat each other's talk, what they make relevant, and to what interactional ends.

The study involved a semi-structured group interview about a broad range of topics concerning overweight in society and people's daily lives, and possible causes and consequences of being overweight. Organising the discussions in a manner that allowed informal conversational exchange helped us to take interaction into account and to allow for new issues to be raised by the participants. The interviews were facilitated by the first author of this article and were held in the Netherlands. They were tape-recorded, video-taped, and transcribed to word-level accuracy, including speech errors, pauses, and overlap in talk.<sup>2</sup> Relevant fragments were translated into English by a native speaker, after having been informed by the analysis that was performed in Dutch<sup>3</sup>.

# 3. Results

We start this section with the finding that participants used two different repertoires to negotiate the role of responsibilities played by individuals in relation to their overweight, namely:

- The 'In the genes' repertoire: accounts that literally used the words 'genes' or 'genetic susceptibility';
- The 'Build' repertoire: accounts that referred to body composition.

Subsequently we show how healthy behaviour more generally is both being downgraded and accounted for by reference to family history and health checks.

# The 'In the genes' repertoire: An interactionally risky account for overweight

We labelled sets of accounts in which participants used the word 'gene(s)', or referred to hereditary factors in an explicit way, as the 'In the genes' repertoire. Like in other studies (Crossley, 2002), participants used repertoires about genes to mitigate responsibility for their overweight in the overweight groups, and for overweight in general in both groups. However, contrary to what was expected (Crossley, 2002; cf. van Exel et al., 2006), in both groups the use of 'genes' for this purpose was treated as problematic. That is, people accounted and needed to account for the fact that they used gene-related explanations. Extracts 1 and 2 illustrate this:

Extract 1	(overweight – Interview	Ι. <i>b</i> .	10.11.	10-31)
Excluded 1	i i i i i i i i i i i i i i i i i i i	· , p.	,	

1	Rose:	For some people it's just in the genes. Yeah,
2		I mean on my mother's side of the family
3		every, everybody is too heavy. I wish I got
4		my father's genes. Everybody on my father's
5		side is very thin.
6	Interv:	Hm
7	Rose:	But yes, I have my mother's genes (laughs).
8		So. Yeah, no um Yeah. Th, that's indeed just
9		the way it is. Yeah, I've, Yeah at a certain
10		point, um. I've dieted a lot. Yeah, the yoyo
11		uhm effect then.
12	Interv:	Yes
13	Rose:	Yeah. (.)
14	John:	[It's just]
15	Rose:	[It's just] eating=

- 16 John: It's eating and it can also be predisposition
- 17 Rose: It's also predisposition=
- 18 John: =It can also be predisposition

Extract 1 demonstrates the kind of resistance that the gene repertoire evokes when it is used to account for overweight. First, it was used after a whole range of other explanations for overweight had been discussed, such as too little physical activity and eating too much. This demonstrates that the participants were reluctant to use it. Second, while drawing on the 'In the genes' repertoire, attempts were made to normalise the fact that genes are indeed involved in being overweight. Rose starts with a general remark that for some people it is 'just' in the genes (line 1). 'Just' is a direct translation of the Dutch word 'gewoon' and it can be used to make something sound normal, e.g. 'just' a cold instead of a more serious disease (Lee, 1987, 1991; Verkuyten, 2003). 'Just' in this context thus emphasises that genes are a self-evident explanation of overweight that is obvious and beyond discussion (cf. Verkuyten, 2003). In this way, possible counter explanations are resisted. Third, accounts about genes were offered and treated as requiring specific evidence. In this case, Rose supports her claim by describing that everybody on her mother's side is heavy (lines 2-3). Finally, participants with overweight introduced the gene accounts with jokes or laughter. In line 7 Rose laughs and makes a joke about her own bad luck, thereby distancing herself from a genetic explanation while introducing it (cf. Barnes et al., 2001 on laughter). Note as well, in lines 16–18, that predisposition as a cause is constructed by Rose as something that (also) is, whereas susceptibility is rearticulated by John as a cause that can be, again displaying the reluctance to treat susceptibility as a legitimate account for overweight.

Extract 2 is another example of the 'In the genes' repertoire used to account for overweight.

