

Diewald, Martin

Article — Published Version

How Can Genetically Informative Research Contribute to Life Course Research?

KZfSS Kölner Zeitschrift für Soziologie und Sozialpsychologie

Provided in Cooperation with:

Springer Nature

Suggested Citation: Diewald, Martin (2024) : How Can Genetically Informative Research Contribute to Life Course Research?, KZfSS Kölner Zeitschrift für Soziologie und Sozialpsychologie, ISSN 1861-891X, Springer Fachmedien Wiesbaden GmbH, Wiesbaden, Vol. 76, Iss. 3, pp. 491-524, <https://doi.org/10.1007/s11577-024-00969-9>

This Version is available at:

<https://hdl.handle.net/10419/315451>

Standard-Nutzungsbedingungen:

Die Dokumente auf EconStor dürfen zu eigenen wissenschaftlichen Zwecken und zum Privatgebrauch gespeichert und kopiert werden.

Sie dürfen die Dokumente nicht für öffentliche oder kommerzielle Zwecke vervielfältigen, öffentlich ausstellen, öffentlich zugänglich machen, vertreiben oder anderweitig nutzen.

Sofern die Verfasser die Dokumente unter Open-Content-Lizenzen (insbesondere CC-Lizenzen) zur Verfügung gestellt haben sollten, gelten abweichend von diesen Nutzungsbedingungen die in der dort genannten Lizenz gewährten Nutzungsrechte.

Terms of use:

Documents in EconStor may be saved and copied for your personal and scholarly purposes.

You are not to copy documents for public or commercial purposes, to exhibit the documents publicly, to make them publicly available on the internet, or to distribute or otherwise use the documents in public.

If the documents have been made available under an Open Content Licence (especially Creative Commons Licences), you may exercise further usage rights as specified in the indicated licence.



<http://creativecommons.org/licenses/by/4.0/>



How Can Genetically Informative Research Contribute to Life Course Research?

Martin Diewald 

Received: 28 July 2023 / Accepted: 25 July 2024 / Published online: 16 September 2024
© The Author(s) 2024

Abstract Genetically informative studies have established a new research field that crosscuts disciplinary boundaries within the social sciences, as well as between social science and biology, with proprietary aims and research questions. This happens, however, at the cost of appropriate integration into the current theoretical and conceptual streams in the social sciences, e.g., sociology. That such a fruitful integration is possible is demonstrated for the case of life course research. The focus in dominantly, though not exclusively, on sociological concepts of the life course. This article first introduces central concepts of genetically informative research and life course research and then discusses possible ways to integrate genetic information into the life course research agenda, giving a brief overview of the main methodological tools available.

Keywords Behavioral genetics · Sociogenomics · Epigenome · Life course · Family of origin

Wie kann genetisch informative Forschung zum besseren Verständnis von Lebensläufen beitragen?

Zusammenfassung Genetisch informative Forschung hat einen deutlichen Aufschwung genommen und etabliert sich als eigenes Forschungsfeld zwischen den verschiedenen sozialwissenschaftlichen Disziplinen und der Biologie. Dabei verfolgt sie zunehmend ihre eigenen Ziele und Fragestellungen. Dies geschieht jedoch auf Kosten einer fruchtbaren Integration in die theoretischen und konzeptuellen

✉ M. Diewald
Faculty of Sociology, Bielefeld University
Universitätsstraße 25, 33615 Bielefeld, Germany
E-Mail: martin.diewald@uni-bielefeld.de

Entwicklungen innerhalb der sozialwissenschaftlichen Disziplinen. Dabei ließe sich leicht eine bessere Integration in vorhandene zentrale Konzepte herstellen. Dies wird am Beispiel der Lebenslaufforschung demonstriert. Der Fokus liegt hauptsächlich, aber nicht ausschließlich auf soziologischen Herangehensweisen. Der Beitrag führt zunächst in zentrale Konzepte sowohl der genetisch informativen Forschung als auch der Lebenslaufforschung ein. Darauf folgt eine Diskussion verschiedener Möglichkeiten, wie genetische Information zentrale Konzepte und Forschungsfragen der Lebenslaufforschung befruchten kann. Eine Übersicht über die methodologischen Herangehensweisen genetisch informativer Forschung rundet den Beitrag ab.

Schlüsselwörter Lebenslaufforschung · Soziogenetik · Verhaltensgenetik · Epigenetik · Herkunftsfamilie

1 Introduction

In the perspective of the social sciences, life course research belongs to the research fields that are most open for interdisciplinary cooperation, including biology. Nevertheless, genetic contributions to understand the life course have not yet found much interest. The main purpose of this article is to fill this gap in a specific way: to inform about the possibilities of genetically informative research to deal with research questions raised in the social sciences, foremost sociology, in the field of life course research.

After long resistance, there are more and more genetically informative studies¹ being published in social science journals. However, this does not yet mean that such studies have already found their proper place in the social sciences in general and in sociology in particular. There is no doubt that all characteristics sociology is interested in are also influenced by genetic contributions,² whether health, skills, attainments, or whatever else. However, identifying correlations between genes, or interindividual genetic variation, on the one side and phenotypic characteristics on the other side does not yet mean that this correlation is causal. What we see in human characteristics and behaviors results from both genetic variation (“nature”) and experiences made in different social contexts and the respective living conditions (“nurture”) and cannot be reduced to only one of these sources of development. In other words, the outcomes that sociology is interested in are neither purely social nor purely genetic, and a first question is to what degree both nature and nurture are relevant. There is no general answer to this question because the relative shares differ from characteristic to characteristic (Polderman et al. 2015) and vary across populations, be it between groups within a society or between different societies

¹ This term refers to studies that, either by twin or adoptive studies or by direct measurement of (parts of) the genome and/or the epigenome, or both, try to identify (epi)genetic influences on phenotypic characteristics and behaviors.

² Therefore, I apply the most cautious term, “contribution.” If appropriate when citing thoughts and hypotheses about genetic contributions to behaviors and characteristics, I use the term “influence” but avoid the term “causal” (see end of Sect. 4).

across time and space. So far, it is widely accepted that genetically informative designs are a helpful tool to cope with unobserved heterogeneity and that it is important to estimate the degree to which phenotypic correlations are biased by genetic confounding.

Separating genetic from environmental influences can become hugely important for answering core sociological research questions, not least about mechanisms creating social advantage and social disadvantage. That concerns about genetic instead of social mechanisms were already raised a long time ago was recently noted by Mills (2022). For instance, Conley et al. (2015) found that the relation between parental and offspring education was up to five-sixths social inheritance, but one-sixth could be traced back to genetic transmission. Social inheritance largely outdoes genetic inheritance. Otherwise, a claimed dominance of genetic influences could have been true, say five-sixths genetic inheritance and only one-sixth social inheritance. Especially in heated debates about the pros and cons of compensatory social policy, it can become important to disentangle the degrees to which social advantage and disadvantage are shaped by genetic variation or variation in environmental conditions. This is not to move in the direction of genetic determinism. Rather, genetically informed approaches can help to estimate the expected effectiveness of institutions and policies. For example, short-time training of cognitive ability proved to be ineffective in the longer run due to a pronounced fade-out effect after the end of the intervention (Protzko 2015). Given the paramount relevance of long-term gene–environment interplay for cognitive development, this had to be expected from the very beginning (Dickens and Flynn 2001). Only steady stimulation has the potential for sustained improvement of intelligence (see Sect. 3 for more elaboration on this point).

Contemporary research goes regularly beyond considering the relative impact of genetic versus social variation alone. It focuses on the various forms of gene–environment interplay and the processes that mediate the relation between the genome and the social phenomena over and above additive contributions. These mechanisms encompass, on the one hand, gene–environment interaction ($G \times E$) and, on the other hand, gene–environment covariation (rGE). A $G \times E$ interaction reflects either changes in environmental contributions occurring with genetic variation or changes in genetic contributions occurring with changes in environmental contexts (Selita and Kovas 2019; Branigan et al. 2013; Tucker-Drob and Bates 2016). For example, in the first case we can think of genetic predispositions affecting a person's sensitivity to environmental circumstances, leading to differences in how individuals react to, for instance, stressful environmental conditions (Plomin et al. 2012). In the second case, we can think of environmental conditions providing different opportunities to the realization of genetic potential and risk. The other form of gene–environment interplay, rGE, means that environmental influences are not always independent of the characteristics and behaviors of an individual and that the latter are shaped to some degree by genes. Consequently, in such cases, the environment is not completely exogenous to an individual's genome but is geneti-

cally conflated, and estimating the additive contributions of genes and environment may be biased.³

Researchers in the field of genetically informative research see themselves firmly as social scientists. Comparably few come from sociology. Aside from economists, demographers, and political scientists, psychologists also often see themselves as social scientists (e.g., Turkheimer 2011; Harden 2021), though they are usually not trained in sociological theories about the life course, social inequality, or social mobility. For all, the core task remains the same: to separate genetic from environmental contributions and study the interplay between the two in the emergence of the phenotypes of interest and their development, and to integrate them into explanatory models. Such study interests do not threaten sociology (or other social sciences) but create a new field for sociological expertise to understand the mechanisms behind phenotypes of highly sociological interest—such as income, status, education, skills, and demographic life events—and the role of genetic variation thereby. Given the rise of genetically informative research, sociologists should not miss “the opportunity to affect the precision and direction of this burgeoning field exactly when their expertise is needed most” (Braudt 2018, p. 2). Genetically informative research also needs expertise in the theory-driven phenotypic modeling of social explanations in the fields of social differentiation and social inequalities, such as models of status attainment, family formation, or the mechanisms of intergenerational transmission of advantage and disadvantage. This also applies to the degree to which phenotypes as predictors in multivariate analyses are genetically confounded, e.g., skills as predictors of educational achievement (e.g., Krapohl et al. 2014). To fully understand the pathways of how an outcome like education is achieved, the predictors also have to be included in partitioning genetic and environmental contributions.

Less clear, however, is how genetically informative research fits into sociological explanations in a way that it can be integrated into the scientific agenda and theoretical body of sociological thinking about how social mechanisms work. This question is far from trivial. There is not a traditional place for the role of genes in the development of the sociological agenda, nor has genetically informative research taken much notice of sociological thinking (see, for example, Plomin 2019). The participation of sociologists has been sparse, with psychologists having a much longer and larger involvement. Even some of those sociologists working successfully in the field are skeptical about a fruitful combination of social science genetics with sociological theories (Fletcher 2023). These shortcomings might frustrate a deeper interest of sociologists in this now rapidly developing field that looks at first quite unfamiliar to them. In consequence, there is not enough motivation for the acknowledgment and integration of research results from genetically informative research in the sociological mainstream.

This paper aims to demonstrate how genetically informative studies can add to and be integrated into life course research as a complex research agenda in sociology informed by several theoretical approaches. Some of them are dominantly sociological, others are more psychological, and some are shared by both disciplines.

³ See Verhulst and Hatemi (2013) for different directions of how this may come about and how relevant distorted estimates might be.

The study of the life course is not “owned” by sociology alone but was from the very beginning an interdisciplinary endeavor in the behavioral sciences. It is not too simplified to see a psychological approach of individual development to a psychic system on the one side and a sociological approach of a social being embedded in a multilayered social environment, from family to state institutions, on the other side. For a long time, these two perspectives were the main drivers of life course research. Following an increasing interest in health issues (Mayer 2009; Hoffmann 2023), the insight that the organism also contributes to understanding of the life course was later included in life course theory (e.g., Kuh et al. 2003). This was not only an additive extension but refers especially to how the organism interacts with psychic and social developments and therefore helps in the understanding of development on both levels. Consequently, an account exclusively drawing on a purely sociological perspective of the life course is difficult to pursue. Moreover, theories in the domain of life course research are comprehensive, since the life course is a frame for many substantive topics and research questions. This allows for a similarly comprehensive overview of different aspects.

Since DNA is fixed and largely immutable from the very beginning of the individual life course, any idea about how genetics⁴ could enrich the study of the life course may seem rather limited. However, as will be demonstrated in the following, longitudinal designs in behavioral genetics and molecular genetics as well as epigenetics can enrich several central concepts of life course research. Before these specific contributions to mainly sociological life course research are demonstrated (Sect. 3), I first give an overview of central terms and definitions relevant here: life course, genes, and mechanisms of gene–environment interaction and covariation (Sect. 2). After these mainly conceptual elaborations, Sect. 4 provides a brief and not exhaustive overview of the major challenges and possibilities of three different methodological approaches to study the interplay of genetic and environmental influences over the life course: twin-based studies, molecular genetics, and epigenetics. For molecular genetics, solely polygenic scores as the by far most often applied approach is considered. The overall goal is to demonstrate the specific capacities of these approaches for identifying genetic contributions to the life course, in addition to and intersecting with environmental forces. The conclusion summarizes successes and problems in existing research and then moves to prospects for genetically informative research in sociology for the near future.

