Epigenetic Concepts of Environment

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Abstract

Epigenetic approaches are increasingly finding their way into various fields of biomedicine and biomedical research. *Epi*geneticists deal with cell processes *beyond* the genes – the environment of genes. At first sight, epigenetics does not just consider environmental influences, it also seems to be the opposite to gene determinism. This contribution, however, aims to take a closer look at epigenetics and argues that the epigenetic concept of environment is a very specific, or even highly problematic one: as epigenetics assumes that genes are coined by the 'environment' – namely behaviour and lifestyle –, the imperatives of genetic responsibility are intensified. Hence, the central claim of the article is that in current epigenetic research this attention to environmental influences has been diverted into an even more sophisticated version of epigenomic susceptibility: epigenetics creates a holistic version of epigenetic risks and responsibilities.

Introduction

Epigenetics often presents itself as something originally new, as the hottest newest branch in biomedicine and the various fields of the life sciences. At first sight then, epigenetics does not only seem to be a solution to problems described by former geneticists, but also seems to have considered some of the critique of social and gender scientists against the geneticization of diseases like breast cancer. Many authors have criticized gene-deterministic research and biomedicine for not considering the complex interactions of genes and environment. By exclusively concentrating on genes the interplay between external influences and genes would not be considered adequately. The role of pathological environmental factors could not be fully understood and the effects of stress, environmental pollution or bad working conditions would not be examined. According to the critics, the individual would be made responsible for his or her disease instead of examining and changing the societal reasons for it (e.g. Gibbon 2007; Lemke 2004; Palfner 2009; Zur Nieden 2007; 2009).

In epigenetics, environment and the interplay between environment and genes is now becoming a crucial focus of interest. Taking these shifts and especially the epigenetic concept of environment as a starting point, I discuss epigenetics critically. The main hypothesis is that epigenetics has not only re-emerged because of previous geneticized research, but has also re-articulated concepts of environment in a manner that it might even intensify some of the risks of the 'gene-deterministic era'. In sum, it can be stated that the analysis of epigenetics is relevant to promoting critical debate on the geneticization of research, as some of the social scientific approaches and critique no longer fit the bill when examining current epigenetic approaches.

Furthermore, this contribution will focus on epigenetic approaches to breast cancer. One reason for this is that many of the social scientific and gender based critics of the geneticization of diseases concentrated on breast cancer (e.g. Lemke 2004). Palfner (2009) claims, for example, that breast cancer was not only the most geneticized disease, but that researchers also hoped it might be possible to translate genetic findings from familial breast cancer into meaningful knowledge and medical applications related to much more common sporadic cancers. Another reason for concentrating on breast cancer is that breast cancer research scientists were also crucial driving forces in the process of establishing central epigenetic research networks such as the Epigenome Project (Bradbury 2003; Esteller 2006; Jones & Martienssen 2006; Rauscher 2005), which is a multinational European network of research scientists. Initiated in 1999, the aim was to 'identify, catalogue, and interpret genome-wide DNA methylation patterns of all human genes in all major tissues' (Genomic Enterprise 2010). The network is financed by government funds as well as private investments via a consortium of genetic research organizations. The main organizations involved are the British Welcome Trust Sanger Institute, the German and US-American Epigenomics AG and the French Centre National de Génotypage (Human Epigenome Project 2010). Breast cancer is also one of the main topics of the network's publications (Epigenomics 2010).

In this article, I try to examine epigenetics related to breast cancer utilizing expert interviews (Littig 2008; Meuser & Nagel 2004) with scientists from Germany and Great Britain who broadly defined them-

selves as epigeneticists working in the field of breast cancer research. These people included cell and developmental biologists, chromosome biologists, molecular geneticists at university clinics, universities and other research centres. In addition, I draw on observations of professional conferences on epigenetics and secondary data including scientific journal articles, websites of expert networks, position statements, popular and press reports.

I begin with a brief introduction to epigenetics. I then argue that the emergence of epigenetic approaches in the field of breast cancer research can be understood, somewhat surprisingly perhaps, in direct relation to genetic determinism. By focusing on the idea of individualization and the epigenetic notion of environment the final part examines some of the shifts in current social settings related to epigenetics. Epigenetics often presents itself as a new approach in the field of breast cancer research. However, as I intend to argue, the seemingly successful re-emergence of epigenetics is only thinkable *in relation* to the research on breast cancer and the focusing on the two so called *br*east *ca*ncer genes BRCA1 and BRCA2.

