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The challenge of genetics to social inequality research



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One of the rising challenges in social inequality research is the inclusion of information about genetic variation into the mechanisms that produce social inequality. In the last decades of the last century the social sciences profited from considerable tailwind supporting their claims to explain human behaviour, and differences and inequalities between individuals within and between social groups, organisations, and societies. Moreover, studies in social action and structure were expected to provide the necessary information to design institutions and policies more effectively and efficiently in accord with societal demands and values. An important example is educational systems and policies, where in Germany the proverbial 'catholic working class girl living in rural areas' was the synopsis for social disadvantage in the educational system that has to be defeated to reach equal opportunities.

However, latest around the millennium the wind turned, and the social sciences encountered it increasingly from the head, while 'life sciences' and especially genetics emerged as new promises to investigate how life unfolded over the life span. Even in the wider public thinking in genetic endowments seemed to become the new paradigm to understand what creates differences and inequalities among human beings. Already in the 1990s in the US the publication of *The Bell Curve* by HERRNSTEIN and MURRAY heated the debate by pretending that, after decades of equal opportunity policy, it is now mostly genetically driven ability that governs social mobility. In consequence, remaining unequal life chances are mostly in line with meritocratic ideals that talent should pay off, and moreover cannot and should not be remedied by public policy anymore.

Also behavioural geneticists who long had investigated the impact of genes on individual characteristics with the help of designs in which family members with different degrees of genetic similarity

are compared, came under pressure: Why rely on shaky modelling assumptions to find out what a black box of unknown overall genetic variation contributes to the variation in an individual characteristic or social outcome if we can read in the genetic makeup itself? Meanwhile it becomes more and more obvious that molecular genetics still needs more time to follow up on the great promise of solving the last riddles of that black box. Despite considerable progress in molecular genetics enabled by the very fast drop in the cost of human genotyping, disillusion has spread about how fast exact pathways from the genome to social outcomes will be detected and measured appropriately. At the moment several approaches with partly complementary strengths and weaknesses stand by to answer questions about how genetic and social forces contribute to the emergence of social outcomes, and all of them have their sometimes overzealous advocates. First, modelling approaches based on genetic similarity of family members, especially twin-based quantitative genetics, have developed more complex and powerful tools to cope with weaknesses of simple twin analyses, like extended twin family designs. Nevertheless, all these approaches still remain black box approaches where genetic variation is estimated but not measured. Secondly, molecular genetics have increasingly moved away from candidate genes to polygenic scores of individual characteristics and social outcomes (VISSCHER, 2016). Candidate genes can focus on mechanisms that translate genetic endowments into social outcomes, like in the case of differential susceptibility (see below) but generally suffer from low replicability and from capturing only a very small proportion of the relevant genetic variation. Polygenic risk scores in contrast are able to capture a much higher proportion of the genes involved but at the moment relate simply to correlations between gene variants on one side and individual characteristics and social outcomes on the other, whereas mechanisms linking genes and outcomes are not targeted. Thirdly, gene transcription patterns and epigenetics promise to disentangle the biological and social forces linking the genome to social outcomes. At the moment, however, these processes seem so complex and difficult to measure in large samples that for humans it is rather hints than proofs for how and to which degree they contribute specifically to the emergence of social inequalities.

Recognising the roles of genetic factors in producing social inequality is important for methodological and substantial reasons. Purely social investigations of family-of-origin influences obscure the statistical confounding that genetic transmission introduces. This strongly challenges any interpretation of advantage and disadvantage based on this research. Alone for this reason it is worthwhile to dismantle genetic effects from social influences to gain a less biased and therefore more reliable estimate of pure social effects. However, beyond these general considerations it is necessary to investigate in detail whether and how the inclusion of genetic information challenges indeed existing knowledge and adds new fruitful perspectives. So what is the use of including genetic information into explanations of social inequality? What has been reached already, and what are promising pathways for future research?

To start with the challenge by *The Bell Curve*: We know now that genes contribute to both: the intergenerational reproduction of social inequality as well as an escape from the destiny arranged by the living conditions of the family of origin. Looking at existing genetically informed studies about educational attainment and income determination we do not see that social influences are much lower or even overridden by genetic ones in the sense that influences formerly assumed to be social are now uncovered to be camouflaged genetic ones. Obviously genetic and social forces contributing to social inequalities coexist to a considerable degree. In other words, social inheritance and genetic inheritance seem to be two distinct and separate sources of inequality (CONLEY, FLETCHER & DAWES, 2014).