To exemplify the respective contributions, I mostly refer to processes of educational attainment and achievement, since education plays a large role in genetically sensitive research, with many studies available for both twin-based and molecular genetic methodology. This focus also includes the individual development of cognitive and other skills as theoretically and empirically important contributors to education. It allows us to demonstrate that patterns of longitudinal gene–environment interplay differ between phenotypes under consideration, even within this field. Given the overall still scarce longitudinal, life course–oriented, genetically informed studies, it would be too ambitious to extend the overview to more phenotypes in other

⁴ To be sure: When looking at genes in this paper, genetic information always relates to genetic information as a population parameter and not to the individual genetic makeup and related questions.

life domains. Therefore, any generalizations of the patterns found here to other life domains are not intended. Also, I restrict my considerations to contributions to primarily sociological concepts of life course research and do *not* intend to additionally cover individual development over various life phases as a whole in genetically informative studies. Such an endeavor would largely overexpand the narrative and what is possible in an article like this one.

2 Genes and the Life Course: Definitions and Mechanisms

In this section, some central definitions and mechanisms are introduced. This is not only to inform readers not yet familiar with genetically informative research or contemporary life course research, but also to avoid potential misunderstanding, as there is a lack of universally shared understandings of some definitions. To start with the term “mechanism”: Broadly speaking, referring to mechanisms means that “explanations should reflect the causal processes actually responsible for the observations” (Hedström and Ylikoski 2010, p. 64). This implies considering also those “cogs and wheels” (Hedström and Ylikoski 2010, p. 54), if they are causally relevant, that are outside sociology’s classical scope.

2.1 What Genes Do and Do Not Do

There are many definitions of what genes are. For our purposes, it may be enough to keep the following in mind: Genes are units of DNA that contain fundamental information for the organism to develop characteristics of an individual. Some of these genes are relevant for characteristics that differ among individuals, e.g., personality, skills, height, diseases, and many others, depending on how specific genes are coded. To contribute to the development of specific characteristics, the DNA has to be transcribed to RNA. The RNA transmits the information for building proteins, which are biologically active and inform the different types of cells how to work in a specific way, i.e., genetic functions can be switched on or off, diminished or enhanced. These processes are called gene regulation.

The DNA is immutable from conception on. It does not change over the life course aside from a very limited number of accidental mutations. Nevertheless, genetic variation can directly influence life course developments, e.g., the onset of puberty (Mancini et al. 2022), aging (Melzer et al. 2020), and learning (Plomin and Kovas 2005).

Gene regulation is not immutable over the life course. Quite the contrary, experiences over the life course, including changes in daily life as well as single events, can affect gene regulation and become embodied in the form of epigenetic markers, which in turn can have enduring influences on physiological development as well as behavioral patterns (Diewald et al. *in press*). Experiences from conception on may lead to changes in the epigenome (e.g., Cao-Lei et al. 2020). Such changes are not irreversible but rather stable. It is still discussed whether the epigenome is even heritable in humans and can be transmitted over generations (Ghai and Kader 2022).

Several biological mechanisms can lead to such changes in gene regulation. Among them, chemical modification of the DNA, called DNA methylation, is the most important one. The so-called epigenome contains information about cellular modifications in the functioning of the genome without changing the DNA itself. These modifications mirror intrinsic biological programming processes as well as modifications due to the accumulated effects of the environment. Epigenetic markers identify the loci where this happens. The increasing focus on gene regulation and epigenetics marks a shift in the conceptualization of genes as fixed biological “equipment” to a more fluid understanding of genetic influences (Turner et al. 2020). It provides a direct link between genetic variation and social experiences over the life course as a biological outcome of gene–environment interaction (see Sect. 2.2). Gene regulation makes evident why DNA is influential but far from deterministic for shaping the life course.

2.2 Genes in Social Contexts: Mechanisms of Genetic Influences

Aside from uniform, additive effects of genetic variation, genetic contributions are moderated and mediated by social contexts in various ways. The mechanisms explaining how this happens can be distinguished in gene–environment interaction and gene–environment correlation.

2.2.1 Gene–Environment Interaction

Gene–environment interaction ($G \times E$) has two variants. In one variant, influences from the environment are moderated by genetic variation. In the other variant, the influence of genetic variation is moderated by characteristics of the environment. Overall, the first case has found much less attention in research than the second one. For this type of $G \times E$, the possibility that an influence of the social environment is moderated by genetic variation, *environmental sensitivity* (Pluess 2015) has emerged as a concept of how individuals react to good or bad living conditions. Other than the juxtaposition of being either vulnerable or resilient, environmental sensitivity means that some people have a generally elevated openness to environmental influences, which means that they both suffer more severely from bad living conditions and strains than others, as well as profit more than others from good conditions and stimulation.⁵ Genetically, environmental sensitivity is predominantly based on genes that regulate the immune system and stress. Methylation of these genes plays a decisive role in which direction the sensitivity is more pronounced, in the direction of either resilience or vulnerability (Daskalakis et al. 2021; Heim and Binder 2012; Yehuda et al. 2016).

For the more frequently investigated second case of the environment moderating the influence of genetic variation, Shanahan and Hofer (2005) have proposed four types: *Triggering*, also referred to as the diathesis–stress model (Broerman 2020), means that a person has a genetic vulnerability that is expressed only in specific

⁵ This is a partially different viewpoint of categorizing environmental sensitivity than that of Mills (2022), who understands environmental sensitivity as a variant of diathesis–stress.

social situations. Here the social context is detrimental and triggers the occurrence of genetic risk. This is surely the most frequently studied type of $G \times E$ in genetically informative research, which might be due to diseases and psychological disorders being important topics (Polderman et al. 2015). The second type, *social compensation*, refers to the opposite: There is a genetic vulnerability, but the social context is helpful and hinders the expression of a genetic risk. For example, genes for aggressive behaviors can be dimmed for individuals growing up in intact families with warm relationships. Note that in both the first type and the second type, a labeling of certain genetic predispositions as bad or unwanted is the starting point. However, what is unwanted or seen as “bad” is not an objective classification of genetic predispositions but is subject to evaluations in the society. The effect of genes is always contingent on the social context: An advantage under certain conditions may be a disadvantage under others. For example, a predisposition for aggression can lead to aggressive behaviors, which then lead to criminal behaviors and consequently to prison.⁶ But for higher social classes, some cultivated kinds of aggressive behaviors, combined with polished good manners, could help the individual enter higher social ranks in highly competitive career pathways. A third type is called *social control*. For it, too, the starting point is a genetic predisposition for unwanted behaviors such as drug use or externalizing. The mechanism here is a restrictive social environment limiting individual behaviors by supervision through parents, neighbors, mentors, policing, or strict societal norms. The fourth type, *enhancement*, is also referred to as the bioecological model (Bronfenbrenner and Ceci 1994). In contrast to the other three types, the starting point is desirable behaviors or outcomes, e.g., self-control, cognitive ability, or higher education. Enhancement describes a social context that accentuates the effect of a genetic predisposition toward socially valued characteristics or behaviors, which is most likely reached via resource-rich environments.

Whereas environmental sensitivity goes beyond vulnerability and resilience as well-known phenotypic concepts in life course research, it is obvious that all four types of environments moderating genetic influences fit standard sociological thinking. However, whereas in genetically informative research the dominant focus is on stressors that trigger a genetic predisposition for unwanted characteristics and behaviors, in sociological thinking about inequality the dominant focus is clearly on the positive role of resources, as in the enhancement type of $G \times E$.

Better understanding of the social mechanisms at play in any of these types, however, requires additional theoretical work that engages with the link between genetic variation and environment on a more detailed and specific level. The first point is to define a resource-rich environment in terms of different resources with a specific impact on the characteristics of interest. What is called socioeconomic status is a mix of several conditions that do not necessarily have the same influence on the development of a specific outcome. Consequently, instead of a composite measure of socioeconomic status, it should be tested which one of the resources linked to socioeconomic status leads to an effect: money in the household, parental occupational status, education, skills, or the absence of stressors (see Mönkediek et al.

⁶ This is an adage used by Conley (2009) to exemplify the context-dependence of social influences filtering genetic influences.

2023 for such an exception). The statistical influence of these resources is closely linked to different social mechanisms at work when creating social inequalities (Tilly 2003; Diwald and Faist 2011). It might not only be interesting whether a higher occupational status matters, but it might also link higher social status or a higher income to societal processes of social closure, opportunity hoarding, or exploitation creating these differences in income and status (e.g., Bol and Weeden 2015). Also, in international comparisons the more or less compensatory role of the welfare state could be of interest.⁷ A lack of material resources, e.g., poverty measured via equivalent household income, may not only have material consequences but may also result in nonmaterial harm in the form of stereotyping and discrimination by others or more within-household conflicts. Moreover, it should be taken into account that these four types (triggering, enhancement, social compensation, social control) may not work independently from each other but are intertwined. According to the resources–demands approach as well as the inducement–contribution approach, higher resources linked to occupational status might be linked to higher strains as well and trigger vulnerabilities despite protective resources (Schieman et al. 2006). At the same time, protective resources linked to higher status may compensate for these stressors. As a consequence, it is an open question whether higher occupations, mostly measured by status scales, are indeed a resource for enhancement or a trigger for stress–diathesis, or both.

2.2.2 Gene–Environment Correlation (*rGE*)

Environments are not always exogenous to individual behaviors. Genetic variation and variation in environments may covary because genetic variants influence the exposure to certain environments and because behaviors relevant for exposure are genetically influenced. There are three ways of how *rGE* may come about (Plomin et al. 1977). First, passive *rGE* may arise from the fact that genes are transmitted from parents to children. Therefore, the parental home environment is shaped by the same genes that are also the genetic makeup of the child. Evocative or reactive *rGE* occurs when an individual's genetically influenced characteristics and behaviors evoke reactions from others. Active *rGE* refers to the fact that social contexts are not always simply given but are selected by individuals who seek to live in environments that fit their preferences, which are to a smaller or larger extent genetically predisposed. Similarly, exposure to risk can stem from individuals selecting themselves into high-risk environments (Kendler and Karkowski-Shuman 1997). In all these cases, environmental measures are to some degree genetic in origin.

When investigating the relative share of genes versus environment or gene–environment interaction, the basal precondition is that both sources of variation are exogenous to each other and not conflated. It is therefore advisable to test whether this is indeed the case before calculating $G \times E$.

⁷ In their study on social origin moderating the genetic influence on IQ development, Tucker-Drob and Bates (2016) speculated about such a moderating role of different welfare state regimes.

2.3 Life Course Research

The term “life course,” or “life course research,” denotes not a single theory but is an umbrella term that covers several heterogeneous theoretical ideas about how life develops, along with methodological approaches used to study them. Although the study of the life course was an interdisciplinary endeavor from the beginning, the different disciplines involved followed different interests and focused on different topics. The sociological view on the life course is dominated by following individuals through the institutions and the social structure of societies, i.e., the different trajectories and events in different life domains (Elder and O’Rand 1995, p. 454), as well as social inequalities. These trajectories and events can be interrelated, sometimes longitudinally, as in the case of occupational careers preceded by educational careers, or cross-sectionally when, for example, insecurity in labor force participation and insecurity in close personal ties are interrelated. However, not only do the events and trajectories themselves define the individual level in life course analyses, but the benefits and burdens linked to them are important, since they are drivers for decisions to stay or switch participation in different life domains.

As international comparisons have convincingly demonstrated, the patterns of interrelated pathways through life domains reflect different institutional characteristics of societies. These patterns are also influenced by other macro conditions such as wars, economic cycles, and pandemics.⁸ Such a reference of individual-level processes and outcomes to influences from supra-individual levels is standard in sociology. However, individual-level processes also display individual behaviors that are not uniformly shaped by supra-individual opportunities and constraints but are shaped to some degree by individual agency directed by preferences, perceptions, and decision-making. Individual agency is a central idea for understanding the interplay between life domains and processes at the different levels and their interdependence. Central to the sociological understanding is the idea that agency is bound not only to present life conditions but also to experiences in the past and expectations about the future (Bernardi et al. 2019). The importance of this concept is illustrated in a definition of the life course by Giele and Elder (1998, p. 22) as “a sequence of socially defined events and roles that the individual *enacts* over time” (italics added by me).

Following some classical overviews (e.g., Elder and O’Rand 1995; Mayer 2015), overarching research questions at the level of behaviors and outcomes of behaviors are as follows: (1) How decisive is the family of origin for later life, and why; (2) what role does individual agency play in coping with environmental opportunities and constraints; (3) how are parallel behavioral processes in work, family, leisure, and other domains interrelated; (4) how do stability and change come about over the life course, not least concerning unequal life chances; (5) how do differences in macro social conditions shape life courses differently across time and place; and (6) what role do significant others play in our lives?