#### Extract 2 (overweight – Interview 2, p. 8, l. 37–p. 9, l. 22)

1	Ellen:	Well actually I agree with with them with
2		them. But it's also a little bit in the genes
3		with us as well. My mother is as well. She's
4		eighty-five, but she's still just like me. The- I
5		am so hefty. And well yes the rest is also fat
6		of course.
7	Interv:	(Laughs) (more laughter)
8	Ellen:	I don't mind that you know that.
9	Ralf:	That is why you you [unclear]
10	Ellen:	[It's just so] totally
11		hefty. big boned.
12	Interv:	Yes.
13	Ellen:	heavy too. Just heavy.
14	Interv:	Yes. (.) So predisposition.
15	Ellen:	Predisposition. Yes.
16	Jorien:	Yes, but I also think that if you've had
17		children.
18		(eight lines omitted)
19	Ralf:	Psychological [they say then]
20	Ellen:	[No I've] actually always had
21		it actually. 'v always been. Sometimes I say
22		I just skipped size thirty six.

As with Extract 1, the 'In the genes' repertoire is the last account drawn upon to explain overweight. Ellen supports her claim by describing her mother's physical appearance (lines 3–4) and introduces her account with laughter and a joke (lines 5–6). Moreover she uses the word 'just' (lines 10 and 13) to emphasise the obviousness of her overweight, and its genetic nature, which is subsequently formulated by the interviewer (line 14). Note how the participants resist Ellen's account, for example, by describing the influence of having children (lines 16–17) and by characterising her explanation as psychological (line 19), which is then again undermined by Ellen's suggestion that she has always been heavy (lines 20–21), without, however, giving an overt reference to her genetic background.

The problematic character of genetic explanations for overweight is also clearly illustrated by the way in which the non-overweight groups treated gene accounts, namely as self-serving and subjectively motivated rather than just true. Dispositional explanations for being overweight are characterised as easy excuses:

#### Extract 3 (non-overweight – Interview 5, p. 17, ll. 39–49)

1	Anneke:	It is easily used as a cover (1)
2	Johan:	Yes
3	Anneke:	I'm predisposed. But there are of course
4		people, but they are mostly, (1) they have
5		uhm problems with something (2)
6		(three lines omitted)
7	Anneke:	But yes, of course it's easy (1) to say (1) I'm
8		uhm (2) predisposed to it.

Anneke treats genetic explanations for overweight as suspiciously motivated (line 1). Interestingly, she presents herself as predisposed to being overweight although she is part of the non-overweight group. This suggests that *despite* her predisposition she is able to remain thin: she has not been using it as an excuse. Thinness is thereby constructed as a personal merit.

Our results show that although the 'In the genes' repertoire is used to negotiate reduced personal responsibility for being overweight: in both the non-overweight groups and the overweight groups it is treated as a problematic and personally motivated explanation. Overweight people were reluctant to use 'genes' as an explanation for their overweight and their fellow group participants resisted gene accounts. Non-overweight groups treated genetic explanations as motivated, i.e. as believing what it suits one to believe. This uptake suggests a normative orientation to behavioural explanations for overweight in both types of group.

#### The 'Build' repertoire: Constructing evidence of predisposition by visible thinness

Descriptions of body size were regularly constructed in relation to overweight so as to provide evidence of a genetic predisposition to overweight. We labelled these constructions the 'Build' repertoire. The 'Build' repertoire was often drawn upon after the 'In the gene' repertoire had been used – in the overweight groups for explaining one's own weight (see for example Extract 2, lines 10–13), and in both groups to account for overweight more generally. The latter use of the 'Build' repertoire mostly consisted of a comparison between thin and overweight children, so as to provide visible and thus neutral proof of the existence of a genetic component in weight.

Extracts 4 and 5 show how the 'Build' repertoire is used to give evidence of an unchangeable bodily component in relation to overweight in general:

#### Extract 4 (non-overweight – Interview 6, p. 22, ll. 20–23)

1	Paula:	I can see it sometimes very clearly in
2		families that um one, one of the children is
3		getting horribly fat
4	Jan:	[Yes yes yes
5	Paula:	And the other one stays [just fine
6	Bea:	[Yes
7	Jan:	Yes
8	Paula:	And then you don't know for sure, but you
9		can assume there are fairly similar ways of=
10	Jan:	=Yes=
11	Paula:	=How we deal with food in this family.

