Kristien Hens succeeds in weaving together experiential expertise of both people with autism and their parents, scientific insights and ethics, and does so with great passion and affection for people with autism (with or without mental or other disabilities). In this book she not only asks pertinent questions, but also critically examines established claims that fail to take into account the criticism and experiences of people with autism.

Sam Peeters, author of Autisti sche Gelukkig and Gedurfde vragen; blog @ Tistje.com

What does it mean to say that someone is autistic? Dynamics of Autism explores this question and many more. Kristien Hens conducts a thoughtful, wide-ranging examination of psychiatric, biological, and philosophical perspectives on autism, as well as its meanings to those who experience it, diagnose it, and research it. Hens delves into the history of autism to inform a contemporary ethical analysis of the models we use to understand autism and explores the various impacts of a diagnosis on autistic people and their families, the relevance of disability studies, the need to include autistic people fully in discussions about (and research on) autism, and the significance of epigenetics to future work on autism.

Rich, accessible, and multi-layered, this essential reading for philosophers, educational scientists, and psychologists who are interested in philosophical-ethical questions related to autism, but it also has much to offer to teachers, allied health professionals, and autistic people themselves.

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Cover Design by Anna Gaye.
This book has described different layers of the meaning of autism, ranging from psychiatric diagnosis to neurodevelopmental disorder to neurological identity. We have seen how, even from its inception, the two founding fathers of autism, Leo Kanner and Hans Asperger, conceive of autism’s essence differently. Although the children they described probably had the same phenotypical characteristics, autism was a child psychiatric and developmental phenomenon for Kanner. He described how behaviour gradually changed over time, how the children ‘extended ‘their cautious feelers’. For Asperger, autism was firmly rooted in one’s personality: he saw it as a trait, or even a disorder, with which one is born and dies. Autism, as a psychiatric diagnosis based on behavioural observation and an assessment of someone’s functioning, allows for certain flexibility: strictly speaking, not everyone with specific cognitive or emotional characteristics needs to receive a diagnosis of autism if these characteristics do not lead to suffering or dysfunction. Nevertheless, in everyday language and scientific papers, autism is often called an innate, genetic, and lifelong developmental disorder.

When considering autism, we often think about specific characteristics or peculiarities that a person might have, which might pose some challenges for them. Moreover, autistic adults often testify about how autism is intrinsically linked with their identity. Since the beginning of the history of autism as we know it, people have considered it a biological disorder. Partly as a reaction to psychoanalytical approaches in the fifties, which were stigmatizing for mothers of autistic children, scientific research into the genetics and biology of autism took flight. For

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the last forty years, indeed, most autism research focused on its causes, often in the hope that some ‘cure’ might be found. Over the last decade, this has changed somewhat: many researchers no longer consider autism something to be cured. Still, autism is primarily conceived of as a phenomenon rooted in genetics. In what follows, we will explore the link between autism and genetics and the meaning of genetics. I will challenge the idea that biology and genetics are necessarily fixed, and argue that the gap between our biology or genetics and our experiences is not that wide.

Conceptualising the Causes of Autism

In chapter nine, we have seen, following Ian Hacking and Erving Goffman, that by giving someone a specific diagnosis, we also change their future and their past. They become an autistic person. Here is an example from our interview study. The person in question was a fifty-two-year-old woman who had just received her diagnosis a week before the interview. She states about the diagnosis: ‘Yeah, I actually thought um... it’s going to be a loss situation like um... if I don’t have it, then it’s because of my past, and it’s a loss situation, and if I do have it, it’s also a loss situation because I, I want to be able to communicate correctly.’

This lady had had a challenging childhood; her parents mistreated her. She had always wondered whether the problems she experienced with social contact were due to her problematic past or the fact that she was different. The diagnosis of autism as an innate and lifelong condition was proof that the latter was the case. Because of this, she also permitted herself to be kinder to herself. She continues:

But I kind of already gave that up for a bit. I’m like, it will grow, uhm, but actually getting the diagnosis was a relief. I’m not putting myself down all the time anymore, yeah, and you can’t, and... I don’t blame myself as much anymore. I want to keep growing, and work on it. It’s not that I give up like, I’ll never be able to, no I want to be able to, but uhm, if I don’t succeed, I no longer give myself a beating. I’ve actually become more relaxed...