Epigenetics

Epigenetics encompasses a range of molecular biological approaches, which essentially deal with three groups of molecules: groups of methyls and enzymes for methylation; histones, namely acetyltransferase and deacetylase and RNA molecules. Epigenetics assumes that all of these molecules influence gene regulation inside a cell: they are considered to influence gene expression without transforming the genetic sequence of the DNA (Landecker 2008, 43). According to Asch and Barcellos-Hoff, epigenetics 'has been defined as the study of heritable changes in gene expression caused by mechanisms not involving changes in DNA sequence' (Asch & Barcellos-Hoff 2001, 151). Epigenetics thus assumes that an epigenome exists, which sits 'on top' of the genome. This metacode is thought to be crucial for genes to 'be read' and for understanding how genes are repressed or expressed.

The emergence of the 'object of knowledge' – the epigenome – appears to correlate with a critical epistemic shift. Whereas genetics seemed to finally situate the nucleus as ahistorical and presocial (Burren & Rieder 2000, 23; Fox Keller 1998, 27–28), epigenetics understands the regulation of genes, the epigenome, not as an entirely biological process, but as also 'culturally' constituted (Parnes 2006, 92). Epigeneticists assume that environmental influences are the main forces for gene activation. These influences in turn form the 'memory of the body' and their consequences are inherited (Epigenome NoE 2010).

A British developmental biologist, who is regularly cited by researchers in the field of breast cancer, described the transgenerational transmission of epigenetic patterns as follows:

> That is passing on not the genetic information but the epigenetic information. (…) There are instances where the memory of the fetal cells (…) or something like that gets transmitted to the subsequent generation, or may escape one generation and goes to the next generation. (Maier 2009)

Epigenetic approaches not only assume that the environment is crucial for gene regulation of some 'present' living singular individual but also for possible future generations. The epigenome is seen within genetic research as the point where nature meets culture. Or to put it another way, the epigenome is the space where culture *coins* nature, where environment becomes a crucial force for gene regulation. The Epigenome Project for example, strives to map these borders and no longer sees its own bioscientific practice as a precise copy of the genetic matter, as an objective and rational depiction of the natural world, but as a procedure in which biology and nature are intertwined. A less reductionist notion of nature/culture seems to be part of the epistemology of epigenetic approaches. At first glance, epigenetics seems to contradict the often critically questioned gene-determinism of genetics (e.g. Lemke 2004) or in the case of breast cancer, the belief in the dominant gene.

Epigenetics and breast cancer research

There is a growing interest in epigenetic approaches in the field of breast cancer research. For example, the German Cancer Research Center hosts an entire division which is dedicated to the study of epigenetics (Deutsches Krebsforschungszentrum 2010). Speakers at scientific conferences on breast cancer research often refer to epigenetics. In addition epigenetic modifications are considered 'key factors in breast carcinogenesis' (Dworking et al. 2009, 165). As a result, epigenetics is described as the 'hottest newest branch of biomedicine (…) where all the hype is' (Dunckley 2008).

However, I demonstrate that epigenetic approaches are in fact not contrary to research which is often described as gene-deterministic. Moreover, following the main argument of this paper, I suggest the shift of attention from strictly genetic to epigenetic approaches and the emergence of epigenetics in the field of breast cancer research must be understood *in relation* to the genetic approaches to breast cancer and the former primacy of the breast cancer genes: epigenetics is not original, but rather promises to resolve some of the 'dead ends' of research on breast cancer. As the example of a German molecular geneticist below illustrates, the interviewees underlined the long history of research on epigenetic mechanisms:

> The influence of DNA methylation has already been known for decades. It is already longer known as that we se- [sequence, u.k.] any genes, well, and the fact that, I don't know, I guess in the 70s this histone complex (...) was identified. That is even older, could be, I have to look it up. Epigenetics exists much longer. (Spanzer 2009)

Research *explicitly labelled* as epigenetics, however, emerged only at the end of the 1990s (e.g. the above mentioned Epigenome Project, a European network of epigeneticists, founded in 1999). Before this time and for many years, the belief in genetics, and more precisely in the dominant gene as the main cause for breast cancer, was so strong that epigenetic approaches did not emerge very strongly in relation to breast cancer. Asch and Barcellos-Hoff, for example, suggest that 'among the reasons why this concept has languished in the backwaters of cancer research lies in the reluctance of molecular geneticists to embrace the revolutionary

principle that not all of the significant changes driving this diseases are due to lesions in DNA sequence' (Asch & Barcellos-Hoff 2001, 151).