Paula presents the differences in body sizes of children in families as strong and clear evidence of the existence of susceptibilities for overweight. Jan immediately and straightforwardly accepts this description. Visible differences in body size are thus constructed and treated as unproblematic proof of the differences in predisposition between children. Extract 5 shows a similar pattern for the overweight group:

#### Extract 5 (overweight – Interview 4, p. 5, l. 33–p. 6, l. 2)

1	Ewald:	Uhm I uhm. I uhm have three girls as I just
2		said.
3	Interv:	hmhm
4	Ewald:	Uhm And the oldest and the youngest (.) are
5		super slim. Superuhm thin. That oldest one
6		eats all day long. (2) She doesn't put on a
7		gram, she just stays at her (1) And the
8		middle one we really have to cut back.
9		Because she can. She she's just getting big
10		as a house.
11		(four lines omitted)
12	Ewald:	You can see very clearly the difference (1)
13		between the one child and the other child.
14	Interv:	Yes
15	Merel:	Yes very apparent

Ewald proves the existence of an unchangeable bodily component by describing the thin appearances of his oldest and youngest daughters, and comparing their appearances with the tendency of his middle daughter to gain weight. In lines 12 to 13 he connects the visible differences to a difference in susceptibility of his daughter for becoming overweight. Merel readily accepts this, i.e. without hesitation, as straightforward and neutral proof. After describing how he prevents his second daughter from putting on weight, Ewald presents his oldest thin daughter as a person who eats the whole day and eats unhealthy foodstuffs (e.g. bags of chips disappear):

#### Extract 6 (overweight – Interview 4, p. 6, ll. 35–40)

- 1 Ewald: Because the oldest one she stuffs herself all
- 2 day long. She she can eat bags uhm uhm
- 3 disappear. Chips it doesn't matter what.
- 4 Interv: hm hm
- 5 Ewald: She doesn't put on a gram.

By constructing a contrast between his one daughter who needs to be 'cut back' and the older daughter who eats constantly without putting on a gram, he presents his older daughter as someone who is more or less resistant to becoming overweight and therefore needs no such guidance.

Second, in both sub-groups a build that indicated thinness was used to make available explanations about parents taking responsibility for the eating habits of their children. In Extract 7 the link between build and resistance to becoming overweight is constructed as to account for possible unhealthy eating habits in the family:

#### Extract 7 (overweight – Interview 2, p. 19, I. 44–p. 20, I. 21)

1	Ellen:	Quickly eats a pizza, or quickly eats some
2		French fries or quickly eats a small
3		something.
4	Peter:	Yes
5	Ellen:	And that's also that that's also a big
6		[cause you know]
7	Peter	[We try t-] We try to limit that as much as
8		possible. We do it sometimes
9		(ten lines omitted)
0	Peter:	No but that uhm.(2) They also don't have
1		that build at all. (2)
2	Ellen:	No no

Peter uses the 'Build' repertoire in relation to his children (lines 10–11) after Ellen has claimed that people often eat unhealthily because of a lack of time. Ellen's explanation invites an account from Peter that he and his family are careful not to eat too much. 'They also don't have that build at all' (lines 10–11) suggests a low chance of becoming overweight anyway because of their physique.

Extract 8 shows another example of how people draw on the 'Build' repertoire to account for the eating behaviour of their children:

#### Extract 8 (non-overweight – Interview 5, p. 5, ll. 12–24)

1	Johan:	But yes right, what is checking one's
2		children?
3		(Everyone speaks at the same time)

4	Tessa:	Well, as long as you still have a lot of
5		influence as [as a mother]
6	Johan:	[Yes but] yes right you have
7		some influence on uhm=
8	Tessa:	=But [(.)but] that wouldn't be right to to
9		check those things
10		[continually checking with that lot (laughs)]
11	Johan:	[And I mean, they look fine] the two of
12		them, and then I don't have a problem if
13		they get a uhm some chips or something in
14		the evening
15	Anna:	No of course not

Here Johan and Tessa negotiate about the extent to which parents can be held responsible for the behaviour of their children. Johan resists Tessa's idea that it is possible to monitor the dietary habits of your children to a great extent. Additionally, he draws on the 'Build' repertoire to account for the fact that he approves of his daughters' 'unhealthy' habits. Anna's reaction – 'No of course not' (line 15) – suggests that Johan's 'Build' explanation is acceptable and obvious.