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2 Kristien Hens and Raymond Langenberg, Experiences of Adults Following an Autism Diagnosis (Cham: Palgrave Macmillan, 2018).
3 Hens and Langenberg, Experiences of Adults Following an Autism Diagnosis, p. 86.
As we have already discussed, how we conceptualise autism and psychiatric disorders as more or less biological or genetic has ethical consequences. Although very little is known about the causes of psychiatric illness in general, empirical research has demonstrated that people think that different psychiatric diagnoses have different levels of innateness (versus acquiredness) and a psychological basis (versus a biological one).

Delphine Jacobs is a child psychiatrist who researches how child psychiatrists and paediatricians view autism. As a PhD student, she investigated how the conceptualisation of autism as genetic and innate influenced how these clinical professionals looked at the prognosis and the possibility of improvement of symptoms. Clinicians who viewed autism as an innate and lifelong condition saw their clients’ future as already fixed and less amenable to change, and often thought that children had less control about their behaviour than children diagnosed with ADHD. People with a diagnosis that is seen as innate, genetic, and biological are thought to be less responsible for their deeds than people with a personality disorder. However, such conceptualisations also influence how people think about their responsibility towards people with a diagnosis. The more a specific phenomenon is seen as biological or innate, the more people think that medication is the best option to tackle the associated problems. Research also suggests that professionals may experience less empathy towards people with a diagnosis that they consider innate rather than psychological, although genetic explanations imply that these people would be less responsible for their deeds. Matthew Lebowitz and Woo-kyoung Ahn, the authors


of these studies, suggest that this may be the case because these people are considered less ‘human’ and more controlled by their genes.\(^6\)

In chapter two, we have discussed how psychiatric diagnoses are not ‘like diabetes’. However, the more a diagnosis of autism is thought of as similar to diabetes, the more the person diagnosed has to accept it as an inescapable condition. This can lead to less importance being attributed to the content, meaning, and understanding of individual behaviours if people consider them to be explicable in mechanistic-biological terms. Think about specific interests of autistic people that are seen as examples of stereotypical behaviour. *For The Love of Dogs* is a documentary published on Aeon about a twelve-year-old boy diagnosed with Asperger syndrome, who has an extraordinary interest in and knowledge about dog breeds. The filmmakers follow him to a big dog show where he enthusiastically interacts with breeders and dogs. At a specific moment in the film, an autism specialist speaks: this type of specific interest, so she said, can be explained because for an autistic person, the outside world is chaotic and frightening, and these interests help them to structure their world: ‘These children will use their particular narrow interests in order to reduce their anxiety’.\(^7\)

There may be some core of truth in this. However, at a certain point, the filmmakers decided to interview the dog owners at the show, not about autism, but about their love of dogs. All the other people at the dog show — probably without a diagnosis — shared the same level of interest: they also found comfort and support in their (interest in) dogs. Perhaps we can explain anyone’s specific interests or hobbies by referring to a need for structure and support in a frightening world. An overly deterministic and fatalistic explanation can also be dangerous in clinical practice. Clinical psychologist Evi Verbeke describes two cases in which autism was presented to recently diagnosed people, adolescents in this case, as a medical diagnosis (such as diabetes) that one has to accept and that is lifelong, rather than as an explanation of and an answer to

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the specific challenges that young people may experience at certain points in their lives. The diagnosis confronted these young people with existential questions about their own identity, which led to a worsening of their problems.\footnote{Evi Verbeke, ‘Diagnoses als mogelijke decompenserende factor’, T I J D S C H R I F T V O O R P S Y C H O A N A L Y S E , 2 2 : 4 ( 2 0 1 6 ) , 2 8 3 – 9 3 .}

