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Trends in
Ecology & Evolution

Opinion

Exploring the interplay of epigenetics and individualization

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The interplay between interindividual phenotypic variation and epigenetic processes remains poorly understood in an ecoevolutionary context. Epigenetic variation is a key driver of individual differences, with genetically regulated, environmentally induced, and stochastic epigenetic variation playing distinct yet interacting roles in promoting the processes leading to individualization. In turn, the realization of individualized niches can induce novel epigenetic variation through the exposure of individuals to new environmental conditions. Epigenetic variation underlying or resulting from individualization alters patterns of selection and thereby affects long-term evolution, partly through transgenerational effects. Studying this bidirectional causal relationship will deepen our understanding of the proximate mechanisms underlying individualization and elucidate how epigenetic variation arises, persists, and contributes to evolution.

Highlights

Recognizing interindividual differences has significantly improved our understanding of ecoevolutionary processes. However, the mechanisms underlying these differences, as well as their long-term evolutionary consequences, remain poorly understood.

Being both a driver and a consequence of the interaction between individuals and their environment, epigenetic variation could be key to niche individualization.

The consequences of altered epigenetic variation for individual phenotypes result in different patterns of selection, affecting long-term evolution.

Integrating our perspective on the bidirectional causal relationship between epigenetic variation and individualization into empirical research will improve our understanding of ecoevolutionary processes.

Individualization and epigenetics

Considering individual phenotypic differences within and between populations enhances our understanding of ecological and evolutionary phenomena [1–3]. Differences in individual needs also shape how phenotypes respond to environmental changes [4]. Recognition of the importance of integrating individual differences into evolutionary and ecological research and theory has led to the conceptualization of the **individualized niche** [5] (see Glossary). Individualized niches are realized through three core processes termed **niche choice, niche construction,** and **niche conformance** [5,6] (Box 1). Although these processes are well documented [5,6], their molecular underpinnings remain poorly understood.

Parallel advances in **epigenetics** (Box 2) have revealed dynamic mechanisms that regulate gene expression and function in many organisms [14]. However, despite progress in this emerging field, many fundamental questions remain unanswered. For instance, it is still unclear why only parts of the epigenome respond to the environment, persist across generations, and have phenotypic, ecological, or evolutionary consequences [30]. These questions are difficult to answer because **epigenetic variation** is inherently complex. For example, epigenotypes can differ among individuals, even within genetically unstructured populations [31], and epigenetic and genetic variation often exhibit reciprocal and functionally interdependent relationships [27].

Building upon the idea that epigenetic variation contributes to phenotypic differences among individuals [32,33] and that niche processes can themselves trigger changes in internal or external environments that induce epigenetic changes [34–36], we hypothesize that niche processes and

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Box 1. Individualized niches and the processes that shape them

An **individualized niche** is the subset of a species' niche realized by a single individual and represents the range of biotic and abiotic conditions under which it can survive and reproduce [5]. It emerges from the interaction between the individual and its environment, affects the phenotype–environment match, and consequently, its fitness. Individualized niches are dynamic, requiring adjustment as phenotypes and/or environments change over ontogeny. Such changes often create **phenotype–environment mismatches**, which, regardless of their intensity, are defined as situations that, if resolved, would increase fitness [5]. Niche choice, niche construction, and niche conformance are the processes that individuals can adopt independently, concurrently, and/or consecutively to resolve these mismatches and reshape their individualized niches. Niche processes are generally not limited to specific phenotypic traits or single life stages but, due to developmental constraints, they often have greater potential to resolve mismatches that emerge during sensitive periods in early ontogeny.

Niche choice occurs when an individual selects a physical or social environment that better matches its phenotype. Niche choice behaviors include habitat choice, as in arboreal anole lizards (*Anolis* spp.), where individuals choose perching sites to optimize the experienced temperature and their camouflage [7]. Niche choice also occurs when individuals select specific parts of the environment to interact with, for example, through the choice of resources or social groups. For instance, in Trinidadian guppies (*Poecilia reticulata*), consistent among-individual differences in the selection of social interaction partners influence the size and strength of social networks [8].