Participants in both the overweight and non-overweight groups designed and received the 'Build' repertoire as visible and neutral proof of the possibility of being predisposed to overweight. The 'Build' mostly contained a comparison between lean and overweight children. Moreover, a version of the 'Build' repertoire – where a thin appearance was referred to – was used to make available explanations about taking or not taking responsibility for the diet of one's children. Thereby constructions of the children's physique were used in a black-and-white manner: either the child had the 'build' that made her/him susceptible to overweight or s/he had not. In the case of overweight children, the 'Build' repertoire worked as a parent's account for taking responsibility and making them eat less, and in the case of slim children, as a powerful account for allowing them to eat 'anything', including unhealthy foods.

Hepburn and Wiggins (2005) point out that body size is a compelling account to give evidence. Their research on helpline conversations in which child neglect was reported showed that a thin appearance was treated as visible and neutral proof of not feeding the child enough rather than a prejudiced or paranoid vision of the caller. Possibly, in relation to overweight, the 'Build' repertoire has this same aura of visibility and therefore the objectivity needed for a strong account for the child's eating habits and the parents' responsibility in this.

#### Healthy behaviour as a form of risk prevention: An accountable phenomenon

As we have just seen, participants in both groups displayed a *normative* orientation to behavioural explanations of overweight. This normative preference could be seen in the reluctant receipt of the explicit genetic version of overweight by the 'In the genes' repertoire, and the affiliative receipts of less overt versions of a genetic influence in the 'Build' repertoire. Moreover, in the 'Build' repertoire overweight people were personally held accountable for their weight (increase).

Despite this normative orientation towards overweight as caused by behaviour rather than genes, participants provided accounts for any proactive behaviour concerning the management of their health.

Extract 9 shows how the need to be health conscious in terms of risk prevention is immediately resisted by Joris. His father, on the other hand, is allowed to be health conscious:

#### Extract 9 (overweight – Interview 1, p. 33, ll. 19–40)

1	Joris:	I'm not even thirty surely I'm not going to
2		think about health risks
3		(two lines omitted)
4		I uhm if I look at my father indeed he is well
5		indeed (.) more on the- not not excessively
6		large or something.
7	Interv:	Hm
8	Joris:	But well that's also with a diabetes
9		diagnosis. And yes (1) then it's the case that
10		almost everything that he eats or does (.) Just
11		think about it for a minute and I mean that
12		he one day (1) you know you hear it and
13		you're shocked like gosh yeah how did that
14		happen. Well yeah that you think about it at
15		one point. Yeah, I don't have that
16		(five lines omitted)
17	Joris:	No (.) I think that my problems with my
18		weight are more like if I wake up in the
19		morning, or if I sit in a chair with a white
20		shirt on that I think like (.) Well that I find
21		worse than the health risks.

Joris resists the idea of thinking about health risks in relation to his own overweight. He uses a description of his father with diabetes to construct a contrasting situation in which it *would* be legitimate to think about health risks. Joris accounts for the fact that he, unlike his father, does not need to think about health risks because he is young and has no complaints that are directly related to overweight. He constructs his physical appearance as a more legitimate account to have a health-conscious diet.

In Extract 10, Maja presents a description of a medical situation to account for the fact that she worries about health risks.

#### Extract 10 (overweight - Interview 4, p. 67, II. 20-40)

1	Maja:	I have uhm two sisters with diabetes. And
2		my father has uhm Type 2 Diabetes. And
3		euhm I I then think like oh uhm it really runs
4		in the family. And I think yeah like being
5		overweight (1) I wasn't doing well with my
6		weight either. And that's also the reason,
7		what I said earlier, that I go to the doctor
8		every six months. And every time I think
9		like now, Maja, now it's going to be bad.
10		Now you're going to get it.
11		(four lines omitted)
12	Maja:	How can you be so stupid. Because it runs in
13		the family and then you en-en-encourage
14		overweight

15		(five lines omitted)
16	Maja:	When I get the same results again, that
17		everything is fine and you're in perfect
18		health (1) then I also push that very easily
19		away, you know?
20	Others:	Yes

Maja draws upon a family history of diabetes to account for a constant worry about her health. The description (lines 16–20) of the disappearance of these concerns after she has been found healthy, however, also carefully constructs a balance between being health conscious (for a reason), on the one hand, and being relaxed about it, on the other.