Conceiving of autism as inherently genetic, innate, and lifelong can therefore also be problematic. Firstly, we only have limited knowledge about how autism develops throughout the lifespan, as there are only very few longitudinal studies that have been conducted. At the same time, the fact that autism is considered biologically real can help people with autism to accept their atypicality, limitations, and talents. Moreover, autism is real as a shared experience on a phenomenological level. Diagnosed people, their psychiatrists, and their peers will acknowledge that. Nevertheless, it would be wrong to mistake this phenomenological reality for a simple biological explanation, for example located in one gene or a specific part of the brain. This might lead us to consider autism simply as located in the individual and as static. However, we do not need genes or areas of the brain to acknowledge the reality of shared experiences. We might wonder if shared experiences are not ‘more real’ than genes or brain concepts. We may even ask ourselves to what extent genes themselves are real, and not merely linguistic representations of messy organic processes.\footnote{John Dupré, \textit{Processes of Life: Essays in the Philosophy of Biology} (Oxford: Oxford University Press, 2012).}

The Meaning of Genes

As I have discussed in the previous chapter, we may have to consider more dynamic conceptions of biology, which require the study of experiences. I previously described the thoughts of one of the godfathers of a more dynamic approach to pathology, George Canguilhem. Now, I want to dig deeper into one of the most commonly researched and discussed themes when dealing with autism: the link with genetics.\footnote{Majia Holmer Nadesan, ‘Autism and Genetics Profit, Risk, and Bare Life’, in \textit{Worlds of Autism: Across the Spectrum of Neurological Difference}, ed. by Joyce Davidson and Michael Orsini (Minneapolis: University of Minnesota Press, 2013), pp. 117–42.} Indeed, as Majia Nadesan has specified, the research
into explanatory autism genes is like the quest for the holy grail. This emphasis on genetic causes can probably partly be explained by the power of biological and genetic explanations as such. In their book *The DNA Mystique*, Dorothy Nelkin and Susan Lindee describe this as follows:

> Introductory biology is presented as a valid, truth-seeking endeavour, untainted by religious, political, or philosophical commitments. It places human beings in a meaningful universe, providing ways of understanding relationships between ethnic and racial groups and between identity and the body. Biology, in a very real sense, has become a philosophical and religious domain, and the genome itself has become a guide to the human condition.\(^{11}\)

After all, it is human to look for simple and understandable explanations of how one struggles. The authors describe how genetic essentialism has taken the place of earlier theistic explanations, but this can also be dangerous: genetic explanations remove individual and broader social responsibility.

The idea of autism as a genetic condition has always existed. Kanner and Asperger considered autism to be innate and saw similarities between parents and their children, although Kanner also acknowledged that there could be psychogenic causes. When the idea of the ‘refrigerator mother’ took root, parents welcomed genetic explanations. They saw these as proof that they were not ‘guilty’ and did not engender their child’s autism. Autistic people sometimes use genetics to demonstrate that autism is a natural and neutral variant with advantages and disadvantages.\(^{12}\) For some time, a distinction has been made between syndromic and idiopathic autism. In syndromic autism, autism is an expression of a genetic syndrome such as Fragile-X. Idiopathic autism is autism for which no genetic cause has been found. There is some hope that new insights concerning Copy Number Variants (CNVs) can blur this distinction. However, it is also possible that in the end, only common variants will be found, which are also present in the general

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non-diagnosed population. A great amount of research money has been poured into the search for the genetic origins of autism. Genetic researchers have associated almost every chromosome with autism, with moderate results. The fact that the vast majority of genetic findings are risk factors, not definite causes, raises the question why no more research is done into the environmental factors that are supposed to contribute to autism.

Genetics is one of the most researched topics in bioethics. Bioethics is a branch of philosophy that deals with the ethical implications of technological developments and research findings in biology and medicine. Specifically, many bioethicists research questions about the ethical implications of genetics. Should we screen embryos genetically, select or even modify embryos to make healthier children or even children in which certain characteristics such as intelligence are enhanced? For which diseases should prenatal genetic diagnosis and termination of pregnancy be allowed? What genetic information should be detected and communicated with people who are already born? We cannot answer these questions without thoroughly reflecting on the underlying concepts. What do we mean by ‘disease’? Which risks are we allowed to take when we introduce new genetic technologies? What about the right not to know? What does responsibility mean? Do parents have the responsibility to choose the children with the most desirable characteristics, or should we accept future children as they are? If we look at autism through a genetic lens, such questions are no different from those asked about other conditions.