Niche construction involves an individual actively modifying its environment to better match its phenotype. Examples include dam building in beavers (*Castor fiber*), nest building in birds, and soil modification by earthworms [9]. Individuals can also alter their social environment, as in paper wasps (*Polistes gallicus*), where subordinate females can challenge and replace dominant queens of the colony [10].

Niche conformance is the process by which an individual's phenotype is altered to optimize its match with the environment. While caused by phenotypic **plasticity**, conformance emphasizes interindividual variation in plastic responses [11], which may be important for the formation of individualized niches when a population is exposed to the same environmental changes. Niche conformance may entail irreversible phenotypic changes, as in water fleas (*Daphnia cucullata*), where inducible defenses during development arise in response to perceived predation risk [12], or reversible changes, as in many plants that increase osmolyte concentrations under drought [13].

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epigenetic variation are causally and bidirectionally linked. Epigenetic variation can change in response to environmental cues, providing mechanistic links between the environment, the epigenome, gene expression, and phenotype that enable niche realization [34]. In addition to **environmentally inducible** and **stochastic epigenetic variation, genetically regulated epigenetic variation** can also influence the realization of individualized niches. Genetic polymorphisms affecting epigenetic regulation [37] or the activity of histone modifiers shape the responsiveness of genotypes to environmental cues, thereby potentially modulating individual tendencies toward niche choice, conformance, or construction. Thus, genetically regulated epigenetic variation interacts with inducible mechanisms to generate consistent individual differences in niche realization [27,28,34,37].

Integrating the concept of the individualized niche with epigenetics may, therefore, help to explain hitherto poorly understood patterns and dynamics of interindividual epigenetic variation in natural populations. We argue that investigating epigenetic and phenotypic variation in the same individuals will deepen our understanding of how such differences arise, interact, and are maintained. Drawing on empirical evidence, we explore the implications of this perspective across ecological and evolutionary timescales and highlight future directions for the emerging field of individualized epigenetics.

Epigenetic mechanisms as drivers of niche processes

Individualized niches emerge through interactions between individuals and their environments. Although epigenetic variation does not always lead to phenotypic variation, variation affecting genes underlying phenotypic traits involved in niche choice, conformance, or construction may lead to niche realization. Individual differences in epigenetic variation can arise in different ways (Box 2). First, epigenetic differences may be genetically regulated, emerging from genetic

Box 2. Epigenetics

The term **epigenetics** refers to biochemical mechanisms that alter gene expression, thereby changing phenotypes without altering the DNA sequence [14]. This is not necessarily true for evolutionary timescales, where epigenetics can bias sequence changes [15]. **Epigenetic marks** and **epigenetic regulators** can enhance or reduce gene transcription and translation [14]. Epigenetic marks are chemical modifications, such as DNA methylation—the addition of a methyl group to a nucleotide base [16]— or histone modifications, which occur on histone tails and alter DNA accessibility for transcription [17]. Different epigenetic marks can also interact with each other [18]. Epigenetic regulators establish, interpret, or remove marks and mediate other processes regulating gene expression [19]. Beyond the enzymes that establish epigenetic marks, a well-known epigenetic regulator is RNA interference, where small [20] or long RNA molecules (siRNA or miRNA, respectively) [21] regulate gene expression by targeting mRNA for degradation or translational repression. Interindividual differences in epigenetic marks and regulators produce epigenetic variation.

Epigenetic variation can be genetically regulated, stochastic, or environmentally inducible [22,23]. Genetically regulated variation arises from underlying genetic variation. Stochastic variation occurs spontaneously and independently of environmental cues. Although considered to be selectively neutral [24], stochastic variation can be detrimental or beneficial, analogous to genetic mutations [25]. By contrast, environmentally inducible variation (subsequently referred to as 'inducible variation') is triggered by environmental factors. The effects of both stochastic and inducible variation differ from those of genetic mutations, as the rate of inheritance of epigenetic variants is three to four orders of magnitude lower [reviewed in Ref. 26]. Genetically regulated and inducible variation are not necessarily mutually exclusive, as the potential for epigenetic responsiveness is also controlled by the density and distribution of the sites in the DNA sequence that can be affected by marks or regulators. For example, across vertebrates, epigenetic variation is often higher for genes involved in regulation and stress responses [27–29].

variation. Second, they may be stochastic, emerging spontaneously and independently of the environment, for example, during early development. Third, they may be environmentally inducible, as commonly studied in the context of phenotypic plasticity. Importantly, epigenetic responsiveness can itself be controlled by genetic variation.