In the above excerpts, we have seen that participants treat healthy behaviour, especially in terms of risk prevention, as an accountable phenomenon. They showed themselves to be health conscious, but for a reason. Overall, the participants showed a concern with being relaxed about their health in terms of medical risks even if they were taking preventive measures.

# 4. Conclusion and discussion

In this study, we examined the extent to which the presuppositions embedded in future nutrigenomics tools, in particular tests for genetic susceptibility to overweight, matched with sociointeractional practices embedded in everyday life. More specifically we looked at how the notion that information about genetic susceptibility empowers people to live healthily is treated in current everyday talk, and to what extent the presentation of healthful living in terms of personal risk management connects with the way people account for this relation in everyday life.

First of all, the study demonstrated the interactional delicacy of drawing on overt genetic explanations for overweight in both the overweight and non-overweight groups, indicating a normative orientation towards *behavioural* explanations of overweight. The 'Build' repertoire, that suggests rather than claims a genetic component through descriptions of different physiques, was more easily accepted. This repertoire leaned on comparisons between thin and overweight children, thereby making available 'visible' evidence of a genetic predisposition concerning overweight (cf. Hepburn and Wiggins, 2005). The repertoire shows that an overweight person on his/ her own is not treated as sufficient 'proof' of genetic influences, whereas the comparison with an 'unsuspected' thin person – who is not supposed to have a stake in producing a genetic explanation – is. A similar asymmetry was found in relation to parental responsibility for children's eating habits in relation to their genetic susceptibility: whereas overweight children were held accountable for their eating behaviour, thin children were not.

This study draws on a small sample of Dutch participants and has not aimed at representativeness. The grounded indications that our analysis put forward could be, however, used to inform a larger study with a more diverse set of participants. The indirect focus on behavioural explanations as the norm and the related treatment of gene explanations as implying a denial of personal responsibility for one's overweight shows the extent to which gene accounts are still connected with attributions of responsibility and blame, and the need for self-discipline. In contrast to e.g. Crossley's (2002) study where genes were used to relieve a person from responsibility especially after that responsibility had been (over)emphasised, this study shows behaviour being treated overwhelmingly as the normatively correct explanation for overweight (and thinness for that matter). This difference may be explained by our more detailed look at the action orientation of the talk. The orientation to the behavioural norm was especially present in participants' accounts regarding their *own* weight, i.e. accounts that may be more susceptible to being treated as subjectively invested (rather than just true) than is the case with general explanations. And perhaps also as a result of including participants' own weight in the discussion, there was relatively little *explicit* self-responsibility talk that could have invited the gene talk (and vice versa), as was the case in Crossley's study.

In line with our study, Throsby's (2009) research on weight loss drugs and surgery shows how these interventions are commonly framed as attempts to cheat at weight loss, and avoid the necessary hard work of diet and exercise. We found a similar preference for behaviour-oriented (rather than genetic) explanations for overweight. (Explicit) genetic explanations were treated by other participants as possible attempts to avoid personal responsibility and blame.

Despite the interactional preference for explaining overweight in terms of individual behaviour, participants in both groups produced *accounts* for acting in response to health risks, namely by referring to concrete complaints or health checks. Participants' talk in this respect retained the emphasis on enjoying life and not being too concerned. This normative orientation to being relaxed about possible health risks and the allied resistance to health fanaticism has also been found in other studies (Bouwman et al., 2009; cf. Sneijder and te Molder, 2004).

Given the normative orientation to behavioural explanations on the one hand and the accountability of health-conscious behaviour on the other – calling to mind Crawford's (2006) classic argument about the tension between the need to control and the need to release – genetic testing for obesity will have to take at least two hurdles before it can act as a catalyst for healthy behaviour. In the case of overweight people, a nutrigenomics test that reveals genetic susceptibilities for overweight will possibly be treated as an invested account, that is, as an explanation of overweight that is informed by an interest in avoiding personal responsibility and/or blame. For the non-overweight, taking a test may be treated as a preoccupation with health risks and being a health fanatic (see also Bouwman et al., 2009). The crucial question is whether test results (that show a genetic susceptibility for obesity and/or diseases related to overweight such as Diabetes 2), instead of solely being used for negotiating blame and responsibility for overweight, could also be drawn upon, in everyday life, as a legitimate ground for displaying preventive behaviour, and if such preventive behaviour would be compatible with presenting oneself as leading a relatively relaxed and pleasurable life.