But the degree to which they contribute is obviously subject to social influences, not least larger social forces. This is demonstrated in a meta-analysis of educational attainment where for individuals born in the latter half of the twentieth century more of the variance in attainment can be explained

by genetic variation, whereas shared environment explains a greater percentage of the variance in attainment for those born in the earlier half of the century (BRANIGAN, MCCALLUM & FREESE, 2013). Education stands out in behavioural genetic research by the fact of a strong shared environment influence, contrary to most physical and psychological characteristics. Therefore, applying genetically informed approaches to the study of inequality in life course outcomes, like educational and status attainment, will challenge some over-generalisations of previous behavioural genetics, in this case that shared environment never matters (TURKHEIMER, 2000).

However, this is only a preliminary statement in the still very fragmentary genetically sensitive research on social inequality. Beyond skill formation and education, social mobility patterns, tertiary education and vocational training, job careers, and earnings and wealth are still hardly researched at the present state-of-the-art methodological levels over and above rather simple heritability estimates. The more this is true for the important question of how genetic and social forces contribute to patterns of accumulation or compensation looking at more than one inequality domain.

Despite consistent evidence for largely additive effects of genes and social forces both in behavioural genetic modeling as well as molecular genetic research, the question of how genes and the social environment interfere receives increasing attention. Some researchers are proclaiming an ‘environmental turn’ which means that we hardly understand anything of genetic influences if we do not take into account the manifold ways by which social environments shape genetic expression over the life course. Two forms of gene-environment interplay are distinguished: gene-environment covariance and gene-environment interaction.

Gene-environment covariance

In general, genetic confounding of social effects can be due to an unequal distribution of relevant alleles among related groups—e. g., lower class children have less favorable genetic propensities than higher class children—or by gene-environment covariance. The latter means that measured social effects are partly genetic effects because the environments we experience are not random but are in part a function of the choices we make, and these choices are in turn a function of genetically influenced talents, dispositions, and interests. Also the social environment’s reactions to us are partly induced by genetically influenced characteristics. Thus, there are hardly any environmental effects that are not confounded with genetic differences. Therefore, without controlling for genetic resemblance of parents and children, a correlation between parents and children cannot simply be seen as “prima facie evidence for sociocultural causal mechanisms” (TURKHEIMER, 2000:162). Take the example of parenting: Musical parents raise their children in an environment that motivates their children to become musician themselves (i. e. instruments at home, listening to music). Being musical might also be genetically transmitted. These children passively receive a social context that fits to their genetic predisposition. An active or evocative correlation describes a situation in which genetically transmitted characteristics provoke specific reactions from the environment. For example highly talented children might receive special attention from parents and teachers which reinforces their talents. And among the possible activities with parents they actively seek those that match their genetically transmitted interests, and try to avoid others.

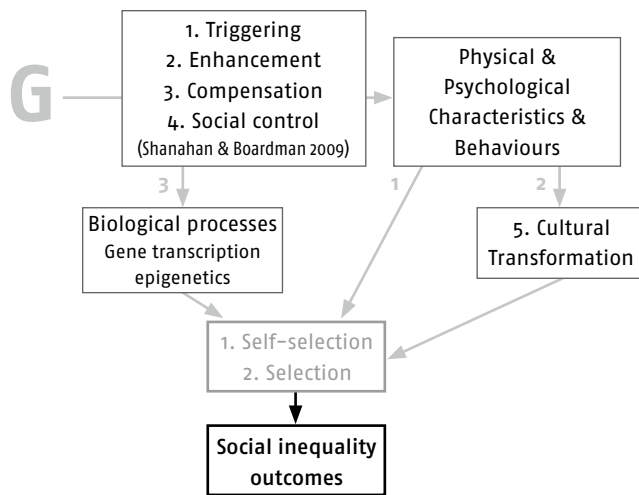
Gene-environment interaction

Individuals with the same genes can experience very different lives if exposed to different experiences and events. *Vice versa*, and this is of paramount interest for social scientists, genetic variation contributes to different individual responses to the same environment. Siblings who experience the same negative life events like danger or poverty react to them very differently, exhibiting vulnerability or

resilience. And likewise they can profit quite differently from positive circumstances, e.g. in the forms of stimulating and supportive homes.