It is a result of the interdisciplinary openness of life course research that answers and explanations for these questions are not limited to the level of behavioral

⁸ The same applies to meso-level constructs such as organizations, neighborhoods, and social networks.

processes and resulting outcomes, as well as supra-individual contexts alone. Aside from being social actors, individuals also have a mental system, which is the main focus of psychological interests. Mental states and processes are interrelated with behavioral states and processes (Diewald and Mayer 2009). Therefore, the explanation for biographical developments may also include mental states and processes. Recently, awareness has risen that for understanding life courses and individual development, one needs to complement these two levels of processes with an understanding of the characteristics and the development of their organism.⁹ Processes in the organism are interrelated with the former two levels and, thus, can also contribute to behavioral and mental processes. This multilevel interdependence of the life course is constitutive of newer conceptualizations of the life course (Bernardi et al. 2019).

3 Perspectives on Integrating Genetic Influences into the Conceptual Framework of Life Course Research

When looking at possibilities to integrate genetic influences into life course research, these classical overarching research questions provide an insightful starting point. However, in the following I slightly deviate from this list in two respects to highlight recent contributions of genetically informative research. First, mechanisms of cumulative advantage and disadvantage are part of considering stability and change over the life course, but they received their own subsection to demonstrate the influence of gene–environment interplay for a research field combining the life course approach in general with studies of social inequality. Second, I added a subsection on how integrating new views on life phases, in which institutions play a lesser role than in other views, nevertheless provides a better understanding of research topics of high relevance in sociological life course research. Finally, I omitted a subsection on significant others. The reason is that there is slowly growing research on genetic confounding of the role of significant others, but to date has little, if any, embedding in life course research.

3.1 What Is the Relevance of the Family of Origin?

Parents not only use their resources to support their offspring's attainment and well-being but also transmit their genes to them. The latter is neglected if “social” origin alone is taken as the starting point of the life course. We have two starting points—the social conditions relevant to growing up in a family, and the DNA. The DNA sequence is fixed at conception except for a few mutations. Because of this, inequalities exist among individuals from birth not only in their social origin but also in their genetic endowments. From the previous depiction of gene–environment interplay, we know that social and genetic origins are already confounded at birth. In addition to the traditional components of social origin such as parental education, occupation, and income, some studies also examine parental cognitive ability and

⁹ For the case of inflammation, see Landecker (2024).

other skills. In this latter case, the conflation becomes a bit easier to trace. Marks and O'Connell (2023) investigated the link between socioeconomic status variables and a child's cognitive ability and school achievement. They found that parental ability was an important mediator in this correlation, with substantive genetic contributions to the parental ability mediators. From this viewpoint, any correlation between parents' social resources and children's characteristics may be due to the role of cognitive ability and other skills rather than to a causal impact of parental resources. In other words, when influences of social origin are investigated in a genetically noninformative design, social origin effects are genetically conflated to an unknown degree. Genetic information is therefore an important contribution to the basic question of the degree to which our lives are predetermined by the family in which we are born. Consequently, relying on a causal impact of parents' social, economic, and cultural resources alone seems to be a poor concept for capturing the "luggage" with which we start our lives. This "luggage" also includes the genetic variation we inherit from our parents. A major challenge is to disentangle these intertwined influences, to separate the social from the genetic transmission, and thereby to avoid bias in phenotypic correlation caused by rGE.

In all of this, as should by now have become quite clear, there is genetic determinism. Rather, genes set an upper and a lower boundary of a developmental range, with subsequent experiences driving the development in one or the other direction. Also, the understanding of genes as "fluid" in the sense of changes in the epigenome is relevant to defining the family of origin as the starting point of the life course. Experiences in the parental home contribute substantially to changes in the epigenome. Especially between conception and the very early years of life, and again during puberty, the organism is wide open to environmental influences. Moreover, these early changes seem to be quite stable over the life course (Danese and Lewis 2017; Turner et al. 2020). Epigenetic markers make evident that already before birth, in the womb of the mother, the genetic makeup interacts with the environment, with often long-term consequences for later behaviors, health, and social advantage and disadvantage (Mastrotheodoros et al. 2023; Raffington et al. 2023; Diewald et al. *in press*).¹⁰ Epigenetic markers are foremost influenced by the mother's exposure to stressors and the amount of resources to cope with them (Li et al. 2022; Cao-Lei et al. 2020), and in early years of life with the amount of resources available in the household (Needham et al. 2015).

How to interpret a higher or lower influence of (epi)genetic contributions as part of the family of origin influences is debated. Sociologists have tended to interpret a high relevance of genetic contributions as "opportunity for achievement" (Nielsen 2006, p. 193) or openness of the opportunity structure: "Favorable environments, permitting fuller expression of potential, are characterized by high heritability. Unfavorable environments, inhibiting expression of native talent, are characterized by low heritability" (Nielsen 2006, p. 198). There are fewer barriers to exploiting one's genetic potential. However, it can also mean more exposure to a genetic vulnera-

¹⁰ The crucial role of prenatal experiences is also discussed in life history theory as part of the adaptation to changing living conditions, preparing the unborn child via gene regulation for what follows after birth (e.g., Coall et al. 2015).

bility because sheltering or compensating social mechanisms are missing. However, Diewald et al. (2015) and later Harden (2021) challenged this viewpoint. It can be argued that individuals cannot be held responsible for their genes. Why should social origin, but not genetic origin, be interpreted as social closure? Is the opportunity structure more open if life chances are largely inscribed in the genes rather than shaped by parental resources? It seems reasonable to consider genetic predisposition, along with parental resources, as the main source for social closure in a society.

However, several international comparative studies indicate that higher genetic contributions to educational or occupational attainment are correlated with more open institutional arrangements and a higher amount of social mobility (Selita and Kovas 2019; Engzell and Troup 2019). A possible explanation could be that the genetic lottery involving the combination of the father's and the mother's genes—children inherit half from both parents—reshuffles these genes in the genetic makeup of the offspring. This results in genetic potentials that are not merely a copy of the parents' but rather are new combinations of genetic potentials and risks. The less that genetic assortative mating occurs, the more diverse these combinations become. These new mixes are a source of social mobility. Therefore, including genetic inheritance as a starting point of life is more than just avoiding genetic conflation of social origin; it has substantive research potential in addition to the study of social inheritance.

3.2 Agency as a Core Concept of Actively Shaping Life Courses

In life course research, a counterpoint to the formative power of social inheritance and social conditions is the role of individual agency. There are many concepts related to agency, all centered on the core idea that individuals have a choice in how they react to their experiences and environments. Whether these reactions in the end effectively and efficiently serve their preferences is an open question, as is the question of whether these reactions are based on conscious decisions or are routinized behavioral patterns. Adhering to the idea of individual agency inherently limits the acceptance of genetic determinism in explaining behavior. Nevertheless, genetic information can add to a better understanding of why individuals' behaviors may deviate from effectively pursuing goals that are good for them and fit their preferences. Phenotypic characteristics well-known for deviating from rational behaviors are, for example, lack of cognitive ability, lack of self-control, or kinds of externalizing behaviors. All of them have sizable genetic sources to varying degrees (Polderman et al. 2015). Knowing about these genetic sources adds to our understanding of the extent to which these characteristics are inherited aside from nurturing and, thus, to what degree behaviors are preshaped by genes that are out of our control. Moreover, the genetic overlap of characteristics and behaviors—a phenomenon called genetic pleiotropy—informs about mechanisms underlying observed correlations between phenotypes. As an example, Koenen et al. (2006) found considerable genetic overlap between low IQ and antisocial behavior. The phenotypic correlation was well-known and mostly attributed to environmental causes. However, the study identified that it was caused 100% by genetic overlap, i.e., the same genes linked to a low IQ were also linked to exhibiting antisocial behavior.

As noted previously, agency refers to the capacity to react to environments according to one's own preferences. There is an obvious correspondence to the active variant of rGE (see Sect. 2.2). Active rGE is defined as self-selection into environments that fit an individual's preferences, which are to a smaller or larger extent genetically predisposed. Therefore, the identification of active rGE can probably be seen as an indicator of agentic behavior. To what degree active rGE does in all cases indicate agency in the positive sense is, however, debatable. It may be appropriate for desirable outcomes, but in the prediction of unwanted outcomes, say, externalizing, active rGE would indicate choosing environments that are not helpful for realizing preferences, though it might nevertheless be an agentic behavior. It would help to learn more about phenotypic correlates of active rGE by combining the identification of active rGE with phenotypic measures of agency. However, I do not know of any study having done this.

The same is true for investigating whether different variants of rGE, active versus passive, are age-graded or more frequent for different life phases and, thus, inform about differences in agency between life phases. For newborns and toddlers, passive rGE should be especially important, since possibilities for actively choosing and influencing environments are limited. However, evocative¹¹ rGE could also be important, i.e., the capacity to evoke benevolence and stimulation from others. More active rGE should be expected from adolescence and young adulthood on, since choosing the right networks as well as institutions of education and training becomes more important. Again, evocative rGE is also expected to be relatively prevalent, since it is important for successfully navigating through education and training to influence gatekeepers positively. In middle and late adulthood, it can be expected that institutionalized career ladders, seniority rule, and long-enacted roles in business and personal life require comparably less active behaviors.

Genomic regulation can influence behaviors as well. Variants in key neuroregulatory genes, interacting with environmental experiences, can have large and enduring influences on behavioral differences. This applies, for example, to the often cited “fight-or-flight” response as an enduring pattern to cope with environmental challenges based on epigenetic regulation (Househam 2023). Epigenetic embodiment of former life experiences can explain why people react to stressors consistently in either the fight or the flight direction, with low awareness of situation-dependent risks and chances. The same demands are perceived either as a challenge and stimulation or, in contrast, as an overwhelming, unmanageable threat. The reason is former experiences of having been helplessly confronted with stressors, especially when experienced in utero and during the first years after birth when it is more likely that such experiences get under the skin in the form of changes in the epigenome. That former experiences are relevant for behavioral responses and decision-making thereafter is well-known in life course research as the “shadow of the past” (Bernardi et al. 2019). In the case of epigenetic embodiment, the programming follows a specific, organismic mechanism with a high probability that it will be enduring, insofar as it influences and will probably also include a “shadow of the future” such that it

¹¹ Evocative rGE can be seen as a variant of active rGE, insofar as it is a way of influencing the environment.

affects “people’s internal sense that they can influence their lives” (Hitlin and Kwon 2016, p. 432) and, consequently, how they shape their expectations, aspirations, and plans for the future.

3.3 Interdependencies Between Life Domains

In life course research, interdependencies between life domains are among the most addressed topics. In sociology, this is mostly done at the individual level of participation patterns in the different life domains of work, family, and sometimes activities outside these two life domains. These are often related to influences of the supra-individual level in the form of the social embedding in partnership, family, and nonkin networks (Elder et al. 2003). On the one side, international comparisons of the influence of welfare institutions, policies, demographic structures, and other macro conditions are addressed as the supra-individual level (Aisenbrey and Fasang 2017). On the other side, “internal” dispositions and mental as well as physical functioning influence how participation in different life domains is patterned (Bernardi et al. 2019). Especially for this internal level, genetics can help in tracing back why interdependencies between life domains at the phenotypic level of participation may occur. As an example, a much-researched topic is the hypothesis that an individual needs safety and trust in educational and occupational attainment to get into long-term, committed relationships, especially family formation, and that, vice versa, family commitments call for safer employment. This is plausible, but it could be that these correlations over time have a common source in a general predisposition of safety needs or risk aversion partly rooted in the genetic makeup or early experiences in the family of origin. In this case, the phenotypic correlation is spurious and not causal. In this direction goes the study of Tropf and Mandemakers (2017) on the relationship between educational attainment and fertility postponement. Though the choice of a genetically informed design for the study was mostly motivated by the suspicion of genetic confounding, the genetic overlap between the two phenotypes under investigation was not strong enough to make the correlation spurious, but in this case the unobserved experiences in the family of origin did.

That the same genes can influence the development of different characteristics was demonstrated by Belsky et al. (2016). What the authors called “success genes” predicted different behaviors in different life domains across the life course, from early acquisition of speech and reading skills through geographic mobility and mate choice and on to financial planning for retirement. Similarly, Demange et al. (2021) showed that education-related genes have an overlap with longevity-related genes. They also provide an explanation via cognitive and noncognitive skills. The genetic overlap between longevity and education is mainly due to genes relevant to skill development.

3.4 New Views on Life Phases

Including the organism as part of life course research directs the attention to life phases in which gene regulation plays a specifically large role. Whereas the DNA is largely fixed from conception on, gene regulation is a lifelong process. There are

quite detailed windows of risk and opportunity for specific experiences to become important for gene regulation. As already mentioned, this starts in utero, which adds to sociological life course research in several respects. First, it extends the window of observation from conception rather than birth. After conception, social influences become relevant, and due to the openness of the organism during pregnancy, they often have a large impact (Heim and Binder 2012), specifically the lasting effects of maternal smoking (Knopik et al. 2012). Second, experiences during this phase can leave their mark in the epigenome. These biological markers can inform about experiences during the prenatal time window for which we usually have little information aside from mother–child health records¹² and the mother’s memories. Due to this embedding in the organism, the DNA methylation signature is rather stable and can have an influence later on, with considerable phases of latency in between (Cao-Lei et al. 2014; Gaunt et al. 2016). For example, it could explain why in a study only perinatal experiences of poverty proved to be predictive for many detrimental health and attainment indicators around the age of 35 years, whereas such experiences later in adolescence and early adulthood were not (Duncan et al. 2010). Due to lack of appropriate data, the authors could not further explore the reasons for this difference. In a second article about the same data, the same authors speculated about biological imprints of prenatal and perinatal experiences as an explanation (Ziol-Guest et al. 2012).

