an interview with

Public Health Genomics and its potential for health psychology: an interview with Angela Brand

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Professor Angela Brand, director of the European Centre for Public Health Genomics (ECPHG), began her career as a paediatrician. Later, at Johns Hopkins University, she became interested and trained in public health, especially in the then emerging field of public health genomics. She was one of the pioneers in Public Health Genomics in Germany and in Europe and established the European Centre for Public Health Genomics (ECPHG) at Maastricht University. Together with Bartha Knoppers from Montreal she is the editor-in-chief of the international journal Public Health Genomics.

How would you describe the role of Public Health Genomics?

We increasingly understand that whether or not a disease develops always depends upon the interaction between genomic factors *and* environmental factors that include social factors, lifestyle factors, and psychological factors. Public health has always been interested in the role of environmental factors in disease, but has so far ignored the genomic part. It is the goal of Public Health Genomics (PHG) to integrate genomics in every public health task, varying from the surveillance of infectious diseases, the improvement of nutrition, or in the psychological field, the empowerment to behavioural change. PHG, therefore, implies doing translational research.

Genomics shows us that there is a permanent interaction between the genome and the environment and that there is not a single 'determinist' factor. Our behaviour, for example, is not only influenced by social factors, but also by our genomic make-up with its multiple variants. If for example, we try to empower people to stop smoking, we see that there are people for whom it is almost impossible to quit, and today we know that to a large extent this may be due to genomic variants that specifically predispose to nicotine addiction (Berrettini & Lerman, 2005).

Genomes are not static, but dynamic and highly complex systems. We know now, that social factors, like certain experiences and life events – including therapeutic interventions - may result in changes at the level of our genomes (Sweatt, 2009). That is what we

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have learned from recent insights in epigenomics and it is highly relevant for public health.

Can you give some further examples of the public health effects of epigenomics: can't it be that environmental factors modify and trigger health outcomes by changing the genome?

Yes. Take obesity, for example. Before and during the Second World War there was not much food. This may have affected the susceptibility of future generations to developing diabetes, as children conceived during the years of famine carry epigenetic 'signatures' of this hunger period (Heijmans et al., 2008). Then suddenly this changed after the Second World War and for the last generations of people a lot of food was available while their body was not adapted to this abundance. So, obesity figures grew. Today, we see that obesity figures do not grow anymore in children, partly because we adapted to the availability of food. That is, our genome no longer carries the epigenetic 'signature' of severe famine. Nevertheless, obesity consists of several different subtypes which are totally different entities. There are subtypes of obesity in which epigenomic effects are not that strong. ➤

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A very different example is the case of child abuse, where interventions normally focus on the social background and family members of the child. New evidence suggests that child abuse has also to do with genomics, and that it has long-lasting epigenomic effects (McGowan et al., 2009; Welberg, 2009).

To what extent does this relate to specifying outcome measures?

We should use the concept of health outcomes instead of diseases, because a health outcome can actually be the state that results from a clustering of different disease phenotypes that we did not bring together in the past. However, some of these diseases may have a common genomic background. Disease associated genes have been mapped to pathways and it has been found that a number of different diseases often share the same pathways. Li and Agarwal, for example, found that diseases as different as myoclonic epilepsy, Turner syndrome and Wegener granulomatosis all map to one pathway (Li & Agarwal, 2009). By linking genetic disorders ("disease phenomes") with known disease related genes ("disease genomes") networks can be constructed that show the various "diseasomes": clusters of related disorders (Goh et al., 2007). However, even if we know the genomic variation, we still do not know which diseasome will develop during the lifespan of the individual. One further relevant issue is that traditional epidemiological models do not fit anymore, since the new developments stress the importance of looking at the individual level and this means that we cannot generalize our findings, as we used to do. It comes down to personalised health care (Brand, 2009), we should focus on long term monitoring of processes within the individual instead of focusing only on comparisons between individuals.

In one of your articles in the European Journal of Public Health you state that "It should be kept in mind that we have to be careful about the message 'prevention and health promotion is good for everybody…'" (Brand, 2005). Could you elaborate on this?

By that I mean that a public health message based on the strategy of "one size fits all" is not adequate. Let's take the example of the message that soya is good for everybody, while in fact it is not. It can be a protective factor regarding certain types of cancer, but in certain situations it can have the opposite effect, for example when a person has a carcinoma in situ. The same goes for olive oil, and also for physical activity. The sudden death among young sportsmen with a particular genomic variant in the beta myosin heavy chain gene (Marian et al., 1994) is an extreme example of this, but it can be prevented even though the incidence rate is low.

One further example is alcohol consumption. Some people can get very aggressive after consuming only a very small amount of alcohol. They can't control themselves anymore. Should they be responsible for that situation? Therefore, prevention for these people should not focus on consuming less alcohol, as may be the general public health recommendation, but on preventing that these people end up in a situation in which their aggressiveness can get triggered by alcohol consumption. Thus, here the message should be, that these individuals should never start drinking alcohol at all. Finding this variant in a genome and communicating this message to the individual is at the core of personalized health care.

What does this mean for public health messages?

The message is that one cannot claim that all generally good interventions are good for everybody. Interventions should be more target-oriented, and in the end it comes down to individual approaches taking also the genomics into account, as early as possible. This is practically and politically very difficult and it raises the question of how early is as early as possible? We can for example test for genomic variation during pregnancy, but what are the consequences? We should debate this issue in a transparent way, as newborn screening is established in almost all developed countries for over 40 years and it can be extended to include many health outcomes. The biggest challenge is how we will manage this. Genetic counsellors will play their part, but there are also possibilities for other health professionals including psychologists.

To conclude, do you have a take home message for health psychologists in general?

The competences of health psychologists are needed, since public health genomics is a multidisciplinary task. Genomics is just one factor among many other factors that we need to consider within the multifaceted task of public health. If in every single task there is a certain awareness of the role of genomics, then we can solve the challenges we face.

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