Even if gene-environment interactions may not play such a decisive role for the overall variance in an outcome compared to additive effects, they are nevertheless of paramount interest for the investigation of social inequality, because they can easily be linked to sociological relational inequality theory: Different social groups dispose of different opportunities to make use of their genetic endowments and have different chances to avoid negative consequences of genetic risks (DIEWALD 2016). The following figure displays the various ways in which such interactions can occur.

$G \times E$ on the way from genes to life chances



The social context can operate in various ways, and so far four ideal types of $G \times E$ interactions have been differentiated (SHANAHAN and BOARDMAN 2009). Triggering and enhancement denote the two most common mechanisms of how genes and environment interact. Triggering is oriented around expression of vulnerabilities and disorders. It means that a person has a genetic vulnerability that is expressed only in specific social situations. For example, individuals with a genetic predisposition for depression are more likely to suffer from depression when having experienced a stressful life event earlier in their lives. Here the social context works detrimental and triggers the occurrence of a genetic risk. This mechanism is also known as stress-diathesis. Enhancement describes a social context that helps to develop genetic 'potential' or, in other words, that increases the genetic predisposition towards socially valued or accepted characteristics or behaviors. The difference to the first type is that enhancement refers to processes and interactions which increase positive functioning. E.g., the effect of genetic predisposition for talent is accentuated via training or good parenting. Compensation refers to the opposite of triggering: Here, the social context is enriched and positively impacts individual functioning by hindering the expression of a genetic risk. Aggressive behaviour can be prevented when growing up in intact families with warm relationships for instance. Compensation and triggering do not necessarily represent an absolute dichotomy, they can rather be seen as two ends of a continuum.

In the fourth type, the environment serves as a mode of social control, which sounds similar to compensation but refers to (institutionalized) belief systems, punishment of disorder, and behaviours of significant others to prevent or redirect disorder. The difference to compensation (i.e. avoidance of low levels of functioning) lies in the substantial mechanisms. The social control mechanism

describes the limitations to individual's behaviour which prevent the realisation of a genetic predisposition.

All four mechanisms refer to the development of physical and psychological characteristics and behaviours.

As an additional fifth possibility I would add that the same alleles might lead to the same or similar general characteristics but in different cultural transformations. As stated by CONLEY (2009, p. 238), "a gene for aggression lands you in prison if you're from the ghetto, but in the boardroom if you're from the manor born". In a similar way, as part of cultural capital, a sense of entitlement may be developed in a form that conforms to the unwritten rules of higher class communication or in a form that by offending closes the doors of institutional gatekeepers.

In sum, the methodological and theoretical inclusion of genetic variation in social inequality research offers an additional step in the causal chain from origin to unequal life course outcomes, namely how favorable characteristics like cognitive and noncognitive skills are developed out of the genome by different degrees of enhancement, and how the development of unfavourable characteristics like antisocial behaviour or aggression is enabled by triggering or inhibited by compensation or social control in different social contexts. Genes matter also for a person's position in society, though there is clearly no gene for income, socioeconomic status, or social class. The only characteristics directly influenced by genes are those that lie 'underneath the skin'. In other words, genetically based similarities in attainment between parents and children must be explained by physical or psychological characteristics and processes that are relevant to reach status-relevant outcomes. These characteristics influence individual behaviours and evoke different reactions in the environment, resulting, for example, in different labour market outcomes and recruitment to different jobs.

All five types of gene-environment interaction define relational inequalities between different social groups, insofar the distinction between women and men, different social origins, or natives and migrants is linked to different chances to develop favourable or to avoid unfavourable characteristics and behaviours out of the same genetic endowments. E.g., GUO and STEARNS (2002) compared the levels of realised genetic potential for intellectual development across different social groups. They showed that children from disadvantaged social backgrounds have lower chances to realise their genetic potential than those from a social background with more resources and no ethnic discrimination. In other words, children from disadvantaged backgrounds are less able to develop their genetic endowments, very much dependent on societal institutions (TUCKER-DROB & BATES, 2015)

Insofar as psychological characteristics are concerned, these mechanisms can quite easily be integrated in social inequality research based on models of educational and status attainment where ability and effort play a central role. Also recently addressed physical attributes indicating sexual attractiveness or authority can easily be integrated, since we know that not only our own talent matters, but also what others think how smart we are. More generally, psychological factors have proved to be important to understand how individuals behave and how they fit into their environment. There are, however, two major inputs of genetically informed research to conventional, i.e. purely phenotypic research designs including these psychological factors: First, it extends the perspective of how advantage and disadvantage emerge over the life course to the starting point of different genetic endowments relevant for the development of such characteristics. Secondly, to understand how environment shapes genetic expression and how genes influence the experience of and reaction to environments directs the attention more to the expression of vulnerabilities and disorders as possibly important inequality-generating factors than usually done in social inequality research.