A second difference often overlooked in sociological life course literature concerns puberty. Though puberty is a topic in sociological life course research, genetically informative research, especially in epigenetics, shows how complex and varied the timing of genetically regulated neurobiological processes associated with puberty can be. Discrete periods of sensitivity, times of heightened plasticity, are very specific for different brain functions (Heim and Binder 2012), and the respective windows of opportunity are considerably smaller than the age bracket usually used to define puberty as a life phase. In other words, to take the cross-level interplay of biological trajectories seriously requires a more finely graded differentiation of windows of opportunity than is covered by puberty age brackets.

These two life phases are highlighted because their relevance as the most sensitive life phases that are more open to biological influences than other phases is specifically neglected in sociological theorizing about the life course.

There are many more examples of how age differences are linked to genetic regulation and how genes become more important or lose importance over the life course. As an example, Haberstick et al. (2005) found that uncorrelated age-specific effects are relevant to change in phenotypic internalizing. At the same time, heritable contributions to phenotypic stability were identified as well. These were largely the same across middle childhood and early adolescence. However, as already clarified in the introduction, it would overstress the agenda of this paper to report in more detail about changes in gene–environment interplay across ages for various characteristics of individual development.

¹² In Germany, all pregnant women get a “Mutterpass,” which offers clinical examinations to ensure a safe pregnancy for mother and child. These maternity records contain the most important medical findings of fetal development during pregnancy.

3.5 Stability and Change over the Life Course

A crucial question in sociological life course research is which factors cause stability and change over the life course or, in other words, what drives the tension between path dependency and turning points. Path dependency goes beyond the recent past and instead focuses on the possible channeling of the life course through important earlier decisions (e.g., educational or occupational choices) or the more or less favorable conditions that were present when making such transitions. For example, wartime or economic crises prevent investments in educational and occupational careers, with long-term consequences for later life (Mayer 2015). Whereas path dependency refers to chains of experiences that are likely or foreseeable based on past experiences, turning points signify radical deviations or disruptions in an individual's trajectory. These are unexpected switches to a new path, whether due to personal decisions or external shocks at a societal level (Bernardi et al. 2019, p. 4). It is not the place here to discuss the multiple variants of how path dependencies and turning points can occur in more detail.

Genetically informative approaches can help us to better understand the mechanisms behind path dependence and turning points. Generally, life events are heritable like all other phenotypes. As Bemmels et al. (2008) have shown, most heritable are life events that occur through one's own initiative and behaviors, such as educational achievement. Most attributable to influences from the environment are life events shared by family members but not initiated by the respondent, such as parental divorce. Unsystematic environmental influences are the largest contributor to life events that are neither shared by family members nor initiated by the respondent. Over the life course, stability in phenotypic characteristics, and presumably also for path dependency as a pattern, is often due to stable genetic influence for most phenotypes.

The role of environmental influences or stochastic perturbation increases over the life course. The degree to which these influences provide plasticity in development or even trigger turning points is an open question and differs for different types of development. Educational achievement is an example of a development in which many institutional arrangements and reforms are launched to influence the development of school achievement positively. A study in the United Kingdom assumed that margins were limited. Overall, school achievement was highly stable. Individual differences in school achievement were to a high extent heritable (around 70%), even when intelligence was controlled for as the most important mediator for genetic contributions (and was then still 60%; Rimfeld et al. 2018).

The fact that heritability generally provides stability while the environment induces change does not preclude the stability of the degree of heritability itself. Heritability can vary across the lifespan due to different reasons: (1) because genetic influences are expressed differently at different biological stages of life (e.g., early childhood, pubertal changes, or old age); (2) because different contexts downgrade or enhance the degree of heritability; and (3) because the possibility for gene–environment correlation rises with age; when an increase in genetic contributions to phenotypes with age is often observed (Polderman et al. 2015), this relates to genetic confounding of environmental influences. In other words, genetic influ-

ences are amplified by correlated environmental influences. If this confounding is not explicitly modeled, but instead modeling comprises purely additive effects, rGE shows up as an increase in genetic influences (see Sect. 4).

Studying gene–environment interaction in longitudinal designs can help to detect not only whether stability or change characterizes phenotypic development but also the degree to which a genetic potential can be actually exploited or a genetic risk triggered, and when this happens. An exemplary research question regarding the exploitation of a genetic potential is how different educational tracks are not only selective for a different genetic potential but also exploit it differently. Educational transitions may (in this regard) lead to stability or change, if not affect turning points. An example of triggering a genetic risk is the relevance of stressful life events. There is abundant literature on which stressors are most important to trigger a genetic risk. These studies confirm that everyday experiences matter more than stressful life events, experiences during sensitive phases matter more than during others, nonnormative experiences matter more than normative ones, and violations through perceived discrimination, mortification, humiliation, or lack of respect matter at least as much as poverty and low socioeconomic status (Diewald 2023; Mullins et al. 2024; Goosby and Cheadle 2024).

For $G \times E$ in the form of changes in the epigenome as a composite of genetic and environmental influences, methylation levels are highly stable over the lifetime (Gaunt et al. 2016). Methylation variation increases over time, most likely due to increased environmental or stochastic influences of the same type as listed above. However, this does mean that changes in the epigenome immediately show up in phenotypic characteristics and behaviors. Influences on developments at the phenotypic level may stay latent over a longer period (Gaunt et al. 2016; Heim and Binder 2012). Therefore, the identification of pathways in life courses may be difficult to identify when changes in the epigenome are involved. In such cases, path dependencies at the individual level of phenotypic life courses may remain undiscovered or be wrongly assigned to other causes that are more easily visible.

That patterns of gene–environment interplay over the life course are not uniform was demonstrated in a comparison of cognitive ability and personality. Briley and Tucker-Drob (2017) found three marked differences between the two: First, the heritability of cognition increases substantially with child age, while the heritability of personality decreases modestly with age. Second, the increasing stability of cognition with age is overwhelmingly mediated by genetic factors, whereas the increasing stability of personality with age is entirely mediated by environmental factors. Third, the timing of stability during life differs: Stability of cognition nears its asymptote by the end of the first decade of life, whereas stability of personality takes three decades to near its asymptote. These differences can be traced back to different patterns of gene–environment interplay. For cognitive ability, genetic influences increase during childhood in both magnitude and stability. As a result, genetic effects increasingly contribute to phenotypic stability in child development. The main mechanism behind this development is gene–environment correlation. It is an upward spiral created by actively seeking stimulating environments beneficial for cognitive development. Moreover, especially in the educational attainment process, positive responses to perceived cognitive ability differences may be identified

and reinforced by teachers, parents, and peers, thus amplifying initially smaller differences to larger ones. This is what Dickens and Flynn (2001) called a genetic multiplier effect (see also Nisbett et al. 2012). However, such a mechanism does not apply, or at least applies much less, to the development of personality. Such reinforcing training as in cognitive ability does not take place here. Rather, with increasing age the diversity of environmental experiences initially grows, but then, due to path dependence, gets less heterogeneous with increasing age. Taken together, genes are the crucial stabilizing force for personality (Briley and Tucker-Drob 2015). Nevertheless, phenotypic stability increases over a lifetime, which cannot be explained by genetic contributions as in the case of cognitive ability. Rather, adaptation to unique environmental demands plays this role.

3.6 Cumulative Advantage and Disadvantage

The gene–environment interplay governing the development of cognitive ability is an example of one of the most prominent ideas of how social inequality develops over time: cumulative advantage and disadvantage (Dannefer 2003). “Disadvantage increases exposure to risk, but advantage increases exposure to opportunity” (Ferraro and Pylypiv Shippee 2009, p. 335). Upward and downward spirals of success and failure lead to an accentuation of inequality in the sense that early limited differences in success and failure become bigger over time.

That biological influences and genetic variation may contribute to generating cumulative advantage and disadvantage is not new (DiPrete and Eirich 2006; Ferraro et al. 2009). As already exemplified for cognitive ability, more endowed individuals choose more stimulating environments, which in turn provide better opportunities to boost cognitive ability, not least because significant others react with more encouragement and more nurturing than to less endowed individuals. This then provides higher levels of development allowing for a higher jump to the next level than for those who cannot profit from such experiences to the same degree. Thus, active and evocative gene–environment correlation go hand in hand and become relevant again and again in subsequent decisions about pathways to follow. What was demonstrated for cognitive ability could be relevant also for cumulative advantage and disadvantage in other outcomes, such as educational and occupational achievement and attainment or health.

Also, the concept of environmental sensitivity and related concepts such as differential susceptibility, vantage sensitivity (Jolicoeur-Martineau et al. 2017), and sensory processing sensitivity (Greven et al. 2019) might help in the understanding of cumulative advantage and disadvantage. These concepts of individual reaction to environmental conditions are based on genetic and epigenetic variants related to immune regulation and brain functioning related to stress regulation (Heim and Binder 2012). While multiple genes operate in multiple environments to induce risky stress, these same genes also seem to enhance the beneficial effects of a positive environment. These differential reactions to environmental forces may contribute to understanding resilience as well as vulnerability as central concepts of life course research on the development of unequal life chances (Spini and Widmer 2023). However, whereas resilience and vulnerability as stable dispositions may make downward

or upward spirals more likely, environmental sensitivity may make turning points more likely, since it accentuates the consequences of both good and bad experiences.

4 Methodological Approaches to Genetically Informative Research

In the following, I give only a short overview of methodological approaches to study gene–environment interplay, with a focus on mechanisms governing the life course. Two methodologies are used to study genetic origin in addition to social origin as point of departure of life courses and individual development (see Sect. 3.1). These two are based on an understanding of genes as (nearly) fixed and immutable over the life course. Genetic variation is traditionally studied by comparing twins or adoptees, sometimes including the family members with whom they live. In this article, solely the twin-based approach, including twin family studies, as the by far most frequent application compared to adoptee studies is presented. More recently, molecular genetic approaches have won ground, starting with candidate genes. Today, the most applied approach is the use of polygenic scores (PGSs), but there are several other whole genome methods as well. This article refers to PGSs only. A third approach, epigenetics, conceptualizes genetic contributions as “fluid.” The focus here is on gene regulation over time—a “genome with a life span” (Lappé and Landecker 2015)—instead of (nearly) fixed DNA (see Sect. 2.1).¹³ Irrespective of what methodology is applied, there are two important supportive conditions for the study of life courses. First, phenotypes should be available to allow for operationalizing the mechanisms of gene–environment interplay presented in Sects. 2 and 3. Second, longitudinal data should be available to follow study participants over time with a sufficient number of cases for sophisticated modeling.

4.1 Variance Decomposition Based on Twin Comparisons

Variance decomposition models utilize twins to study the extent to which variation in genes and environments contributes to the variation of a phenotype—be it personality, skills income, education, or health. Here, the variance of the phenotype is attributed to an additive genetic (A), a shared environment (C), and a nonshared environment (E) component, which together total 100% of the overall variance. These three components are all unobserved latent sources of variation, i.e., neither genes nor social characteristics are measured but are estimated from the comparison of dizygotic (DZ) and monozygotic (MZ) twins. Twin-based models typically assume that DZ twins share on average 50% of their genes, whereas MZ twins are genetically identical. Moreover, it is assumed that MZ and DZ twins share the same environments to the same degree. Consequently, any additional similarity between MZ compared to DZ twins should be attributable to genetic variation. The contribution of the environment is additionally subdivided into two components. The

¹³ It would go too far to discuss in-depth the underlying assumptions and variations for the three approaches. For ACE decomposition, see Knopik et al. (2017) and Diewald et al. (2015). For molecular genetic approaches, see Mills et al. (2020) and Young et al. (2019). For epigenetic approaches, see Li (2021).

difference between a shared (C) and nonshared environment (E) is that in the first case, environmental influences make twins effectively more similar due to the same experiences perceived in the same way, whereas in the second case, environments drive twins apart concerning the characteristic under study. This happens through different experiences and also different perceptions and evaluations of the same environments. The C component is often taken as proxy for all living conditions linked to social origin, whatever the concrete experiences are, and these can also be located outside the family household, e.g., the neighborhood (Freese and Jao 2017). This equation of C with social origin is more appropriate if the sample controls for other possible sources of uniform experiences, i.e., ethnic homogeneity in the population studied, and a cohort–sequential design. It is important to note that the black box approach provides population parameters, i.e. the estimates of the variance components that may differ considerably across different groups in a society and between societies across time and place (Selita and Kovas 2019; Branigan et al. 2013). It is possible neither to generalize results from one population to others nor to infer from population parameters the role of genes for particular individuals.