One reason why epigenetics emerged in the field of breast cancer research was that genetic approaches could not fully answer the questions they posed. The earlier cited British developmental biologist, whom I interviewed personally, claims for example:

> The re-emergence of epigenetics started with several things. One was that when people had started to think about the genetics of development, and particularly when the genome became sequenced, then people started to say well we know that there are 20,000 genes, but how do you actually select which genes you want to use to make different cell types. And that made people start to think about epigenetics. Because that's, how do you read the genome, how do you select the genes at the right time? So that was then that the attention kind of started to shift towards epigenetics. The other thing is that there was more information coming out on epigenetic mechanisms and particularly on various enzymes that are important for introducing histone modifications for example and the methylations. (...) I think that that kind of accelerated the pace of the research. So it is a combination of things. Having the genome sequenced plus the fact that the work on epigenetics had started to increase the knowledge was increasing. That kind of made people think about it a bit more. (Maier 2009)

Ash and Barcellos-Hoff explain the emergence of epigenetics similarly:

> The slow progress made in curtailing breast cancer incidence and mortality during the previous century implies that key pieces of information for fully understanding its initiation and parthenogenesis are still missing.
> (Ash & Barcellos-Hoff 2001, 151)

Both these comments suggest that what makes epigenetic approaches so interesting for scientists at the moment is that they seem to have the potential to answer some of the questions already opened up by a geneticized field of breast cancer research. It is no surprise then that Denise Barlow, geneticist at the Austrian Research Center for Molecular Medicine, claims: 'epigenetics has always been all the weird and wonderful things that can't be explained by genetics' (Epigenome NoE 2010). We can see then that the notion of the epigene is not a truly new version of the gene, but

opens up a new space of problematization between the gene, disease and environment. Moreover in relation to the previously discussed plasticity there is an expectation among scientists, that the epigenome will provide the missing link between genetics, disease and environment (Beck et al. 2005, 265).

Nevertheless, the question of how exactly this space of epigenetics is constituted is not yet explained. In their essay on the relationship between methylation and the emergence of tumours, Prawitt and Zabel state that 'as persuasive and suggestive as these relationships sound, it is still not yet known how it is precisely that effective hypermethylations materialize' (Prawitt & Zabel 2005, 298). Whereas a notion of the gene as a stable entity (like BRCA1 and BRCA2) no longer appears to be productive within the context of research, the uncertainty of the epigene does seem to provide what Rheinberger (2006) has described as the motor of experimental research.

Individualization and environment

Epigenetic approaches to breast cancer are not constituted by one unique concept of environment, but rather by several notions of environment. I suggest three roughly separated, analytical classifications: First, environment can refer to a nuclear environment, i.e. the environment at a micro-level inside a cell. Dworking, Huang and Toland say for example: 'The findings suggest that breast cancer patient microenvironments induced epigenetic changes in this immortalized breast cell line' (Dworking et al. 2009, 165). Second, it can mean the cellular environment, i.e. the environment surrounding the cell or the interaction of different cells. As with the first concept of environment this also refers to the 'interior' of the body. Third, epigenetics also deals with an environment in which the individual is embedded, i.e. diet, stress or therapeutics. This environment means an environment located outside the individual body.

In the following I will focus mainly on the third notion of environment. To explore the last notion of environment it is important to recognise that epigenetic analyses are linked to a practice of individualization at

the level of risk detection as well as treatment of breast cancer. Detection means the detection and interpretation of the individual patient's methylation patterns. How this can be done is for example described by the cancer molecular diagnostics company *Epigenomics*, the main financial supporter of the Epigenome Network:

> Epigenomics is creating an integrated genomics-based technology with the potential to revolutionize the development of personalized medicines. By detecting and interpreting DNA methylation patterns (5th BASE Genomics®) in different cells of the same person, Epigenomics has uncovered a new layer of biological information that will enable a more complete and clearer detection of complex disease. These DNA methylation signals, comparable to a genetic switch, can be digitized to create a Digital Phenotype® that reflects the genetic activity of a particular cell, i.e., whether the cell is healthy or sick. Epigenomics' sophisticated machine learning methods extract valuable information from these extremely high dimensional data to form the basis of a diagnostic component of personalized medicines. (Epigenomics 2010a)

It is claimed that the precise detection of epigenetic patterns will enable a personalized therapy:

> A more focused application of therapy will be possible, though, by a more specific elucidation of the mechanisms of epigenetic modifications and the thereby better measurable effects of transformations of these modifications.
> (Prawitt & Zabel 2005, 302)

A further crucial aspect is that epigenetic modifications are considered to be reversible:

> A further aspect of the epigenetic findings, which should be considered again and again, is the fact, that methylation – such as the other epigenetic modifications – is dynamic and not like DNA-sequence necessarily static, that means epigenetic modifications can be removed. (Prawitt & Zabel 2005, 302)

As these examples illustrate, the aim of epigenetics is to detect individual methylation patterns and, based on these patterns to develop an individual therapy consisting of exact combination of medications, therapies

and/or diet. This aim of personalized medicine was also articulated by genetic approaches, as reflected in the following statement by one of the epigenetic scientists interviewed:

> {e}pigenetics mean (…) that it would in the long term be possible to carry out a fine-tuning, or maybe specification. Specification is the correct word. And that as well when you look at diagnosis as well as therapy. Well with epigenetics it maybe would be possible to make more precise diagnoses and prognoses (…). Is there still something in the blood and then to fine tune, also very precisely, the therapies. Does this woman need chemotherapy or is something different enough? Does she have a high risk for a second cancer? Considering therapy everything becomes important. Chemo, medicaments, diet, reduction of stress (…) Yes that is right. That was also important during genetics, if we call it like that, but that was a big tangled mass. Well we knew that there was something, there is a disposition, but how exactly and if genes were relevant (…) maybe genetics comes in at this point. Well, something like a fine tuning, yes. (Halbert 2009)

This possibility echoes the former hopes of gene therapy, which had promised the ability to intervene in the genome. It also links back to older discourses on BRCA. Current epigenetic approaches, however, do not mean a renaissance of a mere environmental determinism. There are in fact crucial differences.

As mentioned above, the term 'environment' is important for epigenetics because it traces epimutations back to environmental factors. But this epigenetic notion does not correspond with the feminist or social sciences or popular notions of environment – it is not bad living or job conditions or environmental pollution (e.g. Lemke 2004). Rather, epigenetics conceptualizes environment as an individual environment, more precisely as an environment which can and should be created individually by 'choice' related in this instance to smoking, diet, prenatal nutrition or individual stress. Epigenetics assumes that these 'individual lifestyle practices' are transformed into epigenetic markers and can influence the regulation of genes.

Similar sentiments relating to individualized control are illustrated in an article in *Science* entitled 'Why genes aren't destiny. The new field of epigenetics is showing *how your environment and your choices* can influence your genetic code – and that of your kids' (Cloud 2010, italics added).

In the case of breast cancer, epigeneticists assume that patterns of methylation are crucially influenced by environmental influences:

> If for the concerned individual [the one which has cancer, u.k.] an external stimulus like smoking cigarettes is added, this probably leads to the hypermethylation of the tumor suppressor genes. (Prawitt & Zabel 2005, 298)

Furthermore, epigeneticists claim that these epigenetic markers can also be passed from one generation to the next. They are then called 'germ line epimutations' and are currently considered as 'the first step in tumour development and thus directly predispose to cancer' (Dobrovic & Kristensen 2002, 34).

It is important to note that this narrow notion of environment is not gender neutral. This is because epigenetic environment means the 'conditions in the womb' (Cloud 2010, 23), it is first and foremost the female body which forms the environmental context through which the object of epigenetic knowledge is produced. This was how one of the epigeneticists interviewed put it:

> So I think the maternal role, the part that the mother plays, if you like the epigenetic mechanism is possible during pregnancy when the fetus is growing. Because there is a possibility that there could be environmental changes or influences that again impact on the fetus and so these kind of environmental factors could have a big role in (…) [and] for example maybe at the root of some of the diseases such as breast cancer and so on. So that's one of the ideas that people have put forward. And I think that that's fairly logical, because if (…) because you can bring about epigenetic changes in many ways and environmental stress or environmental change can have an effect on gene expression. And therefore you could imagine that a growing fetus, which is somehow exposed to any kind of environmental factors or even stress or something like that could result in that change. So that is one way of thinking about it. (Maier 2009)

By contrast, the paternal side is considered to be less influential, because as Dobrovic and Kristensen note 'complete erasure of epimutations during spermatogenesis has been observed, and so far evidence is only present for maternally derived inheritance and, suggesting that epimutations are less likely to be erased during oogenesis' (Dobrovic & Kristensen 2002,

34). In this sense the noteworthy attention to environmental factors and to sex-specific microcellular processes in epigenetic knowledge turns out to be a gender-specific reduction of the notion of environment to individual choice and responsibility. The notion of the epimutation can mean an intensification of the risks that have been widely critically examined for gene-deterministic approaches.

This is because epigenetics no longer refers to the assumption of a 'genetic disposition' which is fate, but implies plasticity. The epigene has a much greater capacity to be influenced by and be susceptible to the environment defined as relating to individual lifestyle factors.

A key question is whether epigenetic notions of environment signal a renaissance of a simple environmental determinism. All these notions of environment do not imply a direct causal relation, a simple environmental determinism. Moreover, as Hannah Landecker underlined, 'epigenetics examines and understands the milieu as a molecular cloud of factors and signals that wraps us, penetrates us and transforms the inner intricate networks of the exchange of signals and regulation' (Landecker 2009, 53). Nevertheless, all of these notions of environment never mean a social environment, but always an environment which can be managed by the individual, or in terms of developing targeted individual therapies or medication.[1]

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Note

[1] An area where a bigger notion of the environment is entering the genomics field is toxicogenomics, which is interested in the relation between gene and protein activity in response to toxic substances. Sara Shostak (2010) explores the fact that at least some areas of genomic research encompass a wider set of social references.

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