As the increasingly discussed differential susceptibility hypothesis suggests (PLUESS, 2015), the same genes may even contribute to enhancement and stress diathesis, or triggering, at the same time.

In an evolutionary perspective you may ask why risky genetic variants still exist and are not eliminated in earlier generations. One answer is that a species has better chances for survival if there two types of genetic predispositions called 'dandelions' and 'orchids' in the population. Dandelions are those who are generally less susceptible for environmental influences. In other words, they do quite fine under whatever circumstances. They are resilient against shocks and shortage of resources, but on the other hand they do not profit much from rich environments. Other than these robust individuals, the orchids are highly susceptible to environments. Under harsh conditions and exposed to shocks orchids do less well than the dandelions; they are not resilient but vulnerable. But in resourceful and fostering environments, they flourish more than the dandelions. Under moderate conditions dandelions and orchids should develop not so differently, but under more extreme conditions, for better or worse, they should.

An alternative, much less common strategy for studying gene-environment interaction on the way to social inequalities is to investigate biological processes, especially gene transcription patterns in the form of endophenotypes, or epigenetics, which refer more directly to organism's reaction to environmental influences. SHANAHAN (2013) provides a number of examples of a "durable programming of the stress response system", distinguishing between "fight or flight" responses to stressors. Such patterns of transcription regulation may play a crucial role in the link between social origins and socioeconomic outcomes, because on the one hand they begin to operate very early in the life course during the sensitive period around birth, with parental SES and its stability, and parent-child relationships exerting a major impact on the activation or repression of genetic activity that regulates stress. On the other hand, stress regulation appears to play a crucial role in brain development, which in turn is important for later educational and socioeconomic attainment, as reflected, for example, in a higher IQ (NISBETT et al. 2012).

Whereas these approaches mostly focus on the question why early conditions largely influence the later life course, we know much less about the circumstances under which early developments are reversed. Genetically informed research can build hypotheses referring to not only the cumulation of advantage or disadvantage across different inequality dimensions like skill, material and nonmaterial resources, and health, but also on how shared and not shared genetic and social sources contribute to it. E.g., whereas correlations of 'good' genes with good family resources (double advantage) as well as 'bad' genes with bad family resources (double disadvantage) should reinforce continuity, negative correlations of this kind should destabilise it. But this may not be generally true. For example, genetic influences on characteristics considered negative and good family resources could motivate compensatory interventions to avoid downward mobility from the very beginning.

The impact of macro social forces

The bulk of gene-environment interaction studies deal with such proximate contexts as family environment, measured as socioeconomic status or family form and ethnicity. However, contexts shaping gene expression are located also at more distal levels: in neighbourhoods, educational and work contexts, and societal institutions. Up to now, these multilevel interdependencies have been researched little with respect to genetic influences. The proximate and distal levels do not work independently of one another but may constitute chains of risk generation and risk compensation over the life course. For example, the family context may trigger or exacerbate a genetic predisposition toward deviant behaviour that threatens educational success. Although this threat may be counteracted by mentoring programmes in schools, such programmes may fail to produce the desired long-term effects because the schools are situated in disadvantaged neighbourhoods. Thus, in sum, the extent to which genetic predispositions toward specific traits that may affect socioeconomic attain-

ment are expressed and actually affect the life course is shaped by the multilevel contexts in which individuals live, both simultaneously and successively (DIEWALD et al., 2015).

Though meta-studies show that there are large cross-national differences in the role of social interaction between genes and unequal resources for skill development or health, a systematic study of how different institutional regimes and policies influence the interplay between genes and the social environment to produce educational and status attainment is missing. Referring to state-of-the-art comparative life course research, bridging concepts are the shaping of individual life-course trajectories by (1) resource-rich versus resource-poor environments; (2) differences in the frequency of life course risks; and (3) the ways by which welfare-state institutions and policies seek to avoid adversity linked to these risks; (4) more or less tight structuring of educational and labour market institutions, with varying degrees of continuity and foreseeability, and different requirements for personal agency. Hypotheses about possible impacts of welfare state institutions on gene-environment interaction can address all types of gene-environment interaction discussed above: triggering (e.g., by differences in frequency of risky life events such as unemployment or divorce in different political economies), compensation (e.g., by differences in insurance-based risk compensation in case of these risks), social control (e.g., by differences in the relation of promoting and demanding, or rights and responsibilities, as precondition for welfare state support), and enhancement (e.g., by different functioning of educational systems). In a historical perspective the same desideratum can be formulated for the question of how large scale societal events such as economic depressions, wars, or political upheaval shape the role of genes for life courses, and how the responses and adaptations to these events vary with genetic predispositions (DIEWALD, 2015).