I researched the ethical aspects of genetic research and counselling in autism some years ago. I interviewed several Belgian psychiatrists, educational specialists, psychologists, and geneticists who dealt with autism daily. From my research, it became clear that these people had many questions about fundamental genetic research into autism. Firstly, there is the question of who can participate in such research. A diagnosis of autism is a clinical diagnosis. People with the same

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genetic characteristics do not necessarily all receive the same diagnosis, and people with the same diagnosis do not necessarily have the same underlying biological characteristics. People thought that the diagnosis itself was not sufficiently fine-grained to enable useful genetic research. Some were also concerned that genetic findings would pave the way towards a policy of prevention:

We are giving an ambiguous message. On the one hand we say that it is not bad to have it, on the other hand we say it is better to prevent it. Especially for the people with ASD I believe that it is a kind of ethical dilemma, is it bad to have it or not. I doubt that geneticists ever wonder about this, but they should.15

I am very ambiguous about this one. I am thinking, are we then going to develop drugs to make them all normal? I find that very difficult, because I do not believe in medication for that, especially not in developmental disorders.16

If we are talking about clinical genetic research in children or families in which, through psychiatric diagnostics, autism has been established, respondents often stress the power of genetics to remove blame or feelings of guilt.17 Hence, the fact that a biological-genetic cause can be attributed enhances the deculpabilising effect of the diagnosis itself:

The advantage of looking for a gene, it can mean a lot for these people if they are given a cause, or a reason why something goes wrong, a reassurance that it is not the way they raise the child, or because they have smoked or drank, the question of who is to blame. That is the positive side of the story. Except if you are dealing with a condition with an inherited susceptibility.18

Another advantage of finding a genetic cause for autism is that clinicians can explain to a family what is the risk that their next child will receive the same diagnosis. Here we enter rugged ethical terrain. We can ask whether autism is grounds for embryo selection, or prenatal

15 Hens, Peeters, and Dierickx, ‘Shooting a Moving Target’, p. 35.
16 Ibid.
diagnostics and pregnancy termination, and who decides that. Majia Nadesan describes an autism genocide clock that was available online for some time:

The possibility that gene-based susceptibility tests might be developed has raised considerable concern within particular subsets of the autism-advocacy movement. For example, for a time, there existed online an "autism genocide clock" that purported to count down years, days, and minutes to the seemingly inevitable developments of an autism prenatal test that would result in an autism holocaust. This clock was uploaded in 2001 in response to concerns that genetic knowledge about autism would lead to the patenting of susceptibility genes, which in turn could be used to develop commercial prenatal tests.¹⁹

Indeed, in 2018 a patent was approved for the development of diagnostic tests. This patent was based on a gene suspected of playing a role in the development of autism.²⁰ These diagnostic tests could not only identify a genetic cause in someone with a diagnosis of autism but could also reduce or even replace current diagnostic practices. Often, diagnosticians say that they would like to have clearer ways of diagnosing autism and prefer to have a more reliable diagnosis by using genetic markers. However, such an endeavour also has several challenges. Firstly, there is the fact that genetic factors are risk factors. Autism is a diagnosis given at the level of the phenotype based on behaviour. A diagnostic test based on genetics is, by definition, impossible within the current DSM-5 definition of autism. Furthermore, although a straightforwardly genetic and ‘somatic’ diagnosis of autism may seem to be more scientific than a behavioural diagnosis, as it would take away some uncertainty and avoid any element of guesswork, it would also bring new and complex challenges, some of which I have described earlier in the book. Collapsing psychiatric diagnosis with genetics may mean that experiences, which are also symptoms of autism, are neglected by caregivers. Behaviour that is directly explained by genetics may become more challenging to incorporate as part of one’s identity. It is, however, precisely because autism is flexible and, at the same time, a shared

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experience that the diagnosis can work therapeutically. Moreover, there will always be people for whom no genetic ‘cause’ can be found, but for whom a diagnosis is still helpful.