The fact that these variance components constitute explanatory black boxes seems at first glance a drawback compared to precise measurements. However, they have advantages as well. They allow for a rough overall estimate of the size of the contributions that genetic as well as social origin provide for predicting life courses.¹⁴ Especially for educational attainment, these genetic estimates are much bigger than estimates for parental resources, parenting, or cultural capital as the favorite concepts in sociology for explaining the long shadow of the family of origin (Mönkediek and Diewald 2022).

Though in ACE decomposition models genetic origin is often conceptualized with a fixed DNA in mind, these models also allow modeling of changes in the contribution of genes to phenotypic developments over the life course, as was done in the study of Briley and Tucker-Drob (2017) on the different developments of personality and cognitive ability, as well as in studies about the changing impact of genes and environment on educational achievement (e.g., Johnson et al. 2009). Studies using ACE modeling can include rGE as well as $G \times E$, and moreover interdependencies between different strands of development, e.g., when the heritability of education is explained by the heritable contribution of cognitive and so-called noncognitive abilities such as conscientiousness (Krapohl et al. 2014; Starr and Riemann 2022). Also, the intergenerational transmission of educational attainment can be modeled

¹⁴ This approach has been criticized for its underlying assumptions, the violation of which has led to bias in estimates (e.g., Burt and Simons 2014). There is the equal environment assumption that DZ and MZ twins share environmental influences to the same degree; that there is no assortative mating of the parents concerning the characteristics of interest; that there is neither gene–environment interaction nor gene–environment correlation and also only additive genetic effects; and, finally, that twins and their families are in all respects representative of the population as a whole. Most of these concerns, though not all, can be checked and resolved by more complex variants of the ACE decomposition, most notably by modeling gene–environment interaction and covariation (Turkheimer and Harden 2014) and by using a twin family design including parents and siblings (Wolfram and Morris 2002). In sum, most researchers in the field, as well as those with a primarily molecular genetic background, agree that a twin-based approach provides reasonable estimates of the role of genes for, in principle, all phenotypes that exist, if they are only included in such studies.

(for a comparison between classical twin design and a nuclear twin family design, see Wolfram and Morris 2022).

Moreover, ACE decomposition can be used not only in univariate analyses but also in bivariate analyses to detect whether a correlation between two phenotypic variables is confounded by shared genes or shared environments. For example, Stienstra et al. (2021) investigated the association between cognitive ability and educational attainment dependent of social origin. In a design not controlling for genetic confounders or shared environmental influences, parents of high socioeconomic status seem to compensate for the lower cognitive ability of their children. However, when possible confounding by genetic variation and the shared environment are included as latent variables, this compensation effect is no longer significant.

Finally, the biggest strength of twin-based modeling is the fact that to date there is no other possibility to provide a reasonable estimate of the whole genome effect. This strength is at the cost of not knowing which genes contribute which effects to unravel what the genetic makeup has to do with the development of phenotypic characteristics and behaviors. For example, a high A for attainment is often interpreted in a way that attainment is not limited by social barriers preventing the exploitation of one's respective genetic potential. However, a high A could also be based on being left alone with genetic risks for attainment, e.g., chronic inflammation or anxiety. Moreover, a major disadvantage of twin-based modeling is the limited availability of large samples of twins and their families representative of the whole society, combined with a rich selection of phenotypic measurements. Most twin samples are small and selective, with few exceptions. This shortage limits the generalizability of results and the possibility of making use of more complex, especially longitudinal ACE modeling.

4.2 Molecular Genetics and Epigenetics

The era of molecular genetic approaches started with the so-called candidate gene approach. Compared to the twin-based methodology, it was attractive to have a genetic variant that could be used as any other variable in multivariate analyses, which makes things much easier for social scientists given the methodological background they are mostly trained for. Moreover, instead of a black box, we now have specific alleles that are hypothesized to have considerable effects on biological processes relevant to an individual characteristic under consideration. This allows a theory-guided investigation of genetic effects. As appealing as candidate genes are, most studies failed to replicate and seemed to be false positives (Dick et al. 2015). Moreover, the growing number of genome-wide association studies showed how small the proportion of variance is that is explained by single genes. As a consequence, interest switched from hypothesis-guided testing of single alleles to exploiting the whole genome for an exploratory, hypothesis-free searching of the genetic sources of a phenotype of interest that does not follow simple Mendelian monogenic inheritance but is polygenic, i.e., many genes contribute to its development. In the following, I concentrate on polygenic scores (PGSs) only because they are by far presently the most frequent molecular genetic approach applied in empirical social research. They are calculated as the weighted sum of genetic variants, where the

weights are proportional to the strength of the association between a genetic variant and the outcome under consideration. As a rule, the effects of single variants are tiny. The underlying mechanisms by which these effects contribute to the phenotype of interest are only partly known. Though molecular genetic research is increasingly identifying the roles of single genes for phenotypes, the inclusion of single genes into a score is purely correlational, which means that even if some of the genes included are known for their specific functioning, the PGS is not.

Polygenic scores still suffer considerably from “missing heritability.” This term denotes the gap between heritability from twin-based variance decomposition and heritability estimates from genotyped data. Up to now and in the near future, only a smaller part of the whole genome contribution can be identified by the latter method. Therefore, working with PGSs instead of ACE decomposition is only an imperfect move away from a black box approach, one that in addition is available only for a limited, though rapidly growing, number of PGSs.¹⁵ By far, the most predictive PGS is that for educational attainment. In its latest version (Okbay et al. 2022), 3952 significant single-nucleotide polymorphisms (SNPs) were identified, which together explain 12–16% of the variance of educational attainment—compared to about 40% for the A component (Silventoinen et al. 2020). For most PGSs, the explained variance is much lower, especially for more specific characteristics or subdimensions of broader concepts (for an overview, see Becker et al. 2021). It is unclear to what degree the missing heritability systematically distorts the results because it is not random which SNPs are captured and which ones are not. A second problem is environmental confounding. Burt (2023) points to several reasons why genetic influences are hard to distinguish from environmental influences, especially in surveys based on unrelated individuals, and how this may come about.¹⁶ As Burt (2023) infers convincingly, this may lead to obscuring structural disadvantages and cultural influences as environmental factors with high relevance in sociological thinking.

Nevertheless, working with a PGS by simply using it as a variable in multivariate analyses has many practical advantages.¹⁷ Wickrama et al. (2021) provide an example of how the PGS for education, interacting with early socioeconomic adversity, influences educational and economic attainment by life course processes. Influences of genes and environment contribute to persisting disadvantage not only additively but also by creating chains of failure through circles of cumulative disadvantage, with contributions of partly different experiences across life stages.

¹⁵ <https://www.pgscatalog.org/>

¹⁶ Polygenic scores are often interpreted as indexing individual genetic risk for a trait, but they can also capture the environmental risk for the family (Kong et al. 2018). Therefore, as a rule, within-family genetic effects are smaller than between-family effects. Genome-wide association studies of unrelated individuals represent a combination of inherited genetic variation (direct effects, which are what they intend to measure), as well as indirect genetic effects based on population stratification (systematic ancestry differences), assortative mating, and genetic nurture from relatives (meaning that influences from family members around us, and to a much lesser degree also from nonkin significant others, are genetically confounded).

¹⁷ Moreover, it is information that can be collected later in life and therefore be of use in long-running panel studies that are representative of the whole population, even long after their start.

Methodologically, PGSs provide additional possibilities to cope with gene–environment interaction and covariation. It is possible to calculate active gene–environment correlation, which cannot be calculated in a twin design. However, because genetic variation and environmental variation are confounded for unrelated individuals as a rule, molecular genetic studies usually overestimate the role of genes if measured for unrelated individuals; within-family estimates are only about half of the estimates of unrelated individuals (Young 2019). The reason is that a within-family design removes the total influence of indirect genetic effects from family members, assortative mating, and population stratification, all of them nurturing rGE.

Like twin-based designs, PGSs capture average genetic effects within a particular environment, and their effects cannot be simply transferred to populations other than those from which the genome-wide association study discovery sample was drawn. As in twin-based methodology, molecular genetic variation does not tell transcendent, ever-valid truths about nature but provides population-specific parameters. But other than twin-based methodology, molecular genetics allows for creating supra-individual, aggregate measures of populations—for the whole society, for selected regions and neighborhoods, and for groups such as immigrants (Abdellaoui et al. 2019).

Both ACE decomposition and molecular genetics do not allow the study of biological developments parallel to social and mental development. The idea of possibly intersecting processes comprising not only the social and mental development but also the organism is realized in looking at changes in the epigenome (see Sects. 2.1 and 3.1).

Working with *epigenetic* data is similar to working with molecular genetic data. Methylation may have a causal role consistent with an infinitesimal model in which many methylation sites each have a small influence, amounting to a large overall contribution that can be captured by aggregate scores, such as several epigenetic clocks predicting the pace of biological aging as the surely most applied example (Horvath and Raj 2018). It allows prediction of life expectancy better than chronological age. There are other applications as well, such as an inflammation-related score (Stevenson et al. 2020) and combinations of genetic and epigenetic information (Shah et al. 2015). For studying the life course, combining genetic and epigenetic information is especially useful for theoretical constructs that include both ideas about a fixed component rooted in DNA variation and epigenetic variation dependent on the accumulation of life experiences. This is, for example, the case with environmental sensitivity, which can be expressed more into the direction of vulnerability or more into the direction of resilience due to diverging life experiences within, and probably also across, generations (see Sect. 2.2).

4.3 Combining Twin (Family) Designs with Molecular Genetic and Epigenetic Data

There is not one approach that covers everything best. Rather, a comparison of results across different approaches and especially a combination of genome- and epigenome-based methods with (twin) family designs are best practice, since these allow for combining the strengths and compensating for the weaknesses of the

various approaches (Young et al. 2019; Friedman et al. 2021). Even better than comparing results across approaches is to combine them; PGS-based analyses profit from within-family designs in the first instance by the best control of rGE and population stratification in ancestry, which makes analyses more reliable for assessing pure genetic contributions to an outcome of interest. Analyzing unrelated individuals with molecular genetics overstates the genetic contribution due to correlation with the environment. Family-based quasi-experimental designs such as sibling comparison, adoption, and extended family studies are effective means to disentangle direct genetic effects from indirect ones (McAdams et al. 2023; Selzam et al. 2019).

In a family design, not necessarily a twin family design, it is possible to separate direct genetic transmission from parents to children from nontransmitted alleles as indirect genetic transmission, also called genetic nurture. Genetic nurture is an indirect influence of parental genotypes on children's characteristics and behaviors through the family environment influenced by parental genes (Baier and Lyngstad 2024). Because direct genetic transmission is calculated separately from indirect transmission, the latter is a form of passive gene–environment covariation restricted to the nontransmitted genes (Kong et al. 2018). In other words, it is a parsimonious way to disentangle the genetic influence of parents from the environmental influence of parenting. This possibility made a remarkable career in sociogenomics in a very short time. For example, the nonnegligible influence of genetic nurture on education was not always found, but in the majority of studies on genetic nurture, it was (Wang et al. 2021).

For life course research, there are three obvious added values of family-based molecular genetic studies. First, this possibility can add new insights to the classical question of “linked lives” (Elder 1994), i.e., how significant others, not least family members, influence individual life courses. Second, a sibling comparison could contribute to a better understanding of privilege and affront experienced in the family. Still, within-family inequality is much less researched, and therefore less understood than between-family inequality, with the first contributing about a third to the overall inequality structure in contemporary societies (Conley 2005; Grätz 2018). In studies about compensation and accentuation of inequality among siblings, a crucial problem is to exhaustively operationalize all possibilities of how parents treat their offspring, especially in the case of largely age-divergent siblings. A possibility to circumvent this problem, as well as to control for differences in ability and effort between siblings, is to study differences in the degree to which a given genetic potential is realized. A PGS of education or cognitive ability can be taken as a genetic predictor of this potential and can easily be integrated as a variable in such studies (Fletcher et al. 2023). Thus, we do not know without additional phenotypic information why siblings profit differently from the home environment in realizing their genetic potential, but we know to what degree this is the case without problems of improper measurements of age-dependent environmental influences. Third, the separation into direct and indirect effects could also be helpful for another research problem mentioned previously: that genetic inheritance can be on the one hand a mechanism contributing to status stability in the social mobility regime, and on the other hand a mechanism contributing to upward and downward mobility, depending on different mixes of direct and indirect genetic transmission.

In medical research, a case co-twin design with monozygotic twins discordant in their experiences of interest has been established as the gold standard. Such a design takes advantage of the mostly shared early life environment, starting in utero, as well as almost completely identical genetic makeup. Moreover, there are no age or sex differences as third variable confounders. Thus, MZ twins strengthen causal claims compared to less controlled designs and are an alternative to randomized controlled trials. Especially when social, psychological, and biological developments are part of the research as parallel and intersecting processes, such a controlled design is required (e.g., Tan et al. 2015 for epigenetics). Against the identical twin folklore, the often quite low shared environment component in the twin-based ACE decomposition of developmental outcomes and human traits makes evident that there is a less uniform impact of environments on individuals than is commonly assumed, which is not least because a shared environment may impact differently on individuals, even MZ twins. This has, for example, been shown for perceived chaos in the parental household (Starr et al. 2023). Moreover, parents often decide to put MZ twins into different extrahousehold contexts, at school or in extracurricular activities, to encourage them to develop individuality (Segal and Russell 1992).

