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Rapid adaptive responses to climate change in corals

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Pivotal to projecting the fate of coral reefs is the capacity of reef-building corals to acclimatize and adapt to climate change. Transgenerational plasticity may enable some marine organisms to acclimatize over several generations and it has been hypothesized that epigenetic processes and microbial associations might facilitate adaptive responses. However, current evidence is equivocal and understanding of the underlying processes is limited. Here, we discuss prospects for observing transgenerational plasticity in corals and the mechanisms that could enable adaptive plasticity in the coral holobiont, including the potential role of epigenetics and coral-associated microbes. Well-designed and strictly controlled experiments are needed to distinguish transgenerational plasticity from other forms of plasticity, and to elucidate the underlying mechanisms and their relative importance compared with genetic adaptation.

The unprecedented rate of environmental change that characterizes the Anthropocene[1] has raised concerns over whether the pace of organismal adaptation will be sufficient to mitigate projected detrimental effects on populations, communities and ecosystems[2]. The appearance and fixation of new adaptive genetic mutations generally requires many generations, suggesting that only organisms with short generation times will be able to adapt at rates matching the pace of environmental change. However, genetic adaptation can sometimes occur remarkably rapidly — within just a few generations — when standing genetic variation and recombination rates are high[3] (Box 1). Furthermore, it is increasingly recognized that acclimatization through phenotypic plasticity may buffer populations against rapid environmental change, allowing genetic adaptation to catch up over the longer term[4].

The fate of tropical coral reefs is of particular concern due to their high social, ecological and economic value, and their sensitivity to environmental change[5]. Hermatypic scleractinians (reef-building corals), the ecosystem engineers of coral reefs, live close to their upper thermal limits, and elevated summer temperatures can cause mass coral bleaching and mortality[6]. Some reef-building corals are also sensitive to the declining saturation state of carbonate ions that accompanies ocean acidification[7], and declining water quality associated with altered land use and precipitation regimes[8]. Reef-building corals provide shelter, food and habitat, and therefore loss of live coral and associated structural complexity leads to declines in the diversity and abundance of other reef organisms[9,10]. The future of coral reefs will therefore depend on the capacity of these foundation species to respond adaptively to rapid environmental change.

Recent experiments indicate that some coral and reef fish species can, at least to some extent, acclimatize to warming and acidifying oceans via developmental and/or transgenerational plasticity (TGP)[11,12] (Box 2). However, there are profound limitations to our current understanding of the underlying mechanisms of TGP and how these might interact with genetic adaptation[13]. While it has been suggested that epigenetic processes may be involved[14], there are divergent opinions on the strength of evidence for transgenerational inheritance via epigenetic marks, even in some well-characterized model organisms[13,15]. Moreover, exact mechanisms and the extent to which they have an effect are still unclear and under discussion[15]. Understanding multigenerational effects in corals is further complicated by the intimate relationships that they form with diverse suites of microorganisms that may contribute to phenotypic plasticity[16,17]

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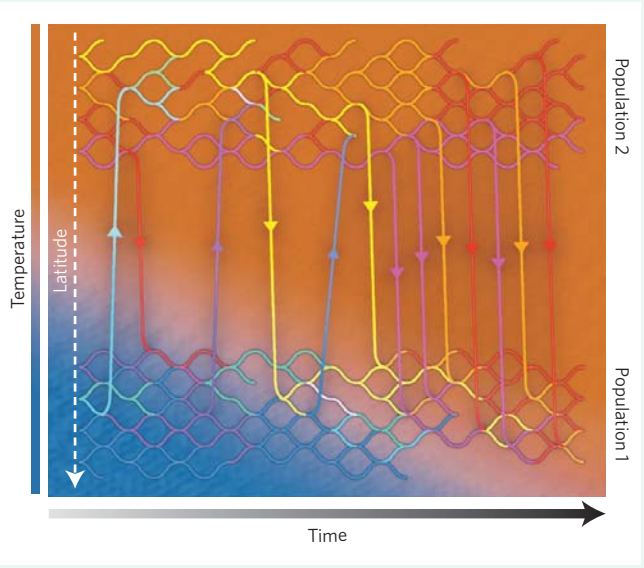
Box 1 | The pace of genetic adaptation.

A common misconception is that genetic adaptation occurs slowly and cannot possibly match the rate of ongoing climate change. Genetic adaptation is the change in allele frequencies in a population between generations, leading to a shift in mean trait values. This process does not require the appearance of new beneficial mutations (which potentially requires many generations); instead, it recombines and redistributes existing genetic variants, termed 'standing genetic variation'. In genetically diverse populations, such redistribution can happen very rapidly, potentially leading to positive selection fuelling adaptation[111]. Metapopulations inhabiting broad environmental gradients can collectively harbour extensive standing genetic variation, creating an additional opportunity for genetic adaptation via the spread of adaptive alleles among populations through migration ('genetic rescue'; see the figure below)[112]. A major unknown is the relative importance of genetic adaptation versus phenotypic plasticity in responding to rapid environmental change and how the two may interact.



Rapid genetic adaptation to global warming in a metapopulation, based on standing genetic variation. Two populations are each represented by a network of genetically diverse genotypes, recombining through time. Occasional migration events (vertical lines) tie the two networks together and provide a way to share adaptive alleles. Warmer genotype colour indicates higher heat tolerance. In this example, the warm-adapted low-latitude population 'rescues' the cool-adapted high-latitude population by supplying heat-tolerant alleles.

Box 2 | Ecological and mechanistic context of TGP.

TGP occurs when the phenotype of a new generation is influenced by the environment experienced by the previous generation(s). TGP is adaptive when the exposure of parents to a particular environment leads to improved performance of offspring in the same environment[20], with classic examples of adaptive TGP including morphological defences in animals[19] and the shortening of lifecycles in plants[55]. Parents can influence the phenotype of their offspring through a range of mechanisms, including the transmission of nutrients or other cytoplasmic factors, such as hormones and proteins, or, in some cases, through epigenetic processes, such as CpG methylation, histone modifications and variants, or non-coding RNAs. The transmission of epigenetic marks between generations (transgenerational epigenetic inheritance via the gametes) is of particular interest because it has the potential to explain many examples of transgenerational phenotypic effects that are not easily accounted for by inherited genetic variation[113].

Distinguishing TGP from developmental plasticity is challenging. A number of recent studies have shown that negative effects of projected future climate change on marine organisms are greatly reduced if both parents and their offspring experience the same altered environmental condition[11,12,114]. These studies show that the parental environment can affect the offspring phenotype and may be examples of TGP. However, in all of the examples cited, the developing eggs or embryos (for example, in the mother) also experienced the altered environmental conditions, therefore it is not possible to rule out that the observed improvement in offspring performance is induced during early zygotic development rather than being TGP *sensu stricto*. While distinguishing between these possibilities is not critical if we simply want to know whether performance improves when multiple generations experience the same novel environmental conditions, it is important in terms of establishing the mechanistic basis of the changes observed. Future studies that aim to understand the mechanistic basis of TGP in marine organisms, while logistically challenging, will need to employ more complex experimental designs and spanning at least two to three generations (see Fig. 1). Research so far has generally assumed a simplistic situation where each generation is considered to be completely discrete (Case A, Fig. 1), and consequently phenotypic differences in F2 offspring between treatments are considered to be TGP by F1 parents. However, for most species it is unknown when the primordial germ cells develop, and consequently, TGP cannot be conclusively distinguished until the F3 generation (Case B). Ideally, the timing of germ cell development, or any effect on the developing reproductive cells is known before commencing TGP experiments, enabling divisions between treatments to be completed at the correct time (Case C).

and by their propensity for asexual reproduction. While the long lifespans and extensively overlapping generations typical of scleractinian corals might be expected to restrict the pace of genetic adaptation, this effect may be offset by other characteristics, particularly their close associations with a diverse range of microbes, high standing genetic variation (Box 1), colonial organization and high fecundity[18].