Dynamic Genetics: The Strange Case of Epigenetics

Autism seems to be, to no small extent, familial, and a large amount of scientific resources are allocated to research into the identification of autism genes. Decades-long genetic research has, however, not provided a direct causal explanation for autism. It looks as if environmental factors play an essential role in the development of the autistic phenotype. It is probably the interaction between genes and environment that can result in atypical development and the challenges that lead to a diagnosis of autism. There are probably also protective factors in the background that mean someone with a genetic susceptibility to develop autism may never receive the actual diagnosis. There have been many prenatal, perinatal, and postnatal factors associated with autism. Early socio-demographic factors such as income, education, and employment of the parents can influence the probability of a subsequent diagnosis. Factors that influence the chance that people receive a diagnosis later in life have not been researched extensively.

Nevertheless, we can state that merely looking at genetic and neurological factors within the individual is not sufficient to predict that someone will get a diagnosis. The explanation of why research into environmental factors is still in its infancy has probably to do with the questionable and unscientific status of some of the earlier claims. On the one hand, the ‘refrigerator mother’ idea has led to problematic stigmatization of parents, specifically mothers. On the other hand, the faulty suggestion that a simple environmental factor such as vaccination may cause autism has also had far-reaching and disadvantageous effects.

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11. Autism and Genetics

It may make more sense to look at genetics from a dynamic perspective. We do not look for one etiological explanation in early development, but we consider an individual as continuously in interaction with their environment. In what follows, I will explain epigenetics and how we can use this, as ethicists, to look differently at certain phenomena such as autism. What follows is a translation of a piece I wrote for the Dutch periodical *Karakter.*

In a recent article in *Science*, Adam Klosin and colleagues describe how environmental factors influence gene expression and how these changes can be passed on to subsequent generations. To examine this, they use a transgene *C. elegans.* The roundworms were genetically modified to light up if they arrived in a warmer environment. If the worms were in an environment of twenty degrees Celsius, they glowed a little bit. When it became warmer, the gene that caused the fluorescence was switched on, and the worms started to glow more brightly. The fact that environmental factors influence gene expression is in itself not striking. When the temperature lowered again, the worms kept their intense glow. Furthermore, their descendants inherited the glow, and for seven subsequent generations, glowing worms were born. When *C. elegans* were kept in a warm environment for five generations, the glow characteristic was passed on to fourteen generations. This looks much like the inheritance of acquired characteristics as it was suggested by the French naturalist Jean-Baptiste Lamarck (1744–1829) but later discredited by Neo-Darwinism and modern genetics. It goes against what we call the ‘central dogma’ of genetics. This central dogma states that the transcription and translation of DNA to RNA and subsequently to proteins is one-way traffic. Inheritable changes in DNA, so people thought, can only happen by mutations in the genes themselves.

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25 *C. elegans* (Caenorhabditis elegans) is a roundworm that is 1 mm long and that is often used in genetic research, because the animal has a relatively simple genome and reproduces quickly so that generations quickly follow after another.
Of course, it has been known for a long time that this is not the whole story. The mechanism that is described above is known under the term ‘epigenetics’. Skin cells and brain cells perform different functions. Hence, different genes must be expressed. Therefore, based on the environment in which the cell is found, there should be a mechanism that influences which genes express themselves and which do not. In 1911, Wilhelm Johanssen (1857–1927), who first named the distinction between genotype and phenotype, suggested that identical genotypes can produce different phenotypes. In 1942, Conrad Waddington (1905–1975) suggested using the term ‘epigenetics’ to describe the mechanisms involved in gene expression. ‘Epi’ is the Greek word for ‘on, with’. In recent decades, the study of this epigenetic layer has taken off rapidly. One of the most studied mechanisms in this context is methylation, which occurs above the DNA level. If methyl groups (as small carbon compounds) are added or removed from specific regions, genes become accessible for transcriptions. Techniques such as Genome-Wide Methylation Analysis allow for the study of methylation patterns.