In contrast to genetically regulated epigenetic variation, which likely has similar consequences for niche realization as genetic variation itself, we suggest that inducible and stochastic epigenetic variation differ in their importance for individualized niche realization. First, stochastic variation arises spontaneously and is often selectively neutral [24]. However, it may be non-neutral, and when transmitted across generations, it could encode information about past selection regimes [26]. Second, newly emerging induced epigenetic variation is also frequently neutral or maladaptive. However, on evolutionary timescales, selection often favors adaptive phenotypic plasticity in fluctuating environments [38]. Hence, past selection on phenotypes should have rendered much inducible variation functionally significant. Accordingly, we argue that inducible variation is generally more relevant for the realization of individualized niches than stochastic variation, which often remains functionally neutral.

We further hypothesize that genetically regulated, stochastic, and inducible epigenetic variation differ in their impacts on the three core processes underlying niche realization. Specifically, phenotype–environment mismatches can trigger changes in inducible epigenetic variation, leading to phenotypic adjustments. Because niche conformance involves individuals modifying their phenotypes to resolve such mismatches, it is likely to be mediated by epigenetic variation that is induced by the mismatch itself (Figure 1) and which mediates the phenotypic changes underlying niche conformance (Box 3). By contrast, during niche choice and construction, individuals actively select or construct their niche to resolve a phenotype–environment mismatch. As these processes rely on immediate individual responses (i.e., to choose or alter the environment, respectively), they are likely influenced by epigenetic variation existing prior to the phenotype–environment mismatch. Accordingly, we expect that early-life inducible variation, as well as genetically regulated variation and stochastic variation, shapes niche choice and construction (Figure 1).

Glossary

Ecological inheritance: inheritance of the parental environment and its inherent processes, and effects on individuals, potentially including epigenetic variation induced by the environment in the offspring generation.
Ecotype: a subset of a species that survives as a differentiable group under local environmental conditions, potentially because of specific genetic and/or epigenetic adaptations.
Epigenetics: biochemical mechanisms and modifications that induce changes in gene expression and function without altering the DNA sequence.
Epigenetic variant: different variants of a gene that are only distinguished by their epigenetics.
Epigenetic buffering: the process by which populations enduring challenging conditions maintain or generate high levels of epigenetic variation, increasing the likelihood of coping with environmental stress and enhancing population persistence.
Epigenetic mark: chemical modifications made to DNA or histone proteins that influence gene expression without altering the DNA sequence.
Epigenetic regulator: protein, enzyme, or molecular complex that writes, reads, or erases epigenetic marks, orchestrates changes in chromatin structure, and affects transcription.
Epigenetic variation: reversible interindividual differences in the biochemical changes (both epigenetic marks and epigenetic regulators) that modify gene expression in the absence of changes to the DNA sequence.
Experience-mediated inheritance: the process by which environmentally induced changes in the parental phenotype induce epigenetic variation in their offspring, for example, through altered parental care.
Environmentally inducible epigenetic variation: epigenetic variation at a defined genomic location that is controlled by the environment.
Genetic assimilation: the process whereby a phenotype initially induced by the environment becomes genetically encoded.
Genetically regulated epigenetic variation: epigenetic variation that arises nonrandomly as a consequence of genetic variation.
Germline epigenetic inheritance: the process by which epigenetic variation is