Unlike studies that look at how people *cognitively* understand science, we have shown how 'gene talk' can be *deployed* to shift responsibility for overweight problems, or how it can be drawn upon asymmetrically so as to allow thin children to eat fatty food. It is not the perception of genes per se, or health risks for that matter, but the way these notions are put to use in everyday talk. The acceptability of a future nutrigenomics test will depend not only on scientific quality, but also on the extent to which it is treated *interactionally* as an appropriate piece of evidence and reason for health conscious behaviour.

Harvey (2009) argues that nutrigenomics differs from traditional genetics in that it promotes a genetic entrepreneurship by which people create their own healthy future in terms of optimal wellness. Looking at current everyday talk on health and genes, we find that genetic information is not (yet) constructed as a window of opportunity – an incentive for proactive health behaviour – but as an opportunity for responsibility talk. But technologies develop through constant interaction with society (cf. Akrich, 1992). A gene-based 'wellness' focus on health may prove to be a helpful account for preventive behaviour, that is, more in tune with the broader everyday notion of health. Whether this is a desirable route to go partly depends on the extent to which consumers can be, and are, provided with adequate information (cf. Einsiedel and Geransar, 2009), especially where it concerns products marketed as lifestyle products, with an as-yet-unclear status somewhere between medicine and consumer culture (Saukko et al., 2010). But the application of nutrigenomics data

and techniques, and its underlying scripts of health and 'the good life' (Komduur et al., 2009), is not just a matter of the individual citizen or consumer (Swierstra and te Molder, 2012). What we, as users and designers, can expect from nutrigenomics tools, and whether we, for example, want the kind of healthy life that they implicitly stand for, are questions for public debate rather than merely issues of informed choice. The uncovering of underlying assumptions about healthy living and the role of genes therein, both in consumer–citizen and expert–designer discourses (Veen et al., 2011a; Veen et al., 2011b), would be an important starting point. Such a dialogue would also have to include the assumed relation between genetic make-up and behaviour. Here lies an important task for (nutri)genomics scientists. As long as the relation between genes and behaviour is reproduced as a pure dichotomy, there is little chance of turning gene talk from a blaming device into an accountable and nuanced incentive for healthy behaviour.

#### Funding

The research for this article is part of the research programme of the CSG Centre for Society and the Life Sciences (Netherlands). It is funded by the Netherlands Organisation for Scientific Research (NWO) and by Wageningen University (Netherlands).

#### Notes

- 1. BMI is an index for categorisation between underweight, overweight and heavily overweight: below 18 is underweight; above 25 is overweight; and above 30 is heavily overweight.
- 2. The notation used here is a simplified version of the original transcription system developed by Gail Jefferson:

ne next
-

3. The original extracts in Dutch are available from the authors.

#### References

- Akrich M (1992) The de-scription of technical objects. In: Bijker W and Law J (eds) Shaping Technology/ Building Society. Cambridge, MA: MIT Press, pp.205–224.
- Barnes B, Palmary I and Durrheim K (2001) The denial of racism: The role of humor, personal experience, and self-censorship. *Journal of Language and Social Psychology* 20(3): 321–338.
- Barraso I (2005) Complex disease: Pleiotropic gene effects in obesity and type 2 diabetes. European Journal of Human Genetics 13: 1243–1244. Epub ahead of print 26 October 2005. DOI: 10.1038/ sj.ejgh.5201514.
- Bouwman LI, Koelen MA and Hiddink GJ (2008) The personal factor in nutrition communication. In: Bouwman LI, Kok F and Desiere F (eds) *Personalized Nutrition: Principles and Applications*. London: CRC Press, pp.169–185.