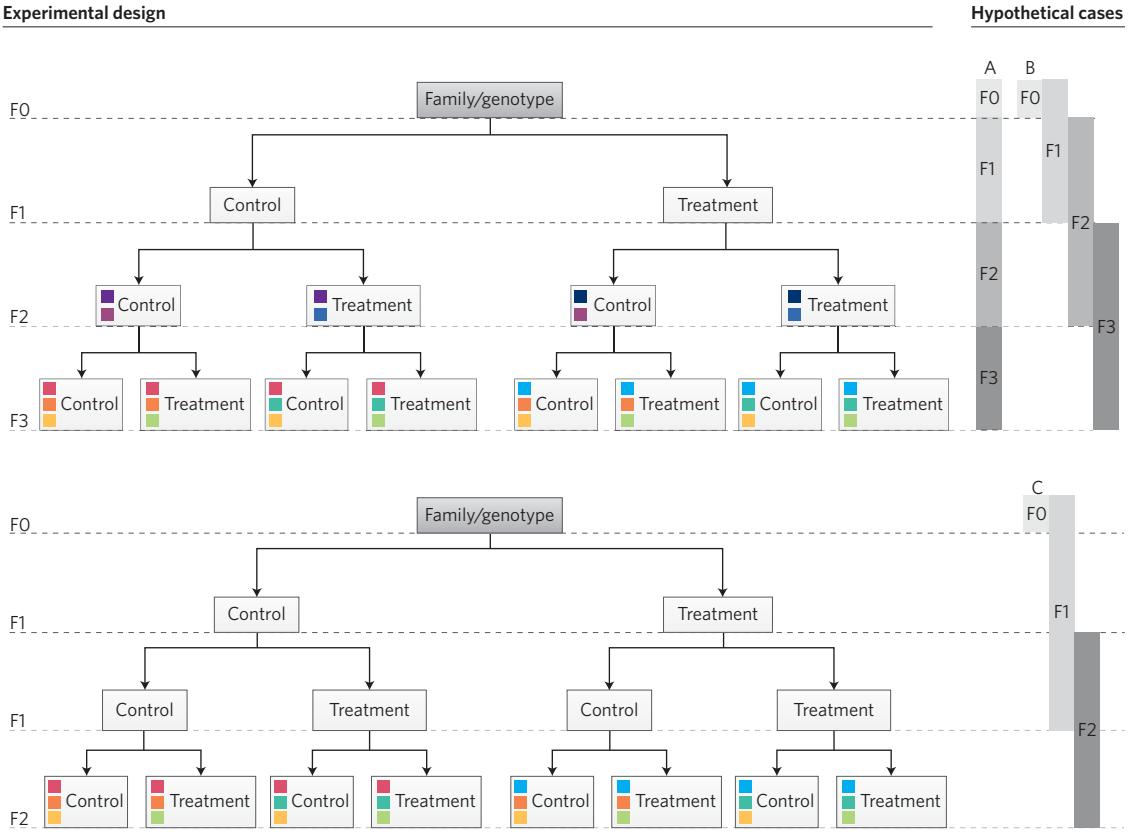
In this Perspective, we discuss mechanisms that could potentially enable plastic responses to climate change in reef corals. We provide a brief review of the available evidence (and the lack thereof) for the scope of transgenerational epigenetic inheritance to effect rapid phenotypic change in corals. We then predict the relative importance of TGP in various life-history traits, and strategies that are shared among, or unique to, foundation coral-reef species. Lastly, we discuss the potential of microbes to facilitate acclimatization in the coral holobiont.

Potential mechanisms for TGP

Phenotypic plasticity is a ubiquitous phenomenon that is increasingly gaining scientific attention as we focus on understanding the potential for organisms to respond to rapid changes in their environment. As global climate change is likely to occur on timescales that span multiple generations of corals (and many other multicellular organisms), attention has focussed on exploring the potential

for adaptive TGP (Box 2). While TGP has now been documented in a range of organisms at the phenotypic level[19–21], the underlying mechanisms are largely unknown.

Recent developments in omics technologies have enabled greater insight into the molecular pathways associated with plastic phenotypic responses and, in some cases, identified key genes whose altered expression may contribute to buffering against adverse environmental conditions within a generation[22,23] and across multiple generations[24,25]. Epigenetics, a term originally coined by Waddington in 1940, was intended to explain the phenomenon of cellular differentiation in multicellular organisms from a single genome[26]. More recently, the concept has evolved to include all mechanisms that potentially regulate gene expression, such as DNA methylation, histone modifications and variants, and noncoding and antisense RNAs. The discovery that some epigenetic marks are meiotically heritable (for example, the maternal DNA (CpG motif) methylation state of the *agouti* locus in mice[27,28]) led to an explosion of interest around epigenetic mechanisms driving transgenerational



Comparisons of performance reaction norms between highlighted groups

	A	B	C
versus	F1 TGP	F1 TGP and/or F2 development	—
versus	F2 developmental	F2 developmental	—
versus	F1 TGP	F1 TGP and/or F2 TGP	F1 TGP
versus	F2 TGP	F2 TGP and/or F3 developmental	F1 TGP and/or F2 developmental
versus	F3 developmental	F3 developmental	F2 developmental

Figure 1 | Identifying TGP in offspring depending on generational overlap in exposure. Three hypothetical cases of overlap between generations (right) highlight the difficulties of determining TGP from developmental plasticity in a common experimental design (left). Phenotypic differences observed in the experiment could be due to transgenerational and/or developmental plasticity (as shown in the bottom table) depending on the overlap of environmental exposure between generations (Cases A–C). Case A depicts a situation where environmental treatments affect only one generation at a time; this is often assumed to be the case in TGP experiments. Case B depicts a situation where primordial germ cells are present at birth and thus the current and subsequent generations are exposed to the environmental treatment at the same time. Case C depicts a situation where the timing of effect on the subsequent generation is known, and division between treatments can be completed at the appropriate time. In all cases, critical to distinguishing phenotypic change due to TGP, or what may be a mixture of TGP and developmental plasticity, is the division of siblings (sexual) or clones (asexual) between the treatments at the commencement of the experiments (F1), and full orthogonal crossing of treatment conditions in each generation (or appropriate generational split). Interactions between exposures of generations, that is, TGP resulting from exposure of the parents versus grandparents to environmental change, can also be determined in the highlighted cases (when reared to the F3 generation) due to the orthogonal example experimental design displayed.

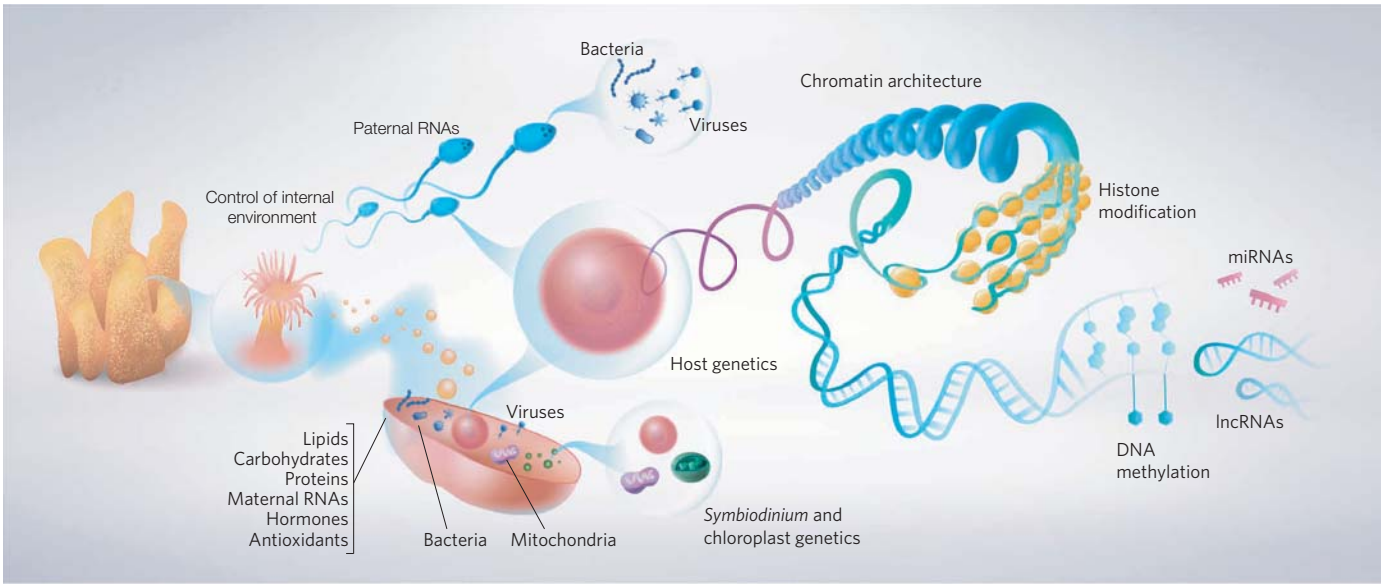


Figure 2 | Potential pathways that may enable TGP in corals include somatic, genetic and epigenetic factors of the coral gametes as well as their associated microbes transmitted vertically from one generation to the next. For details, see section 'Potential mechanisms for TGP'.