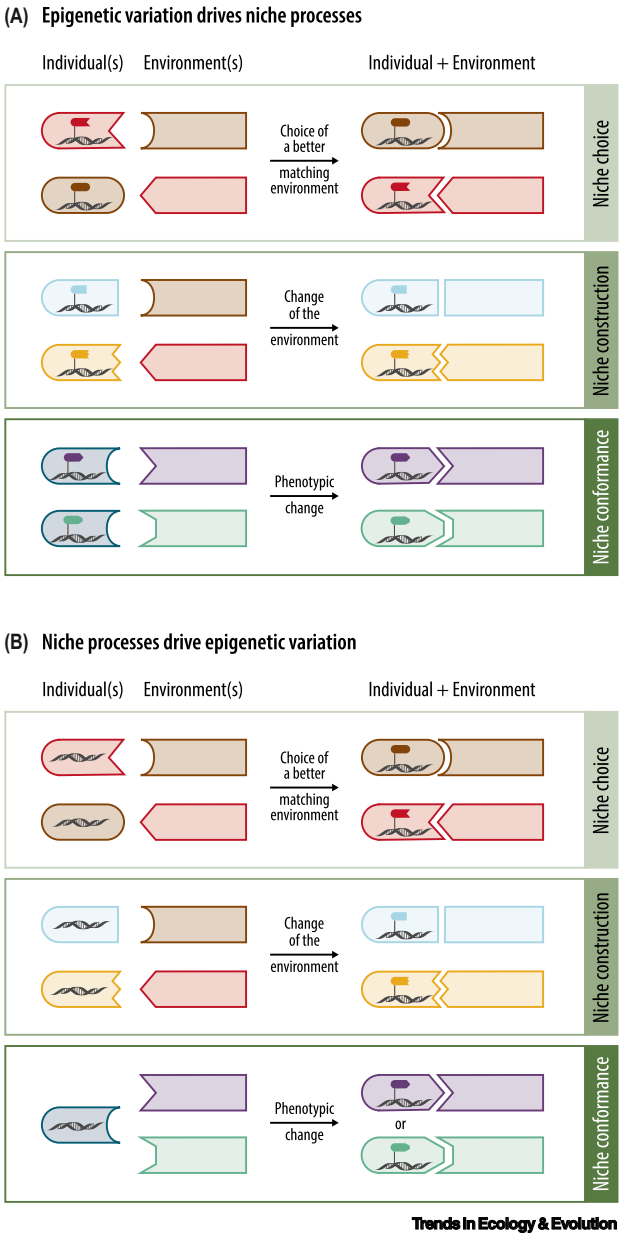

Figure 1. Bidirectional causal relationships between the three processes involved in realizing individualized niches and epigenetic variation. Individuals encounter environmental conditions that either match or mismatch their phenotype. Through niche choice, construction, and/or conformance, individuals achieve a suitable match with their environment, leading to the realization of their individualized niche. Epigenetic variation can drive these processes (Panel A), as it can predetermine niche choice and construction or enable niche conformance. Yet, epigenetic variation can also emerge as a consequence of niche processes (Panel B), whereby exposure to novel environmental conditions can promote the emergence of environmentally inducible epigenetic changes. Within individuals, shapes above the DNA strands represent epigenetic variants. The shapes and colors on top of genotypes and those of individuals denote their epigenotypes and associated phenotypes. Thus, matching shapes and colors on the environmental level indicate an improved phenotype–environment match.

transmitted from one generation to the next through germline cells.
Individualized niche: the range of environmental conditions under which a specific individual with a given set of traits could possibly live and reproduce. It is a subset of the species' niche that arises from the interaction of the individual with its environment.
Niche choice: the process through which an individual selects an environment to increase its phenotype –environment match and fitness.
Niche conformance: the process through which an individual adjusts its phenotype to increase its phenotype –environment match and fitness.
Niche construction: the process through which an individual modifies the environment to increase its phenotype –environment match and fitness.
Phenotype–environment mismatch: a situation in which the environmental conditions encountered by an individual do not fit its phenotype, triggering an individual response (niche choice, niche construction, or niche conformance) aimed at resolving the mismatch.
Plasticity: the ability of a given genotype to generate distinct phenotypes. Plasticity can occur within a single generation or inter-/transgenerationally when offspring phenotypes are altered by the environment experienced by the parents or previous generations.
Stochastic epigenetic variation: epigenetic variation that arises spontaneously independent of the environment and is a potential target of natural selection.