4.4 Problems of Causality

Many researchers in the field, including myself, started to study gene–environment interplay motivated by the awareness that not considering genetics makes purely phenotypic calculations partly spurious due to unobserved genetic and environmental influences. Many studies confirmed this suspicion. However, identifying the role of genes and environments properly is problematic as well. Whereas behavioral geneticists working with ACE decomposition mostly avoided the term of causality for their analyses of variance decomposition, molecular geneticists fight against criticism with rapid statistical development of ways to separate genetic from environmental contributions. Taken together, there is much effort devoted to making this separation statistically convincing. This is, not only in this research field but more generally, an important criterion to distinguish between more or less causally robust analyses. However, in a mechanistic view of causality, following the cogs and wheels operating on the way from (epi)genetic variants to variation in phenotypic developments, it would be necessary to disentangle what exactly they contribute to an understanding of the genetic sources of phenotypic developments. Despite all methodological efforts, this is not a realistic aim for the moment. Many insights are more exploratory than deduced from the theoretical ground. Genetics and epigenetics cover important components of the interplay of biological and social forces. Nevertheless, even together they still simplify the biological processes linked to gene–environment interplay. Most discussed are endocrinological processes of hormones and the microbiome to understand these biological mechanisms more deeply (Landecker 2024). In the face of these gaps in the chain of interlinked biological and social processes between the genome and phenotypic variation, any causal claim in a strict sense cannot be fulfilled. However, seen from the other side, there are methods that are more or less causally robust, with increasing tendency to more robust modeling. Even imperfect, biased modeling is better than negating genetic

contributions, as it implies comparably smaller biases and less omission of causally relevant information.

5 Conclusions

Genetics could enrich life course research by studying the ways that “social and biological forces interact in complex and dynamic ways to define ranges of likely behaviors.” This was already stated by Shanahan et al. (2003, p. 599) about 20 years ago. Life course research seems an ideal example of how different disciplines can contribute to a better understanding of some of its central premises and research questions. Nevertheless, life course research has gone its way largely without including genetically informative approaches. Vice versa, genetically informative research, though longitudinal modeling exists, has not yet taken much notice of life course theories. In this paper I focused on how genetically informative designs can contribute to answering research questions more properly than can designs without genetic information. When theorizing and operationalizing social mechanisms in gene–environment interplay, genetically informative research seems only loosely linked to current theoretical developments and research questions. The social sciences, particularly sociology, do not call very loudly for including genetic information (Hopcroft and Schnettler 2024). Consequently, researchers in the field of genetically informative research often orient themselves more to a common denominator with other researchers in the field, coming from a variety of scientific backgrounds, than to the refinements of their disciplinary approaches.

This paper has tried to demonstrate for the life course approach that it is fruitful to go this way as well: Take concepts and research questions of life course research as a starting point and look at how genetically informative research can visibly add to explanations over and above correcting for unobserved genetic confounding. Though maybe not fully exhaustive, it could be shown that for central concepts and questions, the inclusion of genes and the epigenome can indeed enhance our understanding. More generally, relating to the state-of-the-art research questions in a discipline can help to integrate genetically informed research into the disciplinary mainstream as well as profit from up-to-date conceptualizations of environmental influences.

The inclusion of PGSs into many big studies with a large number of cases should open the field to many more interested researchers. The same is true for the methodology of using PGSs almost like any other variables in multivariate modeling. Especially when applied in a household or family design, which is often the case in long-running surveys, this provides a reliable control for confounding. Reasonable estimates are possible without very large investments in a new methodology, though it may not always be the very best solution available.

This does not at all mean that genetically informative research only serves the theories and concepts of research usually neglecting genetic contributions. Rather, including genes can raise new questions that were not in the foreground before, such as the distinction of shared and nonshared environmental influences. If we intend to raise the quality of, e.g., school environments, this is a gain, but in behavioral genetic

research it has been shown that it also has the potential to increase the inequality of life chances, since the more talented should profit more from resourceful and stimulating environments than the less talented. This is not unavoidable but calls for accompanying countermeasures to avoid such unintended consequences. Including genetic variation and gene regulation, rather than focusing solely on social origin, allows us to trace from the very beginning of life how a society shapes the individuals within it. This shaping occurs both as a whole and within subsystems such as education, occupation, and the welfare state, and it includes changes in the genetic pool through immigration, outmigration, and spatial mobility within society's borders.

Acknowledgements I would like to thank the anonymous reviewers as well as Sebastian Schnettler and Johannes Huinink as the two managing editors for many helpful comments that helped to improve the manuscript.

Funding Open Access funding enabled and organized by Projekt DEAL.

Conflict of interest M. Diewald declares that he has no competing interests.

Open Access This article is licensed under a Creative Commons Attribution 4.0 International License, which permits use, sharing, adaptation, distribution and reproduction in any medium or format, as long as you give appropriate credit to the original author(s) and the source, provide a link to the Creative Commons licence, and indicate if changes were made. The images or other third party material in this article are included in the article's Creative Commons licence, unless indicated otherwise in a credit line to the material. If material is not included in the article's Creative Commons licence and your intended use is not permitted by statutory regulation or exceeds the permitted use, you will need to obtain permission directly from the copyright holder. To view a copy of this licence, visit <http://creativecommons.org/licenses/by/4.0/>.

References

- Abdellaoui, Abdel, David Hugh-Jones, Loic Yengo, Kathryn E. Kemper, Michel G. Nivard, Laura Veul, Yan Holtz, Brendan P. Zietsch, Timothy M. Frayling, Naomi R. Wray, Jian Yang, Karin J. H. Verweij and Peter M. Visscher. 2019. Genetic correlates of social stratification in Great Britain. *Nature Human Behaviour* 3(12):1332–1342.
- Aisenbrey, Silke, and Anette Fasang. 2017. The interplay of work and family trajectories over the life course: Germany and the United States in comparison. *American Journal of Sociology* 122(5):1448–1484.
- Baier, Tina, and Torkild Hovde Lyngstad. 2024. Social Background Effects on Educational Outcomes – New Insights from Modern Genetic Science. This issue.
- Becker, Joel, Casper A. P. Burik, Grant Goldman, Nancy Wang, Hariharan Jayashankar, Michael Bennett, Daniel W. Belsky, Richard K. Linnér, Rafael Ahlskog, Aaron Kleinman, David A. Hinds, 23andme Research Group, Avshalom Caspi, David L. Corcoran, Terrie E. Moffitt, Richie Poulton, Karen Sugden, Benjamin S. Williams, Kathleen Mullan Harris, Andrew Steptoe, Olesya Ajnakina, Lili Milani, Tõnu Esko, William G. Iacono, Matt McGue, Patrik K. E. Magnusson, Travis T. Mallard, K. Paige Harden, Elliot M. Tucker-Drob, Pamela Herd, Jeremy Freese, Alexander Young, Jonathan P. Beauchamp, Philipp Koellinger, Sven Oskarsson, Magnus Johannesson, Peter M. Visscher, Michelle N. Meyer, David Laibson, David Cesarini, Daniel J. Benjamin, Patrick Turley and Aysu Okbay. 2021. Resource profile and user guide of the Polygenic Index Repository. *Nature Human Behavior* 5:1744–1758.
- Belsky, Daniel W., Terrie E. Moffitt, David L. Corcoran, Benjamin Domingue, HonaLee Harrington, Sean Hogan, Renate Houts, Sandhya Ramrakha, Karen Sugden, Benjamin S. Williams, Richie Poulton and Avshalom Caspi. 2016. The genetics of success: how single-nucleotide polymorphisms associated with educational attainment relate to life-course development. *Psychological Science* 27(7):957–972.

- Bemmels, Heather R., S. Alexandra Burt, Lisa N. Legrand, William G. Iacono and Matt McGue. 2008. The heritability of life events: an adolescent twin and adoption study. *Twin Research and Human Genetics* 11(3):257–265.
- Bernardi, Laura, Johannes Huinink and Richard A. Settersten Jr. 2019. The life course cube: A tool for studying lives. *Advances in Life Course Research* 41:100258.
- Bol, Thijs, and Kim A. Weeden. 2015. Occupational closure and wage inequality in Germany and the United Kingdom. *European Sociological Review* 31(3):354–369.
- Branigan, Amelia R., Kenneth J. McCallum and Jeremy Freese. 2013. Variation in the heritability of educational attainment: an international meta-analysis. *Social Forces* 92:109–140.
- Braudt, David B. 2018. Sociogenomics in the 21st century: an introduction to the history and potential of genetically informed social science. *Sociology Compass* 12(10):e12626
- Briley, Daniel A., and Elliot M. Tucker-Drob. 2017. Comparing the developmental genetics of cognition and personality over the life span. *Journal of Personality* 85(1):51–64.
- Broerman, Rebecca. 2020. Diathesis-stress model. In *Encyclopedia of personality and individual differences*, eds. Virgil Zeigler-Hill and Todd K. Shackelford, 1107–1109. Cham: Springer.
- Bronfenbrenner, Urie, and Stephen J. Ceci. 1994. Nature-nurture reconceptualized in developmental perspective: a bioecological model. *Psychological Review* 101(4):568–586.
- Burt, Callie H. 2023. Challenging the utility of polygenic scores for social science: environmental confounding, downward causation, and unknown biology. *Behavioral and Brain Sciences* 46:e207.
- Burt, Callie H., and Ronald L. Simons. 2014. Pulling back the curtain on heritability studies: biosocial criminology in the postgenomic era. *Criminology* 52(2):223–262.
- Cao-Lei, Lei, Renaud Massart, Matthew Suderman, Ziv Machnes, Guillaume Elgbeili, David Laplante and Moshe Szyf. 2014. DNA methylation signatures triggered by prenatal maternal stress exposure to a natural disaster: project Ice Storm. *PLoS One* 9(9):e107653.
- Cao-Lei, L., S. R. De Rooij, S. King, S. G. Matthews, G. A. S. Metz, T. J. Roseboom and Moshe Szyf. 2020. Prenatal stress and epigenetics. *Neuroscience & Biobehavioral Reviews* 117:198–210.
- Coall, David A., Anna Callan, Thomas E. Dickens and James S. Chisholm. 2015. Evolution and prenatal development. *Social, Emotional, and Personality Development* 3:57–105.
- Conley, Dalton. 2005. *The pecking order: a bold new look at how family and society determine who we become*. New York: Vintage.
- Conley, Dalton. 2009. The promise and challenges of incorporating genetic data into longitudinal social science surveys and research. *Biodemography and Social Biology* 55(2):238–251.
- Conley, Dalton, Benjamin W. Domingue, David Cesarini, Christopher Dawes, Cornelius A. Rietveld and Jason D. Boardman. 2015. Is the effect of parental education on offspring biased or moderated by genotype? *Sociological Science* 2:82–105.
- Danese, Andreas, and Stephanie J. Lewis. 2017. Psychoneuroimmunology of early-life stress: the hidden wounds of childhood trauma? *Neuropsychopharmacology* 42(1):99–114.
- Dannefer, Dale. 2003. Cumulative advantage/disadvantage and the life course: cross-fertilizing age and the social science theory. *Journal of Gerontology: Social Sciences* 58:S327–S337.
- Daskalakis, Nikolaos P., Changxin Xu, Heather N. Bader, Chris Chatzinakos, Peter Weber, Iouri Makotkine, Amy Lehrner, Linda M. Bierer, Elisabeth B. Binder and Rachel Yehuda. 2021. Intergenerational trauma is associated with expression alterations in glucocorticoid- and immune-related genes. *Neuropsychopharmacology* 46:763–773.
- Demange, Perline A., Margherita Malanchini, Travis T. Mallard, Pietro Biroli, Simon R. Cox, Andrew D. Grotzinger, Elliot M. Tucker-Drob, Abdel Abdellaoui, Louise Arseneault, Elsje van Bergen, Dorret I. Boomsma, Avshalom Caspi, David L. Corcoran, Benjamin W. Domingue, Kathleen Mullan Harris, Hill F. Ip, Colter Mitchell, Terrie E. Moffitt, Richie Poulton, Joseph A. Prinz, Karen Sugden, Jasmin Wertz, Benjamin S. Williams, Eveline L. de Zeeuw, Daniel W. Belsky, K. Paige Harden and Michel G. Nivard. 2021. Investigating the genetic architecture of noncognitive skills using GWAS-by-subtraction. *Nature Genetics* 53(1):35–44.
- Dick, Danielle M., Arpana Agrawal, Matthew C. Keller, Amy Adkins, Fazil Aliev, Scott Monroe, John K. Hewitt, Kenneth S. Kendler and Kenneth J. Sher. 2015. Candidate gene–environment interaction research: reflections and recommendations. *Perspectives on Psychological Science* 10(1):37–59.
- Dickens, William T., and James R. Flynn. 2001. Heritability estimates versus large environmental effects: the IQ paradox resolved. *Psychological Review* 108(2):346–369.
- Diewald, Martin, Yixuan Liu and Dmitry Kuznetsov. In press. Ungleiche Lebenschancen durch das Epigenom? Zum Zusammenwirken biologischer mit sozialen Mechanismen. In *Handbuch Sozialstrukturanalyse*, eds. Dirk Konietzka and P. Boehnke. Heidelberg: Springer.