phenotypic plasticity across a wide range of organisms. While an increasing number of studies demonstrate association between epigenetic marks and overall phenotypes (including gene expression), causality remains to be established[29]. Moreover, the mechanisms involved seem to be highly variable across the tree of life, suggesting that there is no universal regulator of gene expression. For example, transgenerational inheritance linked to patterns of CpG methylation seems common in plants[20], but has been established in only a very limited number of cases in animals[28,30,31]. These examples mostly implicate atypical genomic regions, for example, retrotransposons that affect the transcription of neighbouring genes[13,30]. Furthermore, the low levels of correlation found between the transcriptome and the methylome of several multicellular organisms[32,33], combined with the lack of a CpG methylation system in some of the most widely studied model animals, including the fruit fly *Drosophila* and the roundworm *Caenorhabditis*[34,35], weakens the case for its significance as a universal regulator of gene expression[15,36], and hence a universal mediator of TGP. In corals, DNA methylation levels correlate strongly with gene function; broadly and uniformly expressed 'housekeeping' genes are strongly methylated, whereas genes responsible for inducible or cell-specific functions are weakly methylated[37,38] (Fig. 2). Nevertheless, it remains to be seen whether this divergent methylation causes or is caused by differences in gene expression, whether it responds to environmental cues[14], and whether it can be passed across generations. In summary, we do not dismiss a potential role for epigenetic inheritance in TGP of corals, but evidence is currently largely lacking, and mechanisms other than DNA methylation need increased attention.

Non-coding and antisense RNAs from the maternal cytoplasm can potentially affect zygotic transcriptional activity and provide short-term epigenetic memory that fades out with cell divisions[39] (Fig. 2). However, for some genes, transcriptional states established early in development can be maintained through mitotic divisions by epigenetic mechanisms[40]. Furthermore, epigenetic cross-talk[41,42], for example a positive feedback loop between chromatin and small RNAs, can promote long-term epigenetic memory in some organisms[40], but again this field remains highly understudied in corals.

Histone tail modifications and non-canonical histones modulate chromatin structure, and hence gene expression[43,44] (Fig. 2). In the cases where TGP is associated with histone modifications over multiple generations, it is likely that multiple epigenetic mechanisms affect target genomic regions. For example, temperature-induced changes in gene expression in *Caenorhabditis* last for over 14 generations, and are strongly associated with a histone modification that alters the chromatin structure and triggers a cascade that affects RNA-mediated gene silencing[31]. In corals, histone modifications are virtually unstudied, representing a major research gap that hinders our understanding of molecular mechanisms of TGP.

In addition to epigenetic mechanisms, parents can affect their offspring via a range of factors transmitted to the embryo through paternal and maternal germ cells[45] (Fig. 2). For example, nutritional factors passed through the oocyte's cytoplasm, such as lipids and carbohydrates, may directly influence the metabolic capacity of the early zygote and larva. Maternal provisioning of proteins can equip the oocyte and zygote with inaugural machinery for important functions before zygotic translation begins. Furthermore, the pool of maternal mRNA provides templates for early protein synthesis in the embryo, before zygotic transcription begins. In a range of plant species, hormones have been shown to play major roles in transgenerational environmental effects on offspring growth and development[20]. Transmission of mitochondria represents another potentially important pathway for maternal effects, especially in eukaryotic cells where cross-talk is assumed between the nuclear genome and mitochondria, with the organelle essentially acting as an interface between the environment and the epigenome[46] through metabolites[47–49].

Genetic information inherited from parents can contain copy number variations, repeat expansions or contractions, and the products of recombination events. Finally, gametes, embryos or larvae might undergo natural selection for alleles that provide advantage in the parental environment, particularly in highly fecund species. Such selection within full-sib larval families has been demonstrated experimentally in corals[50]. The resulting shift in the distribution of offspring phenotypes could be misinterpreted as TGP but is actually due purely to genetic adaptation.

These examples illustrate the diversity of mechanisms by which the parental environment could influence offspring phenotype, and warrant consideration in explaining TGP. Understanding the causal molecular mechanisms underlying adaptive phenotypes will be a major challenge, even in well-studied model organisms, but is needed to better predict the potential of these processes to enable organismal acclimatization to environmental changes.

In the next two sections, we first evaluate some of the common and unique life-history traits of corals that could enhance or hinder TGP. Secondly, given that oocytes could theoretically act as transgenerational vectors for the parental microbiome, we discuss the potential contributions that microbes, including bacteria, viruses and symbiotic protists, such as *Symbiodinium spp.*, could make to the phenotype and fitness of the coral host, as well as to the capacity for rapid adaptive responses in the holobiont.

Predictors of TGP in corals

Evidence of phenotypic plasticity across a range of coral life-history stages and traits is mounting, highlighting significant capacity for scleractinian corals to respond to altered environmental conditions. Within a lifetime, some corals can modulate their gross colony growth form to optimize light environments for photosynthesizing endosymbionts[51], physiologically acclimatize to elevated temperatures[22], and show signs of acclimatization under pH stress[14,23]. These examples suggest that corals may retain phenotypic plasticity in their adult life stage, which can itself be a trait affected by the corals' environment[52]. In tandem with high levels of intragenerational plasticity, multigenerational exposure of corals to altered environmental conditions can equip their offspring with enhanced stress tolerance[12]. In the brooding coral *Pocillopora damicornis*, the parental generation suffered metabolic depression under elevated temperature and CO_2 conditions, but the F1 larval offspring showed partial metabolic restoration to elevated conditions compared with offspring from un-exposed parents[12]. It is unclear, however, whether these beneficial parental effects last throughout the lifespan of the F1 generation and beyond. Furthermore, as explained in Box 2, it is difficult to disentangle TGP from developmental plasticity in this type of experiment, because the brooding larvae experienced the same environments as the parents. Regardless of the underlying mechanisms, these results highlight the importance of considering the ecological implications of multigenerational exposure to projected future environmental conditions when predicting the response of reef corals to climate change.

Corals vary enormously in their life-history traits, some of which may promote, and others impede, TGP. For example, adaptive TGP might be expected when the parental environment is a reliable predictor of environmental conditions that their offspring will experience[53,54]. Because short-range offspring dispersal typically enhances environmental predictability among generations[55], the benefits of TGP are expected to be inversely proportional to the dispersal capacity of the organism. The three main reproductive strategies that characterize coral-reef species — broadcast or pelagic spawning, benthic or demersal spawning, and brooding — represent a spectrum of dispersal potential, and hence differences between parental and offspring environmental conditions. Broadcast spawning, the most common mode of sexual reproduction in tropical reef corals[56], potentially provides greater offspring dispersal compared to demersal spawning; while brooding represents the least dispersive reproductive mode[57]. The high offspring-dispersal potential of broadcast spawners suggests that, in these cases, there may be limited correlation between the environmental conditions experienced by parents and offspring. Thus we predict TGP is least likely to be observed in broadcast spawners, as it should provide little selective advantage. Instead, broadcast spawners are predicted to produce offspring with a high capacity for developmental plasticity or offspring with a wide range of phenotypes (bet-hedging)[58,59]. TGP is more likely to be adaptive in brooding corals because the offspring are more likely to settle in a habitat that is similar to that of the parents. However, the relative importance of TGP across coral-reef species can only be understood via testing a range of species with robust experimental designs (see Fig. 1).