Inducible epigenetic variation is often developmentally constrained, with distinct ontogenetic windows, such as embryogenesis, metamorphosis, or early postnatal life showing heightened responsiveness to environmental cues [14]. Environmental inputs during these sensitive phases can leave stable molecular signatures that persist into adulthood, as seen in maternal care-dependent epigenetic variation in rodents and temperature-driven sex differentiation in fish [47,48]. Such early-life induction may serve as anticipatory plasticity, aligning developmental trajectories with the anticipated environmental conditions and promoting later-life niche processes [49,50]. This epigenetic variation may thus predispose individuals to respond in particular ways to phenotype–environment mismatches, influencing individual decision-making processes underlying niche choice and construction.

Box 3. Empirical examples of how epigenetic mechanisms may mediate the realization of individualized niches

Niche choice in capelin

Capelin (*Mallotus villosus*; Figure I) exhibit two ecomorphs differing in spawning habitat: one spawns near the ocean floor, while the other adopts a beach-spawning tactic. These ecomorphs show epigenetic differences [36]. Because temperature influences DNA methylation during embryonic development in fish [39], and the two habitats differ substantially in temperature, these epigenetic differences may arise during early life and predispose individuals to choose a particular spawning habitat.

Figure I. Capelin (*M. villosus*). Photo credits: By Ryan Hodnett—Own work, CC BY-SA 4.0, https://commons.wikimedia.org/w/index.php?curid=89571629.

Niche construction in cichlids

Males of an African cichlid fish (*Astatotilapia burtoni*; Figure II) engage in social niche construction by establishing dominance hierarchies through aggressive behavior, which governs reproductive access [40]. While two paralogous androgen receptor genes appear to control social status [41], experimental manipulation of global DNA methylation using methylating or demethylating agents significantly alters an individual's likelihood of ascending in rank [42]. This suggests that variation in DNA methylation may influence social behavior and contribute to the establishment of social status.

Figure II. Dominant *A. burtoni* male. Photo credits: By (Image: Russell D. Fernald and Sabrina S. Burmeister)—Social opportunity produces brain changes in fish. PLoS Biology Vol. 3/11/2005, e390 doi:10.1371/journal.pbio.0030390, CC BY 2.5, https://commons.wikimedia.org/w/index.php?curid=1431648.

Niche conformance in nematodes

The ground-dwelling nematode (*Caenorhabditis elegans*, Figure III) has evolved the ability to conform to stressful conditions, both within and across generations. Exposure to starvation stress induces adaptive developmental arrest through changes in siRNA expression [43]. These siRNAs interact with specific histone modifications [44] and can be transmitted transgenerationally through germline epigenetic inheritance, targeting genes related to nutrition in consecutive generations [45] and increasing starvation resistance [46].

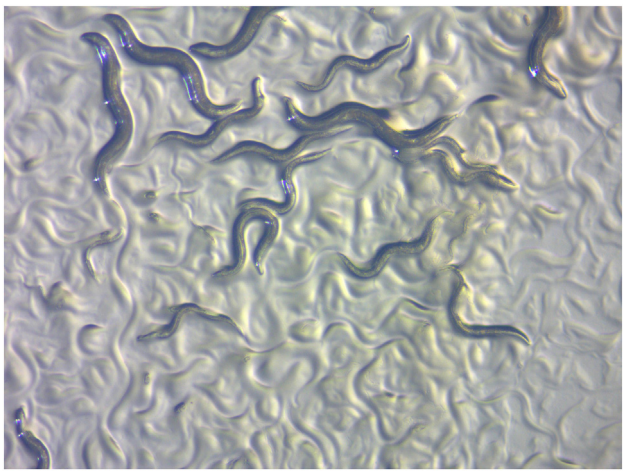

Figure III. *C. elegans.* Photo credits: By ZEISS Microscopy from Germany—*C. elegans*, model organism in life sciences, CC BY 2.0, https://commons.wikimedia.org/w/index.php?curid=52989519.