- Diewald, Martin. 2023. Health as a consequence of genetic variation, gene transcription and life course experiences. In *Handbook of health inequalities across the life course*, Hrsg. R. Hoffmann, 59–73. London: Edward Elgar.
- Diewald, Martin, and Thomas Faist. 2011. Von Heterogenitäten zu Ungleichheiten: Soziale Mechanismen als Erklärungsansatz der Genese sozialer Ungleichheiten. *Berliner Journal für Soziologie* 21(1):91–114.
- Diewald, Martin, and Karl Ulrich Mayer. 2009. The sociology of the life course and life span psychology: integrated paradigm or complementing pathways? *Advances in Life Course Research* 14(1–2):5–14.
- Diewald, Martin, Tina Baier, Wiebke Schulz and Reinhard Schunck. 2015. Status attainment and social mobility. *Kölner Zeitschrift für Soziologie und Sozialpsychologie* 67:371–395.
- DiPrete, Thomas A., and Gregory M. Eirich. 2006. Cumulative advantage as a mechanism for inequality: a review of theoretical and empirical developments. *Annual Review of Sociology* 32:271–297.
- Duncan, Greg J., Kathleen M. Ziol-Guest and Ariel Kalil. 2010. Early-childhood poverty and adult attainment, behavior, and health. *Child Development* 81(1):306–325.
- Elder, Glen H. 1994. Time, human agency, and social change: perspectives on the life course. *Social Psychology Quarterly* 57(1):4–15.
- Elder, Glen H., and Angela M. O’Rand. 1995. Adult lives in a changing society. In *Sociological perspectives on social psychology*, eds. Karen S. Cook, Gary Alan Fine and James S. House, 452–475. Boston: Allyn and Bacon.
- Elder, Glen H., Monica Kirkpatrick Johnson and Robert Crosnoe. 2003. The emergence and development of life course theory. In *Handbook of the life course*, eds. Jeylan T. Mortimer and Michael J. Shanahan, 3–19. Boston, MA: Springer.
- Engzell, Per, and Felix C. Tropf. 2019. Heritability of education rises with intergenerational mobility. *Proceedings of the National Academy of Sciences* 116(51):25386–25388.
- Ferraro, Kenneth F., and Tetyana Pylypiv Shippee. 2009. Aging and cumulative inequality: how does inequality get under the skin? *The Gerontologist* 49(3):333–343.
- Fletcher, Jason. 2023. Often wrong, sometimes useful: including polygenic scores in social science research. *Behavioral & Brain Sciences* 46:e213.
- Fletcher, Jason M., Yuchang Wu, Zijie Zhao and Qiongchi Lu. 2023. The production of within-family inequality: insights and implications of integrating genetic data. *PNAS Nexus* 2(4):pgad121.
- Freese, Jeremy, and Yu-Han Jao. 2017. Shared environment estimates for educational attainment: a puzzle and possible solutions. *Journal of Personality* 85(1):79–89.
- Friedman, Naomi P., Marie T. Banich and Matthew C. Keller. 2021. Twin studies to GWAS: there and back again. *Trends in Cognitive Sciences* 25(10):855–869.
- Gaunt, Tom R., Hashem A. Shihab, Gibran Hemani, Josine L. Min, Geoff Woodward, Oliver Lyttleton, Jie Zheng, Aparna Duggirala, Wendy L. McArdle, Karen Ho, Susan M. Ring, David M. Evand, George Davey Smith and Caroline L. Relton. 2016. Systematic identification of genetic influences on methylation across the human life course. *Genome Biology* 17(1):1–14.
- Ghai, Meenu, and Farzeen Kader. 2022. A review on epigenetic inheritance of experiences in humans. *Biochemical Genetics* 60(4):1107–1140.
- Giele, Janet Z., and Glenn H. Elder (eds.). 1998. *Methods of life course research: qualitative and quantitative approaches*. London/Thousand Oaks/New Delhi: Sage.
- Goosby, Bridget, and Jacob Cheadle. 2024. The immune system is a complex system: inflammatory morbidity and structural racism. This issue.
- Grätz, Michael. 2018. Competition in the family: inequality between siblings and the intergenerational transmission of educational advantage. *Sociological Science* 5:246–269.
- Greven, Corina U., Francesca Lionetti, Charlotte Booth, Elaine N. Aron, Elaine Fox, Haline E. Schendan, Michael Pluess, Hilgo Bruining, Bianca Acevedo, Patricia Bijttebier and Judith Homberg. 2019. Sensory processing sensitivity in the context of environmental sensitivity: a critical review and development of research agenda. *Neuroscience & Biobehavioral Reviews* 98:287–305.
- Haberstick, Brett C., Stephanie Schmitz, Susan E. Young and John K. Hewitt. 2005. Contributions of genes and environments to stability and change in externalizing and internalizing problems during elementary and middle school. *Behavior Genetics* 35:381–396.
- Harden, K. Paige. 2021. *The genetic lottery: why DNA matters for social equality*. Princeton: University Press.
- Hedström, Peter, and Petri Ylikoski. 2010. Causal mechanisms in the social sciences. *Annual Review of Sociology* 36(1):49–67.

- Heim, Christine, and Elisabeth B. Binder. 2012. Current research trends in early life stress and depression: review of human studies on sensitive periods, gene–environment interactions, and epigenetics. *Experimental Neurology* 233(1):102–111.
- Hitlin, Steven, and Hye Won Kwon. 2016. Agency across the life course. *Handbook of the life course*: Volume II, eds. Michael J. Shanahan, Jeylan T. Mortimer and Monica Kirkpatrick Johnson, 431–449. Cham/Heidelberg/New York/Dordrecht/London: Springer.
- Hoffmann, Rasmus (ed.). 2023. *Handbook of health inequalities across the life course*. Cheltenham: Edward Elgar.
- Hopcroft, Rosemary, and Sebastian Schnettler. 2024. Herausforderungen und Errungenschaften evolutionärer und biosozialer Ansätze in der Soziologie. *Evolutionäre Sozialwissenschaften*, eds. Manfred Hammerl, Sascha Schwarz and Kai P. Willführ, 59–82. Wiesbaden: Springer VS.
- Horvath, Steve, and Kenneth Raj. 2018. DNA methylation-based biomarkers and the epigenetic clock theory of ageing. *Nature Reviews Genetics* 19(6):371–384.
- Househam, Ayman Mukerji. 2023. Effects of stress and mindfulness on epigenetics. In *Vitamins and hormones*, ed. Adrenal Gland, Vol. 122, 283–306. Cambridge/San Diego/Oxford/London: Academic Press.
- Johnson, Wendy, Ian J. Deary and William G. Iacono. 2009. Genetic and environmental transactions underlying educational attainment. *Intelligence* 37(5):466–478.
- Jolicoeur-Martineau, Alexia, Jay Belsky, Eszter Szekely, Keith F. Widaman, Michael Pluess, Celia Greenwood and Ashley Wazana. 2020. Distinguishing differential susceptibility, diathesis-stress, and vantage sensitivity: Beyond the single gene and environment model. *Development and Psychopathology* 32(1):73–83.
- Kendler, Kenneth S., and Laura Karkowski-Shuman. 1997. Stressful life events and genetic liability to major depression: genetic control of exposure to the environment? *Psychological Medicine* 27(3):539–547.
- Knopik, Valerie S., Matthew A. Maccani, Sarah Fracanzio and John E. McGeary. 2012. The epigenetics of maternal cigarette smoking during pregnancy and effects on child development. *Development and Psychopathology* 24(4):1377–1390.
- Knopik, Valerie S., Jenae M. Neiderhiser, John C. DeFries and Robert Plomin. 2017. *Behavioral genetics* (Seventh edition). New York: Worth Publishers, Macmillan Learning.
- Koenen, Karestan, Avshalom Caspi, Terrie E. Moffitt, Fruhling Rijdsdijk and Alan Taylor. 2006. Genetic influences on the overlap between low IQ and antisocial behavior in young children. *Journal of Abnormal Psychology* 115(4):787–797.
- Kong, Augustine, Gudmar Thorleifsson, Michael L. Frigge, Bjarni J. Vilhjalmsón, Alexander I. Young, Thorgeir E. Thorgeirsson, Stefania Benonisdóttir, Asmundur Oddsson, Bjarni V. Halldorsson, Gisli Masson, Daniel F. Gudbjartsson, Agnar Helgason, Gyda Bjornsdóttir, Unnur Thorsteinsdóttir and Kari Stefansson. 2018. The nature of nurture: effects of parental genotypes. *Science* 359(6374):424–428.
- Krapohl, Eva, Kaili Rimfeld, Nicholas Shakeshaft, Maciej Trzaskowski, Andrew McMillan, Jean-Baptiste Pingault, Kathryn Asbury, Nicole Harlaar, Yulia Kovas, Philip S. Dale and Robert Plomin. 2014. The high heritability of educational achievement reflects many genetically influenced traits, not just intelligence. *Proceedings of the National Academy of Sciences* 111(42):15273–15278.
- Kuh, D., Y. Ben-Shlomo, J. Lynch, J. Hallqvist and C. Power. 2003. Continuing professional education: life course epidemiology. *Journal of Epidemiology and Community Health* 57:778–783.
- Landecker, Hannah. 2024. How the Social Gets Under the Skin: From the Social as Signal to Society as a Metabolic Milieu. This issue.
- Lappé, Martine, and Hannah Landecker. 2015. How the genome got a life span. *New Genetics and Society* 34(2):152–176.
- Li, Yuan. 2021. Modern epigenetics methods in biological research. *Methods* 187:104–113.
- Li, Shuai., Zhoufeng Ye, Karen A. Mather, Tuong L. Nguyen, Gillian S. Dite, Nicola J. Armstrong, Ee Ming Wong, Anbupalam Thalamuthu, Graham G. Giles, Jeffrey M. Craig, Richard Saffery, Melissa C. Southey, Qihua Tan, Perminda S. Sachdev and John L. Hopper. 2022. Early life affects late-life health through determining DNA methylation across the lifespan: a twin study. *EBioMedicine* 77:103927.
- Mancini, Alessandra, John Christopher Magnotto and Ana Paula Abreu. 2022. Genetics of pubertal timing. *Best Practice & Research Clinical Endocrinology & Metabolism* 36(1):101618.
- Marks, Gary N., and Michael O'Connell. 2023. The importance of parental ability for cognitive ability and student achievement: implications for social stratification theory and practice. *Research in Social Stratification and Mobility* 83:100762.