Longevity of some corals means that a genotype selected at the recruitment stage for an environment may be mismatched with changing environmental conditions as the sessile colony ages, so the selective advantages of TGP are likely to correlate with longevity. Modular organisms, such as scleractinians, octocorals, bryozoans and crustose coralline algae often not only have long lifespans but also reproduce asexually[60,61], which may result in exceptional lifespans of the genotype compared to other organisms[60,62], a feat only possible via substantial environmental tolerance or phenotypic plasticity[63]. Importantly, since such old colonies tend to be large and therefore highly fecund[64], they can potentially hinder genetic adaptation of the population by swamping the gamete pool with genotypes that are no longer a good match to the local environment. This can substantially reduce the rate of genetic adaptation in these organisms and may elevate the role of within-generation plasticity and TGP in helping the next cohort of recruits survive.

In long-lived corals, somatic mutations may accrue over the lifetime of modular colonies[18], highlighting another mechanism that could potentially aid phenotypic responses to environmental changes within the lifespan of the colony. Evolution through somatic mutations, as in the case of transgenerational epigenetic inheritance, is more likely to have a role in organisms that lack distinct segregation of the somatic and germ lines, such as fungi, plants and corals (but see ref. 65), or produce larvae asexually. Whether or not such mutations can be passed on to subsequent generations and hence contribute to genetic adaptation (Box 1) in corals remains controversial[65,66].

In summary, we predict that TGP is unlikely to be the main driver of plasticity in most coral species since the vast majority are broadcast spawners[56], for which the parental environment is a relatively poor predictor of the offspring environment. On the other hand, extended longevity in some corals could result in a mismatch between the genotype and present-day environmental conditions, and we predict that such species have evolved substantial capacity for plasticity in the offspring. Brooding corals are expected to benefit from both within-generation plasticity and TGP, because the developing embryo experiences the same environment as both its mother colony and subsequent juvenile and adult stages; and because many brooding corals have relatively short lifespans.

Potential involvement of microbes in coral acclimatization

Corals live in close association with a range of eukaryotic and prokaryotic microorganisms that may adapt or acclimatize faster than their metazoan host, potentially providing additional adaptive capacity to the holobiont. The coral holobiont[67] is an inter-domain community of complex and dynamic associations involving the photosynthetic alveolate *Symbiodinium* and a range of bacteria, fungi and viruses, some of which have been central to the success of the Scleractinia as the dominant contemporary tropical reef-builder[68] (Fig. 3). Although components of the holobiont have separate evolutionary trajectories[69], the intimate nature of some coral–microbial associations implies that their interactions may contribute to the overall fitness of the holobiont[68]. In comparison with the coral host, the orders of magnitude greater diversity, shorter generation times, and remarkable metabolic range of the coral microbiome suggest that some microbes could make contributions to adaptive responses of the holobiont. Here we consider the most prominent members of the coral microbiome and discuss how their evolution might affect coral performance under climate change. Such contributions are particularly relevant in the context of the long generation times of many corals and the rapid pace of current environmental change.

Symbiodinium. The well-studied coral–*Symbiodinium* association best illustrates the potential of microbial symbionts to effect rapid phenotypic change at the level of the coral holobiont, either through their own evolution[70] or changes in community composition (Fig. 3). The dinoflagellate genus *Symbiodinium* contains enormous genetic and functional diversity[71], and communities associated with corals vary among species, environments and host

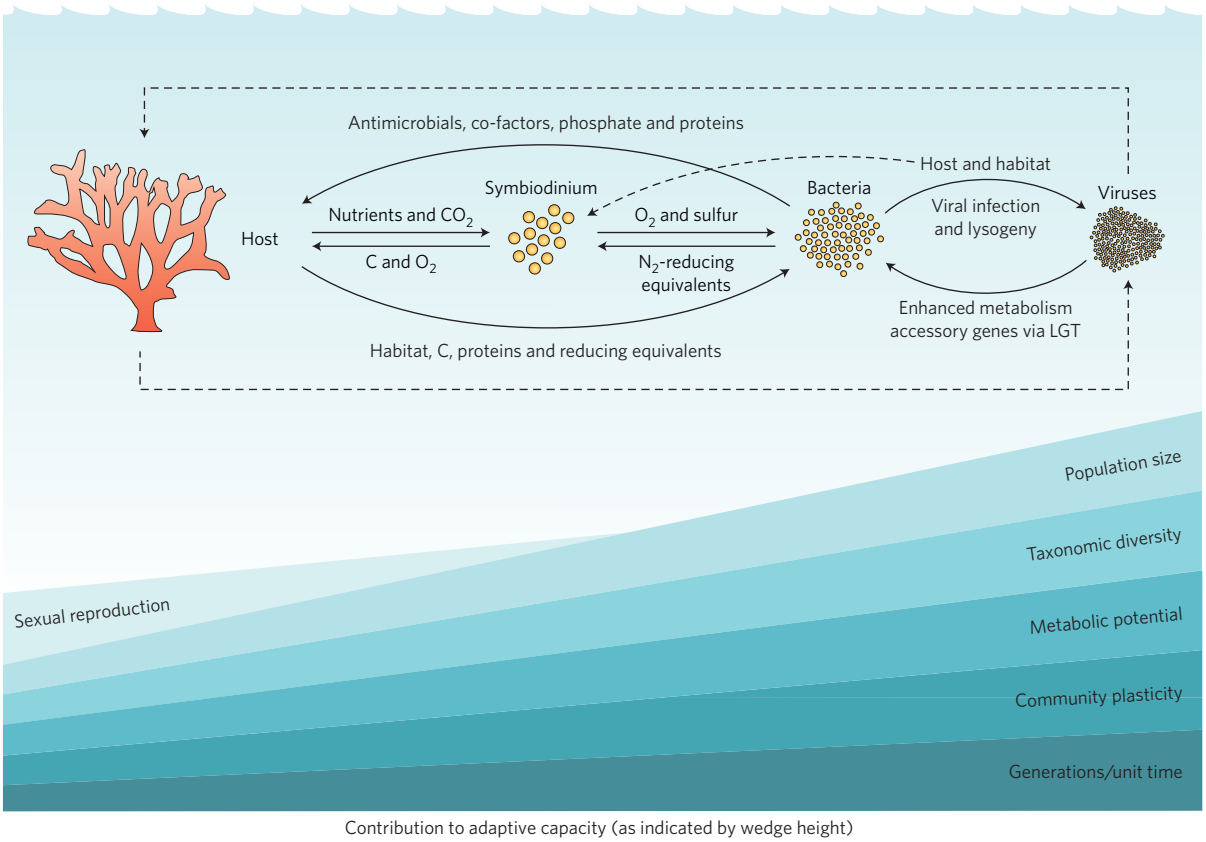


Figure 3 | Illustration showing members of the coral holobiont and their potential for contribution to adaptive holobiont responses. Member interactions are indicated with arrows (known interactions in solid lines, largely unknown interactions in dashed lines). Potential adaptive capacity increases in members of the holobiont, indicated by wedge height, reflected in population size, taxonomic diversity, metabolic potential, community plasticity, shortening intergenerational times, and potential for sexual reproduction.

microhabitats[72]. The short generation time of *Symbiodinium* means that its rate of mutation is much faster than for the coral host[18], and this, combined with its large within-host population sizes, potentially facilitates rapid responses to altered thermal environments, either through selection of existing genetic variants or through the evolution of novel adaptations[73,74]. Alternatively, the composition of host-associated *Symbiodinium* communities may vary temporally in response to environmental conditions or at different host life-history stages[75], either through shuffling of existing symbionts[76] or through acquisition of new *Symbiodinium* types from the environment (that is, switching)[16]. In particular, high genetic and phenotypic diversity among *Symbiodinium* taxa provides scope for some coral species to vary the composition of associated *Symbiodinium* communities, balancing photosynthetic activity (and hence growth) with stress tolerance, a type of acclimatory mechanism for responding to environmental extremes[76,77,78]. If associations enhance host health, they would also be likely to enhance the size and maternal provisioning of eggs and larvae, optimally positioning offspring within the natal environment through maternal effects[79]. Vertical transmission of *Symbiodinium* from maternal parent to gametes or brooded larvae by corals whose larvae typically settle in the parental habitat[59,80] could increase the likelihood that juvenile corals establish a symbiont community suited to ambient environmental conditions. Conversely, the acquisition of symbiotic communities from the environment (horizontally) in the case of broadcast spawning corals, whose larvae typically disperse more widely[79], may represent a strategy to ensure that juveniles settling under a range of environmental conditions acquire *Symbiodinium* types that are locally adapted (but see ref. 75). The generally greater diversity of *Symbiodinium* communities in early life-history stages compared to in adults[79] could be viewed as a bet-hedging strategy, providing juvenile corals with the opportunity to fine-tune endosymbiotic communities to suit ambient conditions. Finally, the retention of low-abundance background *Symbiodinium* types in adult stages of some corals[16,81] may provide further adaptive capacity to the holobiont (but see 82), facilitating future shuffling of dominant *Symbiodinium* types in response to changing environmental conditions[76,83].