Within this framework, individuals with similar functionally significant epigenotypes are expected to occupy similar individualized niches. However, several factors could obscure such a pattern. First, different epigenetic variations may converge into similar phenotypes, particularly for polygenic traits. Likewise, different genotypes may give rise to similar phenotypes by relying on distinct patterns of epigenetic variation, reflecting the interdependence between genetic and epigenetic factors. Second, multiple niche processes may act concurrently or sequentially when a single niche process cannot resolve a phenotype–environment mismatch, and each process may involve epigenetic variation at different genes. Third, niche choice and construction expose an individual to different environmental conditions, which, in turn, may alter natural selection to favor different **epigenetic variants** or induce novel epigenetic variation (see section "Epigenetics, individualization, and evolution").

Niche processes as drivers of epigenetic variation

Niche processes can themselves generate epigenetic variation. By selecting or modifying environments, individuals alter their exposure to external cues, such as temperature, diet, social interactions, or stressors, which can induce new epigenetic states [34–36]. Likewise, physiological or behavioral adjustments associated with niche conformance may feedback on internal hormonal or metabolic states that influence epigenetic regulation. Consequently, all three sources of epigenetic variation—genetically regulated, stochastic, and environmentally inducible—may be shaped by niche processes. Such feedbacks among behavior, internal conditions, the environment, and the epigenome may reinforce individualized niche dynamics and help explain population-level patterns of epigenetic diversity.

Epigenetic pathways allowing niche processes to act across generations

Niche processes can shape evolution either by directly modifying patterns of selection or through transgenerational effects arising from epigenetic variation. While genetically regulated epigenetic

variation is inherited alongside genetic variants, stochastic or inducible epigenetic variation must transiently mimic heritable genetic variation to have long-term consequences. Documented examples exist for at least three pathways through which epigenetic variation can be transferred inter- and/or transgenerationally, namely **germline epigenetic inheritance, experience-mediated inheritance,** and **ecological inheritance** [35].

Germline epigenetic inheritance, the process of epigenomes being directly inherited through meiotic pathways [51], such as methylation patterns in fish [52], can involve both stochastic and inducible epigenetic variation. However, the mechanisms of germline inheritance vary substantially across taxa. Sometimes, epigenetic variation is 'erased' during gametogenesis and embryonic development, as shown for mammal and bird species [53,54]. The extent of erasure can differ between paternal and maternal gametes, as shown in zebrafish *Danio rerio* [55]. In other species, such as the nematode *C. elegans* (Box 3), erasure does not occur, and altered gene expression is maintained for up to 14 generations [45].

Known as experience-mediated inheritance, the phenotypic effects of stochastic and inducible epigenetic variation in the parental generation can alter offspring epigenetic variation [35]. For example, the transfer of hormones via mammalian milk or altered parental care intensities can produce similar epigenetic patterns in offspring, as demonstrated in rodents [35,49].

Lastly, ecological inheritance occurs when parents choose or construct the offspring's environment [35]. This involves only epigenetic variation that is induced by the environment, which is passed down from parents to their offspring. In the absence of germline inheritance, offspring may initially be mismatched with the parental environment when environments change between generations, but environmental cues can trigger niche conformance in offspring, aligning their phenotypes to the ecologically inherited environment. For example, earthworms alter soil function through niche construction [56], which can subsequently influence their offspring's epigenome [57]. We speculate that ecological inheritance might also prime the offspring, once matured, to perform niche processes similar to those implemented by their parents, leading to further environmental changes and transmission of the constructed environment to consecutive generations.

Epigenetics, individualization, and evolution

Epigenetic variation can shape evolutionary trajectories in complex ways. Under certain environmental conditions, genetically regulated or environmentally induced epigenetic changes may buffer the effects of deleterious genetic variation, reducing fitness differences among genotypes and thereby promoting more drift-like evolutionary dynamics [58]. Conversely, genetic variants that modulate the inducibility of epigenetic states may themselves become targets of selection when such responsiveness confers an adaptive advantage [59]. This interplay highlights the dynamic feedback between genetic and epigenetic inheritance, where epigenetic mechanisms can either mask or amplify the effects of selection, depending on the stability and predictability of the environment.