Epigenetics is extremely interesting for bioethicists who are reflecting on genetics. Often, in discussions about the impact of genetics, the unidirectional model promoted by the ‘central dogma’ is assumed. Genes may be edited through CRISPR/Cas9, or embryos can be selected based on the ‘best’ genotype. Bioethicists sometimes think about the influence of environmental factors, for example, in discussions about the extent to which we can force pregnant women to have a healthy lifestyle. These environmental factors are often perceived as secondary because they are considered changeable. Recent epigenetics findings suggest the molecular link between our genetic blueprint and the environment, between nature and nurture.

Moreover, epigenetic changes resonate for a long time, even after the individual has moved on from their earlier environment. It is likely that specific changes are also passed on to future generations. Techniques such as CRISPR/Cas9 that allow us to change pieces of DNA directly are somewhat invasive. Targeted manipulation of the layer above the DNA is perhaps a much easier way to decide which genes can be expressed and which cannot. In this way, specific epigenetic changes could be undone, a method that is being investigated in research carried out into certain cancers.
All these aspects of epigenetics (inheritance, reversibility, molecular linking of environmental factors) influence how we think about responsibility. Who is responsible for the impact of societal change on individuals and vice versa? It has been demonstrated that pollution through particulate matter can induce epigenetic changes, with potentially severe consequences for the health of children yet to be born. However, we might wonder who or what causes the fact that many people can only afford to live close to the highway or in polluted areas. Moreover, even if people move out of polluted areas, will the molecular changes still affect future generations? It is self-evident that parents are responsible for their children’s health, but the question is how far they can control certain factors themselves.

An intermediate conclusion could be that our level of responsibility is disproportionately inflated if we take the implications of epigenetics seriously. The fact that living close to the highway is not healthy is common knowledge. The idea that breathing in particulate matter can resonate on a molecular level, and that harm has already been done by the time we determine the levels of pollution, can lead to an unbearable sense of guilt on the one hand or moral defeatism on the other. If we read scientific literature about epigenetics, we do indeed find terminology that implies blame and responsibility. In 2005, Marcus Pembrey and his colleagues found that when boys start to smoke at a young age, this affects the BMI of their sons born later in life.26 A commentary on this phenomenon in Nature is titled “The sins of the fathers and their fathers”.27 Again in Nature, Richardson and her colleagues warned in 2014 how reporting epigenetic findings can lead to pregnant women becoming overburdened with guilt: they would be held responsible for epigenetic changes during pregnancy.28 Indeed, a couple of years ago, newspaper headlines reported on findings that eating an English breakfast (particularly bacon and eggs) at the beginning of pregnancy could increase the intelligence of the future child.29

Ethicists may wonder if mothers have a duty to ensure that their offspring are as bright as possible. The environment in utero seems to be susceptible to epigenetic influences. Mothers, who traditionally already bear the most significant responsibility for their baby’s welfare, risk also being held responsible for their child’s future health and that of these children’s future children. Research in mice has demonstrated that stress during pregnancy influences the BMI of offspring and can also lead to hyperactive behaviour in offspring. However, this finding does not automatically lead to the conclusion that women must give up a stressful job during the entire pregnancy. Perhaps the emphasis in the media on scientific research into environmental factors in utero is unbalanced. Other factors, for example, the impact of the quality of the sperm of the biological father, might be neglected too often. People frequently forget that environmental factors do not only exert influence during pregnancy. As already mentioned, epigenetic changes in the primordial germ cell of teenage boys, caused by activities such as smoking or drinking, can be passed to the sperm cells they produce. These changes are cumulative during a lifetime. That it is better not to smoke or drink as a teenager is self-evident.