Bacteria. Host-associated bacterial communities could also contribute to the adaptive capacity of their coral hosts, given the enormous breadth of their metabolic capabilities and of mechanisms that contribute to their rapid evolution[84]. Roles in immunity, nitrogen fixation, nutrient cycling, osmoregulation and oxidative stress responses have been suggested for bacteria associated with different microhabitats within the coral host[68]. The potential significance of specific bacterial groups is suggested by their vertical transmission[80] and common presence within the tissues of a wide range of corals[85,86]. In particular, whereas transient, highly variable communities are typically associated with external coral mucus layers, low and relatively stable numbers of 'core' types are more generally associated with host cells[85]. Bacterial community changes and resulting shifts in the holobiont metabolic network may provide further scope for maintaining holobiont functions in the face of environmental change. For example, transplantation of corals to a warmer environment resulted in shifts in the associated bacterial community that correlated with increased holobiont thermotolerance[87]. Additionally, higher bacterial diversity in deep compared to shallow water corals[88,89] suggests that some deep habitat-specific microbes may be involved in nutrient cycling specific to the low-irradiance environments. Both genetic and epigenetic processes contribute

to high phenotypic plasticity and rapid evolution in bacteria[90]. In addition, bacterial pathogens and mutualists are known to induce alterations in host epigenomes, leading to potentially long-lasting imprinting effects that provide a form of plasticity to their hosts[91]. Importantly, although all these examples illustrate how bacteria could, in principle, contribute to plastic responses of the holobiont and generally improve its function, direct experimental evidence of this is lacking, highlighting this area as a research priority[17].

Viruses and other microbiome components. The potential of other components of the holobiont to contribute to the adaptive capacity of corals is unknown. Although viral infections generally have negative consequences for the fitness of their hosts, there are examples from other symbiotic systems of viral infections enacting non-mutational alterations to the host that buffer environmental effects[92]. In addition, viruses of coral-associated eukaryotes and bacteria (bacteriophages) potentially contribute metabolic and functional diversity to the holobiont via several mechanisms. First, viral infection of animal hosts can prevent the invasion of foreign bacteria via signalling and immune system modulation[93]. Second, direct bacteriophage infection and lysis may regulate the abundance of specific bacteria within the holobiont, fulfilling an immunity-like function[94]. Third, phages may be agents of lateral gene transfer between microbial members of the holobiont[95]. Also, phage-induced and virus-induced mortality of bacterial and host cells may contribute to nutrient remineralization within the system, altering holobiont physiology and microbial ecology (the 'revolving door' hypothesis)[96]. Another mechanism by which viruses could influence coral-associated bacterial communities is through genetic rearrangement. For example, shuffling of bacterial genes may result in wider metabolic potential, with coincident beneficial consequences for the coral host, for example, a broader range of products produced by dimethylsulfoniopropionate (DMSP)-metabolizing bacteria might enhance bacteria-mediated production of sulfur-based antimicrobials[97]. Despite such possible beneficial roles, however, viruses more typically have negative effects on host fitness and, in the case of corals, have been implicated in bleaching[98,99] and disease[100].

In summary, the short generation times, large population sizes and high turnover of microbes, combined with their prodigious diversity, provide a range of potential mechanisms to enable the coral holobiont to respond to environmental change on ecologically relevant time-scales. Thus the emergent property of adaptive capacity of the holobiont could simply reflect 'selfish' evolution on the part of the symbiont. However, not all 'symbionts' are beneficial, for example, some *Symbiodinium* types are almost certainly opportunists that provide little or no benefit to their coral hosts[82,101]; a number of bacteria are pathogenic, causing a variety of diseases in corals[102]; and coral-associated bacteria may become pathogenic through the acquisition of prophages[103]. It is also conceivable that proviruses associated with bacteria or *Symbiodinium* could cause host-cell lysis upon emergence from the lysogenic state triggered by environmental stress. Thus, although evidence is accumulating that some host-associated microbes might facilitate adaptive responses in corals, the fitness consequences of climate-change-induced evolution of the coral microbiome are unclear. There is also uncertainty around the extent to which increased stress tolerance might involve physiological trade-offs that compromise host health and fitness[104], and whether selection occurs at the level of individuals or the holobiont.

Summary and future directions

The processes and pathways that could potentially facilitate rapid adaptive responses in reef-building corals are diverse, but there is a great deal of uncertainty around what contributions they will make to climate-change adaptation. Beneficial effects of parental exposure to offspring phenotype have been demonstrated in reef fishes and initial evidence has been presented for corals, however the extent to which TGP occurs in reef organisms can only be elucidated via experiments that tease apart developmental plasticity from TGP (Box 2 and Fig. 1). Understanding the relative contributions of parental provisioning, genetic and epigenetic mechanisms and changes in the microbiome to adaptive responses is paramount for predicting the fate of coral reefs as environmental conditions change. The revolution in omics approaches provides unparalleled opportunities for exploring the roles of the different components in coral adaptive responses if coupled with appropriate experimental design.

While reef-building corals present many challenges for genetic or epigenetic analyses, understanding the adaptive capacity of these critically important organisms requires the application of such molecular approaches within a rigorous experimental framework. Coral research can benefit enormously from advances made on the more tractable 'model' animals and better integration with the mainstream molecular genetics community. Recent technological advances allow transgenesis, gene knockdown, and a range of other methods to be applied to the sea anemone *Nematostella,* a 'near' relative of corals. The symbiotic sea anemone, *Exaiptasia*, holds similar promise as an experimental system of particular relevance to coral biology. However, empirical studies on classical model organisms cannot completely replace those on corals, because many cellular and molecular processes show substantial taxonomic variability. For example, CpG methylation appears to have quite different roles in vertebrates compared with insects, and the methylation patterns implied in corals differ from expectations based on either of these[105].

The potential for adaptive responses of the coral holobiont via its microbial partners is perhaps the most distinct, but also the most controversial, aspect of coral acclimatization. Rapid responses in the coral-associated microbiome do not need to rely on mutation, but may arise from changes in the relative abundance (or lifestyles, for example, pathogenic switch) of associated microorganisms, acquisition of novel microbes (with novel functions) from the environment, or horizontal gene transfer among microbes[106]. Importantly, most of these processes have not been tested or unequivocally proven in the coral holobiont system, highlighting an important research priority[87]. Furthermore, while changes in the genetic and community composition of coral-associated microbes may be fast, their evolution (including that of *Symbiodinium spp.*) is inherently selfish. The available (admittedly limited) evidence suggests that microbes may not coevolve with their coral hosts, and thus adaptation of coral-associated microbes may lead to host-switching,

Box 3 | Future research directions.

1. Demonstrate TGP in corals and other reef organisms via well-designed, strictly controlled experiments (for example, see Fig. 1).
2. Test causality between epigenetic mechanisms and phenotypes.
3. Demonstrate heritability of epigenetic marks in corals.
4. Understand the relative contributions of parental provisioning, genetic and epigenetic mechanisms, and changes in the microbiome to adaptive responses in corals.
5. Further develop model organisms closely related to scleractinian corals, such as the sea anemones *Nematostella* and *Exaiptasia*, on which advanced techniques, such as gene-knockdown and transgenesis are possible.
6. Understand flexibility of coral–microbial associations, including the control of microbial communities by the host and the microbes.
7. Improve models of the interaction of TGP and genetic adaptation.
8. Determine the pace of genetic adaptation in members of the coral holobiont.

non-symbiotic (that is, free-living) or even parasitic (pathogenic) strains, rather than the provision of benefits to their coral host. The likelihood of these alternative pathways will depend on the specificity and strength of coral–microbe associations.