Niche processes may explain individual differences in epigenetic variation. First, by altering the environment, niche choice and construction can trigger the emergence of inducible epigenetic variation. Second, niche processes may buffer selection by producing phenotypes with similar fitness, even among individuals with different genotypes, thereby maintaining population-level epigenetic variation. A single epigenetic variant can influence multiple manifestations of the same niche process (e.g., niche conformance across several traits) or affect different niche processes that together optimize the phenotype–environment match, thereby promoting the rapid coevolution of selected traits. However, more often, we expect traits to evolve independently

because different epigenetic variants usually underlie distinct niche processes. Under such circumstances, genetically regulated, stochastic, and inducible epigenetic variants should be favored under different conditions and have distinct evolutionary consequences.

In contrast to genetically regulated epigenetic variation, where natural selection acts on the underlying genotype, stochastic epigenetic variation is only rarely expected to influence phenotypes [24] or to be stably inherited across generations [22,60]. Such variation should be favored by natural selection when environmental conditions remain predictably stable over long time periods [61], because otherwise, the costs of plasticity would eliminate it [36]. In stable environments, **genetic assimilation** should eventually consolidate these changes, reducing the amount of stochastic epigenetic variation. As explained above, increased genetically regulated and stochastic epigenetic variation should also be linked to the evolution of niche choice and niche construction. In predictably stable environments, epigenetic variants that emerge from these mechanisms and promote successful niche choice should be favored by selection. Similarly, genetically regulated or stochastic epigenetic variants underlying niche construction should be selected for when the possibility of modifying the environment in a given way can be predicted. Consequently, niche choice and construction could promote the partitioning of individuals across different environments, leading to the evolution of **ecotypes** and ultimately to speciation. Consistent with this idea, epigenetic differences have been reported between different ecotypes [62] as well as between closely related species [62], although causal links remain to be established.

While genetic variation controlling epigenetic responsiveness carries information on past selection regimes, this is not true of inducible epigenetic variation, which is not directly affected by natural selection [26]. Instead, inducible variation can mediate intra- and transgenerational plasticity, enhancing environmental tolerance and reducing extinction risk under environmental change [63]. The evolution of plasticity, and hence increased adaptive inducible variation, should be favored in heterogeneous environments with different fitness optima and at least short-term predictability via reliable cues [38]. Plastic traits show low heritability; thus, we speculate that plasticity can evolve because genetic variants that promote the emergence of inducible epigenetic variation may be selected for in fluctuating environments, consistent with the Baldwin effect [64].

Moreover, because most epigenetic variation established in previous generations is removed during gametogenesis and embryonic development [53,54], the evolutionary consequences of stochastic and inducible variation will be largely indirect (see section "Epigenetic pathways allowing niche processes to act across generations"). By contrast, because genetic variation is stably inherited, it can produce the same epigenetic variation anew in every generation. Consequently, natural selection can act on genetically regulated epigenetic variation by favoring phenotypes with the greatest fitness.

Challenging environmental conditions can also increase epigenetic variation, a phenomenon known as **epigenetic buffering** [65] that is mostly mediated by stochastic variation [25]. This may then lead to a greater level of individualization and an increased probability of population persistence in fluctuating or novel environments, as greater population-wide epigenetic variation generates more among-individual phenotypic variation. Accordingly, higher epigenetic diversity is often found in individuals facing environmental challenges, such as in invasive species [66], those at the edge of the species' distribution during range expansion [67], and those in urban populations [68], although other confounding factors, such as changes in reproductive modes, might also contribute to epigenetic diversity.

Studying epigenetic variation contributing to individualized niches

Identifying adaptive epigenetic variation linked to niche processes is challenging, as most of this variation has no phenotypic effects [69], and any remaining effects may be confounded by population history or environmental variation. Experimental evolution paradigms [70] offer a practical way of quantifying epigenetic changes, their heritabilities, contributions to niche processes, and impacts on fitness.