- Mastrotheodoros, Stefanos, Marco P. Boks, Céline Rousseau, Wim Meeus and Susan Branje. 2023. Negative parenting, epigenetic age, and psychological problems: prospective associations from adolescence to young adulthood. *Journal of Child Psychology and Psychiatry* 64(10):1446–1461.
- Mayer, Karl Ulrich. 2009. New directions in life course research. *Annual Review of Sociology* 35:413–433.
- Mayer, Karl Ulrich. 2015. The German life history study—an introduction. *European Sociological Review* 31(2):137–143.
- McAdams, Tom A., Rosa Cheesman and Yasmin I. Ahmadzadeh. 2023. Annual research review: towards a deeper understanding of nature and nurture: combining family-based quasi-experimental methods with genomic data. *Journal of Child Psychology and Psychiatry* 64(4):693–707.
- Melzer, David, Luke C. Pilling and Luigi Ferrucci. 2020. The genetics of human ageing. *Nature Reviews Genetics* 21(2):88–101.
- Mills, Melinda C. 2022. Sociogenomics: theoretical and empirical challenges of integrating molecular genetics into sociological thinking. In *Handbook of sociological science*, eds. Klarita Gerxhani, Nan Dirk de Graaf and Werner Raub, 250–270. Cheltenham: Edward Elgar.
- Mills, Melinda, Nicola Barban and Felix C. Tropf. 2020. *An introduction to statistical genetic data analysis*. Cambridge, MA/London: The MIT Press.
- Mönkediek, Bastian, and Martin Diewald. 2022. Do academic ability and social background influence each other in shaping educational attainment? The case of the transition to secondary education in Germany. *Social Science Research* 101:102625.
- Mönkediek, Bastian, Pia Schober, Martin Diewald, Harald Eichhorn and C. Katharina Spiess. 2023. Does the Quality of Early Childhood Education and Care Centers Mitigate the Risk of Externalizing Problems? A Genetic-Sensitive Study of Preschoolers in Germany. This issue.
- Mullins, Jordan L., Dana E. Díaz, Rengin B. Firat and Kalina J. Michalska. 2024. Ethnic-Racial Discrimination Exposure and Anxiety in Latina Girls: Amygdala Volume as an Indirect Neurobiological Pathway. This issue.
- Needham, Belinda L., Jennifer A. Smith, Wei Zhao, Xu Wang, Bhramar Mukherjee, Sharon L. R. Kardia, Carol A. Shively, Teresa E. Seeman, Yongmei Liu and Ava V. Diez Roux. 2015. Life course socioeconomic status and DNA methylation in genes related to stress reactivity and inflammation: the multi-ethnic study of atherosclerosis. *Epigenetics* 10(10):958–969.
- Nielsen, François. 2006. Achievement and ascription in educational attainment: genetic and environmental influences on adolescent schooling. *Social Forces* 85(1):193–216.
- Nisbett, Richard E., Joshua Aronson, Clancy Blair, William Dickens, James Flynn, Diane F. Halpern and Eric Turkheimer. 2012. Intelligence: new findings and theoretical developments: correction to Nisbett et al. 2012. *American Psychologist* 67(2):129.
- Okbay, Aysu, Yeda Wu, Nancy Wang, Michael Bennett, Seyed Moeen Nehzati, Julia Sidorenko, Hyeok-moon Kweon, Grant Goldman, Tamara Gjorgjieva, Yunxuan Jiang, Barry Hicks, Chao Tian, David A. Hinds, Rafael Ahlsgog, Patrik K. E. Magnusson, Sven Oskarsson, Caroline Hayward, Archie Campbell, David J. Porteous, Jeremy Freese, Pamela Herd, 23andMe Research Team, Social Science Genetic Association Consortium, Chelsea Watson, Jonathan Jala, Dalton Conley, Philipp D. Koellinger, Magnus Johannesson, David Laibson, Michelle N. Meyer, James J. Lee, Augustine Kong, Loic Yengo, David Cesarini, Patrick Turley, Peter M. Visscher, Jonathan P. Beauchamp, Daniel J. Benjamin and Alexander I. Young. 2022. Polygenic prediction of educational attainment within and between families from genome-wide association analyses in 3 million individuals. *Nature Genetics* 54(4):437–449.
- Plomin, Robert. 2019. *Blueprint, with a new afterword: how DNA makes us who we are*. Cambridge, MA/London: The MIT Press.
- Plomin, Robert, and Yulia Kovas. 2005. Generalist genes and learning disabilities. *Psychological Bulletin* 131(4):592.
- Plomin, Robert, John C. DeFries and John C. Loehlin. 1977. Genotype-environment interaction and correlation in the analysis of human behavior. *Psychological Bulletin* 84(2):309–322.
- Plomin, Robert, John C. DeFries, Valerie S. Knopik and Jenae M. Neiderhiser. 2012. *Behavioral genetics*. London: Macmillan Education.
- Pluess, Michael. 2015. Individual differences in environmental sensitivity. *Child Development Perspectives* 9(3):138–143.
- Polderman, Tinca J. C., Beben Benyamin, Christiaan A. de Leeuw, Patrick F. Sullivan, Arjen van Bochoven, Peter M. Visscher and Danielle Posthuma. 2015. Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics* 47(7):702–709.
- Protzko, John. 2015. The environment in raising early intelligence: a meta-analysis of the fadeout effect. *Intelligence* 53:202–210.

- Raffington, Laurel, Peter T. Tanksley, Aditi Sabhlok, Liza Vinnik, Travis Mallard, Lucy S. King, Bridget Goosby, K. Paige Harden and Elliot M. Tucker-Drob. 2023. Socially stratified epigenetic profiles are associated with cognitive functioning in children and adolescents. *Psychological Science* 34(2):170–185.
- Rimfeld, Kaili, Margherita Malanchini, Eva Krapohl, Laurie J. Hannigan, Philip S. Dale and Robert Plomin. 2018. The stability of educational achievement across school years is largely explained by genetic factors. *npj Science of Learning* 3(1):16.
- Schieman, Scott, Yuko Kurashina Whitestone and Karen Van Gundy. 2006. The nature of work and the stress of higher status. *Journal of Health and Social Behavior* 47(3):242–257.
- Segal, Nancy L., and Jean M. Russell. 1992. Twins in the classroom: school policy issues and recommendations. *Journal of Educational and Psychological Consultation* 3(1):69–84.
- Selita, Fatos, and Yulia Kovas. 2019. Genes and Gini: what inequality means for heritability. *Journal of Biosocial Science* 51(1):18–47.
- Selzam, Saskia, Stuart J. Ritchie, Jean-Baptiste Pingault, Chandra A. Reynolds, Paul F. O'Reilly and Robert Plomin. 2019. Comparing within- and between-family polygenic score prediction. *The American Journal of Human Genetics* 105(2):351–363.
- Shah, Sonia, Marc J. Bonder, Riccardo E. Marioni, Zhihong Zhu, Allan F. McRae, Alexandra Zhernakova, Sarah E. Harris, Dave Liewald, Anjali K. Henders, Michael M. Mendelson, Chunyu Liu, Roby Joe-hanes, Liming Liang, BIOS Consortium, Daniel Levy, Nicholas G. Martin, John M. Starr, Cisca Wijmenga, Naomi R. Wray, Jian Yang, Grant W. Montgomery, Lude Franke, Ian J. Deary and Paul M. Visscher. 2015. Improving phenotypic prediction by combining genetic and epigenetic associations. *The American Journal of Human Genetics* 97(1):75–85.
- Shanahan, Michael J., and Scott M. Hofer. 2005. Social context in gene–environment interactions: Retrospect and prospect. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences* 60(Special Issue 1):65–76.
- Shanahan, Michael J., Scott M. Hofer and Lilly Shanahan. 2003. Biological models of behavior and the life course. In *Handbook of the life course*, eds. Jeylan T. Mortimer and Michael J. Shanahan, 597–622. Boston, MA: Springer US.
- Silventoinen, Karri, Aline Jelenkovic, Reijo Sund, Antti Latvala, Chika Honda, Fujio Inui, Rie Tomizawa, Mikio Watanabe, Norio Sakai, Esther Rebato, Andreas Busjahn, Jessica Tyler, John L. Hopper, Juan R. Ordoñana, Juan F. Sánchez-Romera, Lucia Colodro-Conde, Lucas Calais-Ferreira, Vinicius C. Oliveira, Paulo H. Ferreira, Emanuela Medda, Lorenza Nisticò, Virgilia T occaceli, Catherine A. Derom, Robert F. Vlietinck, Ruth J. F. Loos, Sisira H. Siribaddana, Matthew Hotopf, Athula Sumathipala, Fruhling Rijdsdijk, Glen E. Duncan, Dedra Buchwald, Per Tynelius, Finn Rasmussen, Qihua Tan, Dongfeng Zhang, Zengchang Pang, Patrik K. E. Magnusson, Nancy L. Pedersen, Anna K. Dahl Aslan, Amie E. Hwang, Thomas M. Mack, Robert F. Krueger, Matt McGue, Shandell Pahlen, Ingunn Brandt, Thomas S. Nilsen, Jennifer R. Harris, Nicholas G. Martin, Sarah E. Medland, Grant W. Montgomery, Gonneke Willemsen, Meike Bartels, Catharina E. M. van Beijsterveldt, Carol E. Franz, William S. Kremen, Michael J. Lyons, Judy L. Silberg, Hermine H. Maes, Christian Kandler, Tracy L. Nelson, Keith E. Whitfield, Robin P. Corley, Brooke M. Huibregtse, Margaret Gatz, David A. Butler, Adam D. Tarnoki, David L. Tarnoki, Hang A. Park, Jooyeon Lee, Soo Ji Lee, Joohon Sung, Yoshie Yokoyama, Thorkild I. A. Sørensen, Dorret I. Boomsma and Jaakko Kaprio. 2020. Genetic and environmental variation in educational attainment: An individual-based analysis of 28 twin cohorts. *Scientific Reports* 10(1):12681.
- Spini, Dario, and Eric Widmer (eds.). 2023. *Withstanding vulnerability throughout adult life: dynamics of stressors, resources, and reserves*. Singapore: Springer Nature.
- Starr, Alexandra, and Rainer Riemann. 2022. Common genetic and environmental effects on cognitive ability, conscientiousness, self-perceived abilities, and school performance. *Intelligence* 93:101664.
- Starr, Alexandra, Mirko Ruks, Laura Weigel and Rainer Riemann. 2023. What drives the association between home chaos and school grades over time? A biometric cross-lagged panel approach. *Learning and Individual Differences* 104:102287.
- Stevenson, Anna J., Daniel L. McCartney, Robert F. Hillary, Archie Campbell, Stewart W. Morris, Mairead L. Bermingham, Rosie M. Walker, Kathryn L. Evans, Thibaud S. Boutin, Caroline Hayward, Allan F. McRae, Barry W. McColl, Tara L. Spires-Jones, Andrew M. McIntosh, Ian J. Deary and Riccardo E. Marioni. 2020. Characterisation of an inflammation-related epigenetic score and its association with cognitive ability. *Clinical Epigenetics* 12:1–11.

- Stienstra, Kim, Ineke Maas, Antonie Knigge and Wiebke Schulz. 2021. Resource compensation or multiplication? The interplay between cognitive ability and social origin in explaining educational attainment. *European Sociological Review* 37(2):186–200.
- Tan, Qihua, Lene Christiansen, Jacob von Bornemann Hjelmberg and Kaare Christensen. 2015. Twin methodology in epigenetic studies. *Journal of Experimental Biology* 218(1):134–139.
- Tilly, Charles. 2003. Changing forms of inequality. *Sociological Theory* 21(1):31–36.
- Tropf, Felix C., and Jornt J. Mandemakers. 2017. Is the association between education and fertility postponement causal? The role of family background factors. *Demography* 54(1):71–91.
- Tucker-Drob, Elliot M., and Timothy C. Bates. 2016. Large cross-national differences in genexsocioeconomic status interaction on intelligence. *Psychological Science* 27(2):138–149.
- Turkheimer, Eric. 2011. Genome wide association studies of behavior are social science. In *Philosophy of behavioral biology*, 43–64. Dordrecht: Springer Netherlands.
- Turkheimer, Eric, and K. Paige Harden. 2014. Behavior genetic research methods. In *Handbook of research methods in social and personality psychology*, eds. Harry T. Reis und Charles M. Judd, 159–187. Cambridge, UK: Cambridge University Press.
- Turner, Jonathan D., Conchita D'Ambrosio, Claus Vögele and Martin Diewald. 2020. Twin research in the post-genomic era: dissecting the pathophysiological effects of adversity and the social environment. *International Journal of Molecular Sciences* 21(9):3142.
- Verhulst, Brad, and Peter K. Hatemi. 2013. Gene-environment interplay in twin models. *Political Analysis* 21.3:368–389.
- Wang, Biyao, Jessie R. Baldwin, Tabea Schoeler, Rosa Cheesman, Wikus Barkhuizen, Frank Dudbridge, David Bann, Tim T. Morris and Jean-Baptiste Pingault. 2021. Robust genetic nurture effects on education: A systematic review and meta-analysis based on 38,654 families across 8 cohorts. *The American Journal of Human Genetics* 108(9):1780–1791.
- Wickrama, Kandauda A., Catherine Walker O'Neal, Tae Kyoung Lee and Seonhwa Lee. 2021. Early life course processes leading to educational and economic attainment in young adulthood: contributions of early socioeconomic adversity and education polygenic score. *PLoS One* 16(10):e0256967.
- Wolfram, Tobias, and Damien Morris. 2022. *Conventional twin studies overestimate the environmental differences between families relevant to educational attainment* [Preprint]. PsyArXiv. <https://doi.org/10.31234/osf.io/m4eqv>.
- Yehuda, Rachel, Nikolaos P. Daskalakis, Linda M. Bierer, Heather N. Bader, Torsten Klengel, Florian Holsboer and Elisabeth B. Binder. 2016. Holocaust exposure induced intergenerational effects on FKBP5 methylation. *Biological Psychiatry* 80(5):372–380.
- Young, Alexander I. 2019. Solving the missing heritability problem. *PLoS Genetics* 15(6):e1008222.
- Young, Alexander I., Stefania Benonisdottir, Molly Przeworski and Augustine Kong. 2019. Deconstructing the sources of genotype-phenotype associations in humans. *Science* 365(6460):1396–1400.
- Ziol-Guest, Kathleen M., Greg J. Duncan, Ariel Kalil and W. Thomas Boyce. 2012. Early childhood poverty, immune-mediated disease processes, and adult productivity. *Proceedings of the National Academy of Sciences* 109(supplement_2):17289–17293.

Publisher's Note Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Martin Diewald 1958, Prof. Dr., Faculty of Sociology, Bielefeld University. Research fields: social science genetics, life course, social inequalities, reconciliation of work and family life. Publications: Compensation or accentuation? How parents from different social backgrounds decide to support their children. *European Sociological Review* 2024 (with P. Dierker); Does social origin modify the heritability of cognitive ability? A close look at the relevance of different parental resources. *Research in Social Stratification and Mobility* 2023 (with B. Mönkediek and V. Lang).