- Bouwman LI, te Molder H, Koelen M and van Woerkum C (2009) 'I eat healthfully but I am not a freak': Consumers' understanding of healthful eating. *Appetite* 53(3): 390–398.
- Castle D and Ries N (eds) (2009) Nutrition and Genomics: Issues of Ethics, Law, Regulation and Communication. San Diego: Elsevier.
- Crawford R (2006) Health as a meaningful social practice. Health 10(4): 401-420.
- Crossley ML (2002) 'Could you please pass one of those health leaflets along?': Exploring health, morality and resistance through focus groups. *Social Science & Medicine* 55(8): 1471–1483.
- Dembinska-Kiec A (2008) Nutrigenomics and angioenesis in obesity. In: Bouwman LI, Kok F and Desiere F (eds) Personalized Nutrition: Principles and Applications. London: CRC Press, pp.23–32.
- Einsiedel EF and Geransar R (2009) Framing genetic risk: Trust and credibility markers in online direct-toconsumer advertising for genetic testing. *New Genetics and Society* 28(4): 339–362.
- Emslie C, Hunt K and Watt G (2003) A chip off the old block? Lay understandings of inheritance among men and women in mid-life. *Public Understanding of Science* 12(1): 47–65.
- Frich JC, Malterud K and Fugelli P (2007) Experiences of guilt and shame in patients with familial hypercholesterolemia: A qualitative interview study. *Patient Education and Counseling* 69(1–3): 108–113.
- Frosch DL, Mello P and Lerman C (2005) Behavioral consequences of testing for obesity risk. Cancer Epidemiology, Biomarkers & Prevention 14(6): 1485–1489.
- Guston DH and Sarewitz D (2002) Real-time technology assessment. Technology in Society 24(1-2): 93-109.
- Harvey A (2009) From genetic risk to post-genomic uncertainties: Nutrigenomics and the birth of the 'genetic entrepreneur'. New Genetics and Society 28(2): 119–137.
- Harvey-Berino J, Gold EC, West DS, Shuldiner AR, Walston J, Starling RD, Nolan AMY, Silver K and Poehlman ET (2001) Does genetic testing for obesity influence confidence in the ability to lose weight? A pilot investigation. *Journal of the American Dietetic Association* 101(11): 1351–1353.
- Hepburn A and Wiggins S (2005) Size matters: Constructing accountable bodies in NSPCC Helpline interaction. Discourse & Society 16(5): 625–645.
- Hilbert A, Dierk J-M, Conradt M, Schlumberger P, Hinney A, Hebebrand J and Rief W (2009) Causal attributions of obese men and women in genetic testing: Implications of genetic/biological attributions. *Psychology & Health* 24(7): 749–761.
- Hodgetts D, Bolam B and Stephens C (2005) Mediation and the construction of contemporary understandings of health and lifestyle. *Journal of Health Psychology* 10(1): 123–136.
- Johnson RL, Williams SM and Spruill IJ (2006) Genomics, nutrition, obesity, and diabetes. Journal of Nursing Scholarship 38(1): 11–18.
- Kokkonen R (2009) The fat child a sign of 'bad' motherhood? An analysis of explanations for children's fatness on a Finnish website. *Journal of Community & Applied Social Psychology* 19: 336–347.
- Komduur RH, Korthals M and te Molder H (2009) The Good Life: Living for health and a life without risks? On a prominent script of nutrigenomics. *British Journal of Nutrition* 101(3): 307–316.
- Lawes R (1999) Marriage: An analysis of discourse. British Journal of Social Psychology 38(1): 1-20.
- Lee D (1987) The semantics of just. Journal of Pragmatics 11(3): 377-398.
- Lee DA (1991) Categories in the description of just. Lingua 83(1): 43-66.
- Lock M, Freeman J, Sharples R and Lloyd S (2006) When it runs in the family: Putting susceptibility genes in perspective. *Public Understanding of Science* 15(3): 277–300.
- Lucke J, Hall W, Ryan B and Owen N (2008) The implications of genetic susceptibility for the prevention of colorectal cancer: A qualitative study of older adults' understanding. *Community Genetics* 11(5): 283–238.
- Ordovas JM and Corella D (2004) Nutritional genomics. *Annual Review of Genomics and Human Genetics* 5(1): 71–118.
- Oudshoorn N, Rommes E and Stienstra M (2004) Configuring the user as everybody: Gender and design cultures in information and communication technologies. *Science Technology Human Values* 29(1): 30–63.
- Pajari PM, Jallinoja P and Absetz P (2006) Negotiation over self-control and activity: An analysis of balancing in the repertoires of Finnish healthy lifestyles. *Social Science & Medicine* 62(10): 2601–2611.
- Potter J and Wetherell M (1987) Discourse and Social Psychology: Beyond Attitudes and Behaviour. London: Sage.