Challenges of genetically informed research for sociological concepts of environmental influences

Though the influence of the family of origin on offspring's life course outcomes is among the most researched phenomena in the social sciences, there are still highly relevant uncertainties about the mechanisms through which this influence comes about. Most challenging is the fact that even considering family influences on education and training, there is still a persistent direct influence of the family of origin on status attainment. This raises the question of which circumstances contribute to differences within families or tend to make siblings more similar than non-family members. There are considerable differences between common sociological and behaviour genetic views. Integrating the conceptualisation of family-of-origin influences conceived as total family effect comprising parental education, income, and occupational status in sociology with conceptions of sources of individual differences in inequality-relevant personal characteristics from behaviour genetics will enlarge our understanding of the persistent, though varying across nations (JOHNSON et al., 2010) and historically changing influence of social origin on social mobility. Sociologists tend to assume that family of origin has almost uniform influences on offspring's life chances: individuals are born as blank slates for which resourceful, better-equipped family environments uniformly create better chances for attainment. In contrast, behavioural genetic research shows that a shared environment is not important at all for many personal characteristics assumed to mediate the impact of social origin on offspring's life chances, and that for education shared environment is nevertheless substantial, but nonshared environmental influences and genetics play an important role as well. Moreover, common indicators of social origin like maternal and paternal education and occupation, and household income explain only a modest portion of the shared environment effect. Twin-based research designs with extensive environmental measures are better-suited to isolate not only genetic but also social influences contributing to within- and between-family inequality. Together with the fact of ubiquitous gene-environment covariance in within-family relations and interactions we face a very

complex situation where truly exogenous influences are hard to identify and a single SES indicator hardly captures the social origin influences to a sufficient degree.

However, it would be misleading to regard genetically informed research only as a methodological threat for sociological social inequality research. Thinking in mechanisms of gene transcription and gene-environment interaction can also stimulate and enrich sociological concepts of how social environments 'work', and why there is heterogeneity in response to these environments. Though also in genetically informed studies usually simple environmental measures of, e.g., SES, are applied, the mechanisms of gene-environment interaction displayed above call for more complex environmental measures that contain information about enhancing as well as stressing conditions that occur at the same time in life. This is not completely new for sociology. We know that upper positions in society are defined by different kinds of resources: material and cultural ones, and among material resources wealth must not have the same influence as earnings. And among cultural ones there are differences between more embodied cultural habits and know-how and more symbolic educational certificates or knowledge of highbrow culture. Though, as resources, they all are assumed to foster enhancement, the mechanisms linked to them may operate differently in different societies.

More importantly, stress and enhancement can occur simultaneously in the same environment or at the same time in different life domains. Again we know that, more as a rule than as the exception, attaining educational certificates, occupational status, authority, or income is linked to effort, and this necessary effort may be experienced as (positive) stimulation as well as (negative) stress. For labour market participation especially the concept of the employment relationship, the resources-demands approach, and the inducement-contribution theory combine an inventory of resources or gratifications received at the workplace with an inventory of the efforts and strains that have to be delivered to obtain these gratifications. In other words, resource-rich environments do not only foster enhancement but are often in the same time environments that may trigger stress-diathesis. As another example, similar considerations can be applied to social relationships with partly supportive and partly demanding facets.

If we are interested in why different individuals profit or suffer from the same environment it is not only necessary to understand their genetic makeup but also the complex, multi-layered character of the environments they experience synchronically and diachronically. By now this is well acknowledged by geneticists. So in several meetings of our research group PETER VISSCHER postulated a "matrix" of environmental characteristics parallel to the complexity of the gene matrix, and ERIC TURKHEIMER called for "EWAS" complementary to the progress in "GWAS" (genome-wide association studies). Therefore, genetically informed research is not only a peril to social scientists but also an opportunity to bring in their expertise and not least a chance to re-think their concepts, especially in the light of individuals' heterogeneous responses to social environments.

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