Throughout this paper we have largely discussed TGP in relation to its potential to influence offspring phenotype in an adaptive capacity. However, TGP can also be maladaptive[107,108]. This increases the need to understand TGP in response to climate change for conservation and management, since it could potentially constrain evolutionary processes[109] and hinder future species persistence. Correlated effects also need to be explored, as the individual phenotype is comprised of a range of traits that are unlikely to be equally affected by the environment or exhibit the same capacity for plasticity. Different life stages may be oppositely affected[110]. This is further amplified in the coral holobiont where all components may not be plastically and/or adaptively shifting in the same direction or over the same timescales.

Given the enormous momentum in the climate system, the fate of coral reefs in the Anthropocene will largely depend on the rate at which reef-building corals can adapt or acclimatize to environmental change. There is an urgent need to fill important research gaps around TGP in corals (Box 3) to be able to inform conservation efforts and policymaking. This includes research into the cellular and molecular mechanisms, the temporal dynamics (for example, time frame for adaptive response), the strength and speed of host versus microbial plasticity, and the interaction between adaptive plasticity and evolution.

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References

1. IPCC *Climate Change 2014: Synthesis Report* (eds Core Writing Team, Pachauri, R. K. & Meyer L. A.) (IPCC, 2015).
2. Bell, G. Evolutionary rescue and the limits of adaptation. *Philos. Trans. R. Soc. B* **368,** 20120080 (2013).
3. Barrick, J. E. & Lenski, R. E. Genome dynamics during experimental evolution. *Nat. Rev. Genet.* **14,** 827–839 (2013).
4. Munday, P. L., Warner, R. R., Monro, K., Pandolfi, J. M. & Marshall, D. J. Predicting evolutionary responses to climate change in the sea. *Ecol. Lett.* **16,** 1488–1500 (2013).
5. Pandolfi, J. M., Connolly, S. R., Marshall, D. J. & Cohen, A. L. Projecting coral reef futures under global warming and ocean acidification. *Science* **333,** 418–422 (2011).
6. Hughes, T. P. *et al.* Global warming and recurrent mass bleaching of corals. *Nature* **543,** 373–377 (2017).
7. Albright, R. *et al.* Reversal of ocean acidification enhances net coral reef calcification. *Nature* **531,** 362–365 (2016).
8. Fabricius, K. E. Effects of terrestrial runoff on the ecology of corals and coral reefs: review and synthesis. *Mar. Pollut. Bull.* **50,** 125–146 (2005).
9. Graham, N. A. J. & Nash, K. L. The importance of structural complexity in coral reef ecosystems. *Coral Reefs* **32,** 315–326 (2013).
10. Fabricius, K. E., De'ath, G., Noonan, S. & Uthicke, S. Ecological effects of ocean acidification and habitat complexity on reef-associated macroinvertebrate communities. *Proc. R. Soc. B* **281,** 20132479 (2014).
11. Donelson, J. M., Munday, P. L., McCormick, M. I. & Pitcher, C. R. Rapid transgenerational acclimation of a tropical reef fish to climate change. *Nat. Clim. Change* **2,** 30–32 (2012).
Seminal study demonstrating adaptive transgenerational plasticity to climate change in a coral-reef fish.
12. Putnam, H. M. & Gates, R. D. Preconditioning in the reef-building coral Pocillopora damicornis and the potential for trans-generational acclimatization in coral larvae under future climate change conditions. *J. Exp. Biol.* **218,** 2365–2372 (2015).
13. Daxinger, L. & Whitelaw, E. Transgenerational epigenetic inheritance: more questions than answers. *Genome Res.* **20,** 1623–1628 (2010).
Critical review of evidence for transgenerational epigenetic inheritance.
14. Putnam, H. M., Davidson, J. M. & Gates, R. D. Ocean acidification influences host DNA methylation and phenotypic plasticity in environmentally susceptible corals. *Evol. Appl.* **9,** 1165–1178 (2016).
The only study to date that links environmental variation to epigenetic changes in corals.
15. Ptashne, M. Epigenetics: core misconcept. *Proc. Natl Acad. Sci. USA* **110,** 7101–7103 (2013).
16. Boulotte, N. M. *et al.* Exploring the Symbiodinium rare biosphere provides evidence for symbiont switching in reef-building corals. *ISME J.* **10,** 2693–2701 (2016).
17. Webster, N. S. & Reusch, T. B. H. Microbial contributions to the persistence of coral reefs. *ISME J.* http://dx.doi.org/10.1038/ismej.2017.66 (2017).
18. van Oppen, M. J. H., Souter, P., Howells, E. J., Heyward, A. & Berkelmans, R. Novel genetic diversity through somatic mutations: fuel for adaptation of reef corals? *Diversity* **3,** 405–423 (2011).
19. Agrawal, A. A., Laforsch, C. & Tollrian, R. Transgenerational induction of defences in animals and plants. *Nature* **401,** 60–63 (1999).
20. Herman, J. J. & Sultan, S. E. Adaptive transgenerational plasticity in plants: case studies, mechanisms, and implications for natural populations. *Front. Plant Sci.* **2,** 1–10 (2011).
21. Salinas, S., Brown, S. C., Mangel, M. & Munch, S. B. Non-genetic inheritance and changing environments. *Non-Genet. Inherit.* https://doi.org/10.2478/ngi-2013-0005 (2013).
22. Palumbi, S. R., Barshis, D. J., Traylor-Knowles, N. & Bay, R. A. Mechanisms of reef coral resistance to future climate change. *Science* **344,** 895–898 (2014).
Demonstrates the link between environmental change and gene expression levels, as well as rapid acclimatization in corals.
23. Moya, A. *et al.* Rapid acclimation of juvenile corals to CO_2-mediated acidification by upregulation of heat shock protein and Bcl-2 genes. *Mol. Ecol.* **24,** 438–452 (2015).
24. Veilleux, H. D. *et al.* Molecular processes of transgenerational acclimation to a warming ocean. *Nat. Clim. Change* **5,** 1074–1078 (2015).
25. Goncalves, P. *et al.* Rapid transcriptional acclimation following transgenerational exposure of oysters to ocean acidification. *Mol. Ecol.* **25,** 4836–4849 (2016).
26. Waddington, C. H. *Organisers and Genes* (Cambridge Univ. Press, 1940).
27. Wolff, G. L., Kodell, R. L., Moore, S. R. & Cooney, C. A. Maternal epigenetics and methyl supplements affect agouti gene expression in Avy/a mice. *FASEB J.* **12,** 949–957 (1998).
28. Morgan, H. D., Sutherland, H. G. E., Martin, D. I. K. & Whitelaw, E. Epigenetic inheritance at the agouti locus in the mouse. *Nat. Genet.* **23,** 314–318 (1999).
29. Metzger, D. C. H. & Schulte, P. M. Epigenomics in marine fishes. *Mar. Genomics* **30,** 43–54 (2016).
30. Rakyan, V. K. *et al.* Transgenerational inheritance of epigenetic states at the murine AxinFu allele occurs after maternal and paternal transmission. *Proc. Natl Acad. Sci. USA* **100,** 2538–2543 (2003).
31. Klosin, A., Casas, E., Hidalgo-Carcedo, C., Vavouri, T. & Lehner, B. Transgenerational transmission of environmental information in C. elegans. *Science* **356,** 320–323 (2017).
32. Libbrecht, R., Oxley, P. R., Keller, L. & Kronauer, D. J. C. Robust DNA methylation in the clonal raider ant brain. *Curr. Biol.* **26,** 391–395 (2016).
33. Meng, D. *et al.* Limited contribution of DNA methylation variation to expression regulation in Arabidopsis thaliana. *PLOS Genet.* **12,** e1006141 (2016).
34. Lyko, F., Ramsahoye, B. H. & Jaenisch, R. Development: DNA methylation in Drosophila melanogaster. *Nature* **408,** 538–540 (2000).
35. Suzuki, M. M. & Bird, A. DNA methylation landscapes: provocative insights from epigenomics. *Nat. Rev. Genet.* **9,** 465–476 (2008).
36. Bestor, T. H., Edwards, J. R. & Boulard, M. Notes on the role of dynamic DNA methylation in mammalian development. *Proc. Natl Acad. Sci. USA* **112,** 6796–6799 (2015).
37. Dimond, J. L. & Roberts, S. B. Germline DNA methylation in reef corals: patterns and potential roles in response to environmental change. *Mol. Ecol.* **25,** 1895–1904 (2016).
38. Dixon, G. B., Bay, L. K. & Matz, M. V. Evolutionary consequences of DNA methylation in a basal metazoan. *Mol. Biol. Evol.* **33,** 2285–2293 (2016).
39. Klosin, A. & Lehner, B. Mechanisms, timescales and principles of trans-generational epigenetic inheritance in animals. *Curr. Opin. Genet. Dev.* **36,** 41–49 (2016).
40. Holoch, D. & Moazed, D. RNA-mediated epigenetic regulation of gene expression. *Nat. Rev. Genet.* **16,** 71–84 (2015).
41. Cedar, H. & Bergman, Y. Linking DNA methylation and histone modification: patterns and paradigms. *Nat. Rev. Genet.* **10,** 295–304 (2009).
42. Rey, O., Danchin, E., Mirouze, M., Loot, C. & Blanchet, S. Adaptation to global change: a transposable element–epigenetics perspective. *Trends Ecol. Evol.* **31,** 514–526 (2016).
43. Jenuwein, T. & Allis, C. D. Translating the histone code. *Science* **293,** 1074–1080 (2001).
44. Karlić, R., Chung, H.-R., Lasserre, J., Vlahoviček, K. & Vingron, M. Histone modification levels are predictive for gene expression. *Proc. Natl Acad. Sci. USA* **107,** 2926–2931 (2010).
45. Hamdoun, A. & Epel, D. Embryo stability and vulnerability in an always changing world. *Proc. Natl Acad. Sci. USA* **104,** 1745–1750 (2007).