The idea that fifteen-year-old boys could be persuaded not to do this out of a sense of duty towards the health of their future children and grandchildren might be a stretch too far. We must avoid a kind of epigenetic determinism, where we replace a single genetic explanation with a simplistic epigenetic explanation. Such substitution will lead to an unwarranted emphasis being placed on individual responsibilities. If we take epigenetics seriously, we must adopt a complex and dynamic view of organisms, acknowledging the multicausal nature of behaviour. We must also take seriously the impact of experiences on biology, which is often outside of our control. Such a systemic view of the functioning of organisms suggests that epigenetics ought to play a role in the decisions of policymakers. It is, among other things, a wake-up call that the pernicious effects of environmental pollution have systemic and long-lasting effects and need a systemic solution.

The fact that researchers have found such a molecular link between nature and nurture will undoubtedly influence how we see ourselves as human beings in relation to our environment; The image of the human being as built up from a genetic blueprint, only fleetingly influenced
by our milieu, is being challenged. Human organisms become dynamic entities in interaction with the environment on a molecular level. This thought is, of course, not new. As I have previously described, Canguilhem and others presented more systemic approaches to human life. Nowadays, Developmental Systems Theory scholars, drawing on findings in epigenetics, defend the idea that human nature is the result of the entire organic milieu in which development takes place, challenging the primacy of genetics. A human being is not something atomistic or universal. As such, we come close here to the concept of epigenesis.

Epigenesis is at first only tangentially related to epigenetics. It ties into a century-old discussion relating to the form of organisms. Epigenesis means that an organism’s form is not wholly predetermined from the start (as preformationists would say). It is shaped by influences from inside of an organism: for example, the location of a cell in the body influences the function it performs, but so do external influences. In this sense, an epigenetic approach is a developmental approach: organisms are always in development, not solely when they are young. Their nature and functioning are thoroughly influenced by what they experience on their path through life. Hence, modern-day epigenetics could be seen as a vindication of the age-old concept of epigenesis.

Such a developmental approach to what it means to be human also has ethical implications. Autism might be a good example of this. After decades of research into its genetic origins, a consensus is growing that environmental factors and epigenetics play a role. Moreover, autism is heterogeneous and complex, referring to a wide array of cognitive functioning and behaviour that is variable across a lifetime and has a varying influence on wellbeing. Autism is, in the first place, a behavioural diagnosis that is attributed according to DSM-5 guidelines. Simultaneously, autistic people sometimes argue that it is not a condition or disease, but a neutral genetic variant that needs to be accepted and accommodated. Furthermore, epigenetic findings suggest that autism is a genetic adaptation that could be triggered by changes in the environment.

Nevertheless, I am convinced that the emphasis that people sometimes now put on unhealthy environmental factors and lifestyle as a cause of autism, through which it is suggested that autism is due to something that has gone wrong or something that could be avoided, is
misguided and may even be an example of the epigenetic determinism described above. On the contrary, an epigenetic view of organisms demonstrates that searching for simple causes of certain behaviours is naive.

We might consider whether there is something like ‘epigenetic normality’, a baseline from which we can measure deviations. In a seminal paper, Charles Dupras and Vardit Ravitsky ask this question. They give the example of obesity, which is associated with malnourishment during pregnancy: the foetus reacts to this with epigenetic changes that allow for more efficient storage of nutrients. If the child subsequently grows up in normal circumstances, there is an increased likelihood of obesity. Perhaps similar mechanisms are at work in autism. This might support the argument that the challenges and suffering of some autistic people are due as much to the broader environment in which they are situated as their neurological atypicality. As already demonstrated in the context of environmental factors and the responsibility of the pregnant mother, it would be incorrect to replace one explanatory model (the genetic one) with another (a specific environmental factor). Dysfunctioning and functioning are the results of complex interactions, of which we may only know the tip of the iceberg and which are not solely located within the individual. When we appreciate the impact of epigenetics fully, we can assume that there is a molecular basis for a complex, systemic, and plastic concept of human beings, which dynamically change their environment and are being changed by it. This concept is moreover ethically relevant to the aims and methods of medicine. We can question biomedical research that merely seeks to discover the one cause of autism in genes or specific environmental factors. It makes more sense to encourage approaches that consider the autistic person and the challenges they may experience in a particular context, and consider how they cope and can cope with this context. In this way, we can fully appreciate autism as a developmental phenomenon.