- Rief W, Conradt M, Dierk J-M, Rauh E, Schlumberger P, Hinney A and Hebebrand J (2007) Is information on genetic determinants of obesity helpful or harmful for obese people? A randomized clinical trial. *Journal* of General Internal Medicine 22(11): 1553–1559.
- Sanderson S and Michie S (2007) Genetic testing for heart disease susceptibility: Potential impact on motivation to quit smoking. *Clinical Genetics* 71(6): 501–510.
- Sanderson SC and Wardle J (2005) Will genetic testing for complex diseases increase motivation to quit smoking? Anticipated reactions in a survey of smokers. *Health Educucation & Behaviour* 32(5): 640–653.
- Sanderson SC, Humphries SE, Hubbart C, Hughes E, Jarvis MJ and Wardle J (2008) Psychological and behavioural impact of genetic testing smokers for lung cancer risk: A Phase II exploratory trial. *Journal of Health Psychology* 13(4): 481–94.
- Saukko PM, Richards SH, Shepherd MH and Campbell JL (2006) Are genetic tests exceptional? Lessons from a qualitative study on thrombophilia. *Social Science & Medicine* 63(7): 1947–1959.
- Saukko PM, Reed M and Britten SH (2010) Negotiating the boundary between medicine and consumer culture: Online marketing of nutrigenetic tests. *Social Science & Medicine* 70: 744–753.
- Schot J and Rip A (1997) The past and future of constructive technology assessment. *Technological Forecasting* and Social Change 54(2): 251–268.
- Segal M, Polansky M and Sankar P (2007) Predictors of uptake of obesity genetic testing among affected adults. *Human Genetics* 120(5): 641–652.
- Silverstone R and Hirsch E (eds) (1992) Consuming Technologies: Media and Information in Domestic Spaces. London: Routledge.
- Sneijder P and te Molder H (2004) Health should not have to be a problem: Talking health and accountability in an internet forum on veganism. *Journal of Health Psychology* 9(4): 599–616.
- te Molder H and Potter J (eds) (2005) Conversation and Cognition. Cambridge: Cambridge University Press.
- Swierstra T and te Molder H (2012) Risk and soft impacts. In: Roeser S, Hillerbrand R, Peterson M and Sandin P (eds) *Handbook of Risk Theory*. Dordrecht: Springer, pp.1050–1066.
- Throsby K (2009) The war on obesity as a moral project: Weight loss drugs, obesity surgery and negotiating failure. *Science as Culture* 18(2): 201–216.
- van Exel NJA, De Graaf G and Brouwer WBF (2006) Everyone dies, so you might as well have fun! Attitudes of Dutch youths about their health lifestyle. *Social Science & Medicine* 63(10): 2628–2639.
- Veen M, Gremmen B, te Molder H and van Woerkum C (2011a) Emergent technologies against the background of everyday life: Discursive psychology as a technology assessment tool. *Public Understanding* of Science 20(6): 810–825.
- Veen M, te Molder H, Gremmen B and van Woerkum C (2011b) Competing agendas in upstream engagement meetings between celiac disease experts and patients. *Science Communication*. Epub ahead of print 20 December 2011. DOI: 10.1177/1075547011427975.
- Verkuyten M (2003) Discourses about ethnic group (de-)essentialism: Oppressive and progressive aspects. British Journal of Social Psychology 42(3): 371–391.

#### **Author biographies**

Rixt Komduur is a PhD candidate working on ethical and communicative aspects of nutrigenomics. She is affiliated with the Applied Philosophy group at Wageningen University, the Netherlands.

Hedwig te Molder is full Professor Science and Technology Communication at the University of Twente and Associate Professor at Communication Science, Wageningen University, the Netherlands. Her work focuses on how issues of science and technology are handled in people's everyday talk, expert discourse and new forms of science and technology communication informed by discursive psychology. From the American Sociological Association she received the Distinguished Book Award 2007 for *Conversation and Cognition* (with Jonathan Potter; CUP, 2005).