46. Wallace, D. C. & Fan, W. Energetics, epigenetics, mitochondrial genetics. *Mitochondrion* **10,** 12–31 (2010).
47. Marden, J. H. Nature's inordinate fondness for metabolic enzymes: why metabolic enzyme loci are so frequently targets of selection. *Mol. Ecol.* **22,** 5743–5764 (2013).
48. Shaughnessy, D. T. *et al.* Mitochondria, energetics, epigenetics, and cellular responses to stress. *Environ. Health Perspect.* **122,** 1271 (2014).
49. Gibbin, E. M. *et al.* Can multi-generational exposure to ocean warming and acidification lead to the adaptation of life history and physiology in a marine metazoan? *J. Exp. Biol.* **220,** 551–563 (2017).
50. Dixon, G. B. *et al.* Genomic determinants of coral heat tolerance across latitudes. *Science* **348,** 1460–1462 (2015).
51. Willis, B. L. Phenotypic plasticity versus phenotypic stability in the reef corals Turbinaria mesenterina and Pavona cactus. *Proc. Fifth Int. Coral Reef Symp.* **4,** 107–112 (1985).
52. Kenkel, C. D. & Matz, M. V. Gene expression plasticity as a mechanism of coral adaptation to a variable environment. *Nat. Ecol. Evol.* **1,** 0014 (2016).
53. Burton, T. & Metcalfe, N. B. Can environmental conditions experienced in early life influence future generations? *Proc. R. Soc. B* **281,** 20140311 (2014).
54. Burgess, S. C. & Marshall, D. J. Adaptive parental effects: the importance of estimating environmental predictability and offspring fitness appropriately. *Oikos* **123,** 769–776 (2014).
55. Galloway, L. F. & Etterson, J. R. Transgenerational plasticity is adaptive in the wild. *Science* **318,** 1134–1136 (2007).
56. Baird, A. H., Guest, J. R. & Willis, B. L. Systematic and biogeographical patterns in the reproductive biology of scleractinian corals. *Ann. Rev. Ecol. Evol. Syst.* **40,** 551–571 (2009).
57. Richmond, R. H. Competency and dispersal potential of planula larvae of a spawning versus a brooding coral. In *Proc. 6th Int. Coral Reef Symp.* **2,** 827–831 (1988).
58. Crean, A. J. & Marshall, D. J. Coping with environmental uncertainty: dynamic bet hedging as a maternal effect. *Philos. Trans. R. Soc. B* **364,** 1087–1096 (2009).
59. Padilla-Gamiño, J. L., Pochon, X., Bird, C., Concepcion, G. T. & Gates, R. D. From parent to gamete: vertical transmission of Symbiodinium (Dinophyceae) ITS2 sequence assemblages in the reef building coral Montipora capitata. *PLoS One* **7,** e38440 (2012).
60. Highsmith, R. C. Reproduction by fragmentation in corals. *Mar. Ecol. Prog. Ser.* **7,** 207–226 (1982).
61. Ayre, D. J. & Resing, J. M. Sexual and asexual production of planulae in reef corals. *Mar. Biol.* **90,** 187–190 (1986).
62. Devlin-Durante, M. K. & Miller, M. W., Caribbean Acropora Research Group, Precht, W. F. & Baums, I. B. How old are you? Genet age estimates in a clonal animal. *Mol. Ecol.* **25,** 5628–5646 (2016).
63. Reusch, T. B. H. Climate change in the oceans: evolutionary versus phenotypically plastic responses of marine animals and plants. *Evol. Appl.* **7,** 104–122 (2014).
64. Hall, V. R. & Hughes, T. P. Reproductive strategies of modular organisms: comparative studies of reef-building corals. *Ecology* **77,** 950–963 (1996).
65. Barfield, S., Aglyamova, G. V. & Matz, M. V. Evolutionary origins of germline segregation in Metazoa: evidence for a germ stem cell lineage in the coral Orbicella faveolata (Cnidaria, Anthozoa). *Proc. R. Soc. B* **283,** 20152128 (2016).
66. Schweinsberg, M., Pech, R. A. G., Tollrian, R. & Lampert, K. P. Transfer of intracolonial genetic variability through gametes in Acropora hyacinthus corals. *Coral Reefs* **33,** 77–87 (2013).
67. Rohwer, F. *et al.* Diversity and distribution of coral-associated bacteria. *Mar. Ecol. Prog. Ser.* **243,** 1–10 (2002).
68. Bourne, D. G., Morrow, K. M. & Webster, N. S. Insights into the coral microbiome: underpinning the health and resilience of reef ecosystems. *Annu. Rev. Microbiol.* **70,** 317–340 (2016).
69. Douglas, A. E. & Werren, J. H. Holes in the hologenome: why host-microbe symbioses are not holobionts. *mBio* **7,** e02099-15 (2016).
70. Chakravarti, L. J., Beltran, V. H. & van Oppen, M. J. H. Rapid thermal adaptation in photosymbionts of reef-building corals. *Glob. Change Biol.* http://dx.doi.org/10.1111/gcb.13702 (2017).
Experimental demonstration of rapid genetic adaptation of *Symbiodinium* to increased water temperatures.
71. van Oppen, M. J., Baker, A. C., Coffroth, M. A. & Willis, B. L. In *Coral Bleaching* 83–102 (Springer, 2009).
72. Rowan, R. Review—diversity and ecology of zooxanthellae on coral reefs. *J. Phycol.* **34,** 407–417 (1998).
73. Howells, E. J. *et al.* Coral thermal tolerance shaped by local adaptation of photosymbionts. *Nat. Clim. Change* **2,** 116–120 (2012).
74. Hume, B. C. C. *et al.* Ancestral genetic diversity associated with the rapid spread of stress-tolerant coral symbionts in response to Holocene climate change. *Proc. Natl Acad. Sci. USA* **113,** 4416–4421 (2016).
75. Poland, D. M. & Coffroth, M. A. Trans-generational specificity within a cnidarian–algal symbiosis. *Coral Reefs* **36,** 119–129 (2017).
76. Jones, A. M., Berkelmans, R., van Oppen, M. J. H., Mieog, J. C. & Sinclair, W. A community change in the algal endosymbionts of a scleractinian coral following a natural bleaching event: field evidence of acclimatization. *Proc. R. Soc. B* **275,** 1359–1365 (2008).
77. Ziegler, M. *et al.* Coral microbial community dynamics in response to anthropogenic impacts near a major city in the central Red Sea. *Mar. Pollut. Bull.* **105,** 629–640 (2016).
78. Howells, E. J., Abrego, D., Meyer, E., Kirk, N. L. & Burt, J. A. Host adaptation and unexpected symbiont partners enable reef-building corals to tolerate extreme temperatures. *Glob. Change Biol.* **22,** 2702–2714 (2016).
Demonstration of the role of *Symbiodinium* community composition on corals' thermal tolerance.
79. Quigley, K. M., Willis, B. L. & Bay, L. K. Maternal effects and Symbiodinium community composition drive differential patterns in juvenile survival in the coral Acropora tenuis. *R. Soc. Open Sci.* **3,** 160471 (2016).
80. Sharp, K. H., Distel, D. & Paul, V. J. Diversity and dynamics of bacterial communities in early life stages of the Caribbean coral Porites astreoides. *ISME J.* **6,** 790–801 (2012).
81. Quigley, K. M. *et al.* Deep-sequencing method for quantifying background abundances of Symbiodinium types: exploring the rare Symbiodinium biosphere in reef-building corals. *PLoS One* **9,** e94297 (2014).
82. Lee, M. J. *et al.* Most low-abundance "background" Symbiodinium spp. are transitory and have minimal functional significance for symbiotic corals. *Microb. Ecol.* **71,** 771–783 (2016).
83. Bay, L. K., Doyle, J., Logan, M. & Berkelmans, R. Recovery from bleaching is mediated by threshold densities of background thermo-tolerant symbiont types in a reef-building coral. *R. Soc. Open Sci.* **3,** 160322 (2016).
84. McFall-Ngai, M. *et al.* Animals in a bacterial world, a new imperative for the life sciences. *Proc. Natl Acad. Sci. USA* **110,** 3229–3236 (2013).
85. Ainsworth, T. D. *et al.* The coral core microbiome identifies rare bacterial taxa as ubiquitous endosymbionts. *ISME J.* **9,** 2261–2274 (2015).
86. Neave, M. J. *et al.* Differential specificity between closely related corals and abundant Endozoicomonas endosymbionts across global scales. *ISME J.* **11,** 186–200 (2017).
87. Ziegler, M., Seneca, F. O., Yum, L. K., Palumbi, S. R. & Voolstra, C. R. Bacterial community dynamics are linked to patterns of coral heat tolerance. *Nat. Commun.* **8,** 14213 (2017).
88. Hernandez-Agreda, A., Leggat, W., Bongaerts, P. & Ainsworth, T. D. The microbial signature provides insight into the mechanistic basis of coral success across reef habitats. *mBio* **7,** e00560-16 (2016).
89. Röthig, T., Yum, L. K., Kremb, S. G., Roik, A. & Voolstra, C. R. Microbial community composition of deep-sea corals from the Red Sea provides insight into functional adaption to a unique environment. *Sci. Rep.* **7,** 44714 (2017).
90. Casadesús, J. & Low, D. A. Programmed heterogeneity: epigenetic mechanisms in bacteria. *J. Biol. Chem.* **288,** 13929–13935 (2013).
91. Celluzzi, A. & Masotti, A. How our other genome controls our epi-genome. *Trends Microbiol.* **24,** 777–787 (2016).
92. Roossinck, M. J. The good viruses: viral mutualistic symbioses. *Nat. Rev. Microbiol.* **9,** 99–108 (2011).
93. Shui, J.-W. *et al.* HVEM signalling at mucosal barriers provides host defence against pathogenic bacteria. *Nature* **488,** 222–225 (2012).
94. Barr, J. J., Youle, M. & Rohwer, F. Innate and acquired bacteriophage-mediated immunity. *Bacteriophage* **3,** e25857 (2013).
95. Rohwer, F. & Vega Thurber, R. L. Viruses manipulate the marine environment. *Nature* **459,** 207–212 (2009).
96. Vega Thurber, R. L., Payet, J. P., Thurber, A. R. & Correa, A. M. S. Virus-host interactions and their roles in coral reef health and disease. *Nat. Rev. Microbiol.* **15,** 205–216 (2017).
Seminal review of the role of viruses in the phenotypic performance of the coral holobiont.
97. Raina, J. B. *et al.* DMSP biosynthesis by an animal and its role in coral thermal stress response. *Nature* **502,** 677–680 (2013).
98. Correa, A. M. S. *et al.* Viral outbreak in corals associated with an in situ bleaching event: atypical herpes-like viruses and a new megavirus infecting Symbiodinium. *Front. Microbiol.* **7,** 127 (2016).
99. Levin, R. A., Voolstra, C. R., Weynberg, K. D. & van Oppen, M. J. H. Evidence for a role of viruses in the thermal sensitivity of coral photosymbionts. *ISME J.* **11,** 808–812 (2017).
100. Soffer, N., Brandt, M. E., Correa, A. M., Smith, T. B. & Vega Thurber, R. L. Potential role of viruses in white plague coral disease. *ISME J.* **8,** 271–283 (2014).
101. LaJeunesse, T. C., Lee, S. Y., Gil-Agudelo, D. L., Knowlton, N. & Jeong, H. J. Symbiodinium necroappetens sp. nov. (Dinophyceae): an opportunist 'zooxanthella' found in bleached and diseased tissues of Caribbean reef corals. *Eur. J. Phycol.* **50,** 223–238 (2015).