Another challenge is determining whether epigenetic variation is genetically regulated, stochastic, or inducible. This can be achieved through multigenerational common-garden studies [23] or by comparing within- and among-population differences [71]. It is also important to repeatedly assess epigenetic states in the same individuals to differentiate between epigenetic variation that arises before and after niche processes have concluded. However, epigenetic states vary among cell types [72], genotypes [28], and sexes [73], while inducible somatic variation can also be influenced by season [74], age [75], and developmental status [50]. Controlling for these variance components requires well-designed experiments with sufficiently large sample sizes.

Genetic and epigenetic variation are interdependent, which is most obvious in genetically regulated epigenetic variation, but also because epigenetic variation can only emerge at certain genomic locations. Consequently, the role of epigenetic processes in individualization can best be assessed by examining genetic variation and establishing baselines for mutation rates and/or the mode(s) of inheritance of epigenetic variation. This can be achieved through experiments in which selection pressures are minimized and the long-term dynamics of epigenetic processes can be investigated [76]. Study systems with isogenic or inbred lines may be particularly suitable for this purpose. Cross-taxon comparisons are essential for assessing the generality of outcomes.

Given these challenges, the use of clonal species [77] (whose results may not be generalizable to natural populations) or even epigenetic manipulation [78] may be necessary to validate the functional roles of epigenetic variation in individualization. Furthermore, the diversity of epigenetic mechanisms (Box 2) makes multiomics integration essential. This will facilitate the simultaneous investigation of multiple layers of epigenetic regulation, revealing their interactions [79] and clarifying their collective contributions to phenotypic variation and the realization of individualized niches.

Concluding remarks

Niche realization can be mediated by changes in an individual's epigenome, a process we believe is best understood by focusing on the distinction between genetically regulated, stochastic, and inducible epigenetic variation. At the same time, niche realization can alter both individual environments and patterns of selection, thereby influencing the emergence and maintenance of epigenetic variation. Although nongenetically regulated epigenetic variation may not always be inherited directly through the germline, it may still exert transgenerational effects via experience-mediated or ecological inheritance, thereby impacting niche realization across generations. Even in the absence of direct transgenerational effects, as long as specific epigenetic variants affect niche realization, they can alter patterns of selection and affect long-term evolution. We also outline methodological challenges and provide future perspectives on how to link epigenetic variation to the processes leading to individualized niches (see Outstanding questions).

In summary, our understanding of ecoevolutionary processes will benefit from the concurrent analysis of genetic, epigenetic, and phenotypic variation at the individual level. This approach

Outstanding questions

What is the relative contribution of epigenetic versus genetic variation to the realization of individualized niches?

Which parts of genetically regulated, stochastic, and environmentally inducible epigenetic variation contribute to the realization of individualized niches, and what parts are adaptive and/or heritable?

What is the relative importance of genetically regulated, non-neutral stochastic, and environmentally inducible epigenetic variation for individualization?

Are epigenetic variants shared within manifestations of each niche process or among different niche processes, causing joint evolution or evolutionary constraints?

Are the epigenetic mechanism(s) driving niche processes ubiquitous across taxa, and can they be generalized?

Does epigenetic variation, which is stably passed down through the germline and underlies niche processes, represent an alternative evolutionary pathway, and to what extent is this independent of DNA sequence changes?

How does epigenetic variation change over evolutionary timeframes, and does the amount of variance that can be accounted for by individualization change as a result of these long-term dynamics?

promises to uncover the mechanisms driving the realization of individualized niches and, concurrently, to elucidate the origins, functions, and dynamics of epigenetic changes.

Acknowledgments

This research was funded by the German Research Foundation as part of the SFB TRR 212 (NC^3), Project numbers 316099922, 396774617, 396777092, 396780003, 396780709, 396780988, 471672756, 471673093. It was also funded by a Freigeist Fellowship from the VolkswagenStiftung, by a standard DFG grant (Project Number 454606304), by a Humboldt Research Fellowship for Postdocs and Experienced Researchers, and by the European Research Council (ERC) under the European Union's Horizon 2020 research and innovation program (Grant Agreement No. 947636). We are grateful to our colleagues of the SFB for stimulating discussions on the topic of individualized niches. We also thank Annett Boerner for helping in designing Figure 1.

Declaration of interests

No conflicting interests have been declared.

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