102. Harvell, D. *et al.* Coral disease, environmental drivers, and the balance between coral and microbial associates. *Oceanography* **20,** 172–195 (2007).
103. van Oppen, M. J. H., Leong, J. A. & Gates, R. D. Coral-virus interactions: a double-edged sword? *Symbiosis* **47,** 1–8 (2009).
104. Sampayo, E. M. *et al.* Coral symbioses under prolonged environmental change: living near tolerance range limits. *Sci. Rep.* **6,** 36271 (2016).
105. Sarda, S., Zeng, J., Hunt, B. G. & Yi, S. V. The evolution of invertebrate gene body methylation. *Mol. Biol. Evol.* **29,** 1907–1916 (2012).
106. Theis, K. R. *et al.* Getting the hologenome concept right: an eco-evolutionary framework for hosts and their microbiomes. *mSystems* **1,** e00028-16 (2016).
107. Ghalambor, C. K. *et al.* Non-adaptive plasticity potentiates rapid adaptive evolution of gene expression in nature. *Nature* **525,** 372–375 (2015).
108. Kronholm, I. & Collins, S. Epigenetic mutations can both help and hinder adaptive evolution. *Mol. Ecol.* **25,** 1856–1868 (2016).
109. Ancel, L. W. Undermining the Baldwin expediting effect: does phenotypic plasticity accelerate evolution? *Theor. Popul. Biol.* **58,** 307–319 (2000).
110. Marshall, D. J. Transgenerational plasticity in the sea: context-dependent maternal effects across the life history. *Ecology* **89,** 418–427 (2008).
111. Messer, P. W. & Petrov, D. A. Population genomics of rapid adaptation by soft selective sweeps. *Trends Ecol. Evol.* **28,** 659–669 (2013).
Review of mechanisms that produce soft selective sweeps, with a case for soft sweeps dominating rapid adaptation in many species.
112. Whiteley, A. R., Fitzpatrick, S. W., Funk, W. C. & Tallmon, D. A. Genetic rescue to the rescue. *Trends Ecol. Evol.* **30,** 42–49 (2015).
113. Holeski, L. M., Jander, G. & Agrawal, A. A. Transgenerational defense induction and epigenetic inheritance in plants. *Trends Ecol. Evol.* **27,** 618–626 (2012).
114. Parker, L. M. *et al.* Adult exposure influences offspring response to ocean acidification in oysters. *Glob. Change Biol.* **18,** 82–92 (2012).

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Author contributions

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Additional information

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Competing financial interests

The authors declare no competing financial interests.