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Evolutionary significance of plastic responses within and across generations

Zuzana Sekajova

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Evolutionary significance of plastic responses within and across generations

Zuzana Sekajova
Department of Ecology and Genetics / Animal Ecology
Evolutionary Biology Centre
Uppsala University
Norbyvägen 18D
SE-752 36 Uppsala
Sweden

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Contents

Abstract

Until recently, all the resemblances between parents and their offspring were often ascribed to genes. Indeed, there is no doubt that DNA plays a central role in heredity. However, in recent years, it has become apparent that various non-genetic factors, independent of the DNA sequence, can also be passed from one generation to another, and in some cases even over as many as 80 generations. These effects include phenomena such as parental effects and epigenetic inheritance; two forms of trans-generational plasticity. In contrast to trans-generational plasticity, which allows for trait transmission across multiple generations, within-generational plasticity acts within one generation and includes phenomena such as environment-specific trait expression or catch-up growth. Plastic responses are especially advantageous in heterogeneous environment in which genetic mutations may not arise fast enough to allow organisms to cope with new conditions. In such cases, plastic responses may offer a better strategy and character of the environment will determine which form of plastic responses will be present. Both within- and trans-generational plasticity may alter the rate and direction of adaptation and can therefore have important implications for evolution. However, each type of plastic response is distinct and will thus alter adaptation in a unique way. Here I provide a detailed description of some of the most important forms of plastic responses and their consequences for adaptation.

Introduction

In a stable environment, 'classic' genetic inheritance which results in a strong resemblance between parents and their offspring should be sufficient for organismal persistence (Leimar and McNamara 2015). However, the environment (including both, biotic and abiotic factors) is rarely stable, and is often changing from one state to another. In the face of climatic change, this is true more than ever before. Climate change results in increased rate of change in mean measures (gradual change) (Berteaux 2004) but also in higher occurrence and magnitude of extreme events leading to increased environmental variability (Easterling 2000; Palmer and Räisänen 2002; Van Aalst 2006). It remains, however, unclear whether genetic adaptation is fast enough to allow organism to adapt to rapidly changing conditions (Berteaux, 2004). A mechanism that may help organisms to deal with environmental change is phenotypic plasticity (Kingsolver and Buckley 2017), which can either be expressed within a generations or across generations. If the environmental change occurs fast relative to a generation time, environmental variability will result in evolution of within-generational plasticity (Uller et al. 2013), which is an ability of an organism to express a range of phenotypes depending on the environment (West-Eberhard 2003). Plasticity extended between generations is referred to as trans-generational plasticity (Bonduriansky et al. 2012). If transferred only from one generation to the next, this form of plasticity can also be referred to as inter-generational plasticity or as a parental effect. To conclude if an observation is indeed the result of trans-generational plasticity, rather than within-generational plasticity, offspring of the second generation (in the case of paternal transmission) or third generation (in case of maternal transmission) after the stimulus would need to exhibit an altered phenotype (Perez and Lehner 2019). For example, if the environment alters a gestating embryo with already formed oocytes within this embryo, any effect on the oocytes and offspring coming from those oocytes would not be considered a trans-generational plasticity (Bonduriansky and Day 2018). The reason for this is that effects on the offspring (oocytes) could be direct result of environment without the effect being necessarily transmitted through the parents. Not only parental traits, but also the parent's experience or effect of environment or age can be passed onto the offspring (Bonduriansky and Day 2018). For example, smoking in humans has very different effect on the mother and her unborn child; while mothers risk developing respiratory or circulatory problems, the child can be born with a reduced birth weight and behavioral disorders (Knopik et al. 2012). In other words, even traits, that are not apparent or present in the parents can be transferred to the offspring (Pembrey et al. 2014). Mechanisms of trans-generational

transmission of traits independently (or at least partially independently) of genes are numerous: from transfer of microbiome (Fridmann-Sirkis et al. 2014), hormones (Groothuis and Schwabl 2008), nutrients (Wells 2003), antibodies (Boulinier and Staszewski 2008), epigenetic markers (such as small RNAs), methylation or modifications of histone from parents to offspring to effects of behavior and niche construction (Bonduriansky and Day, 2009; Bonduriansky et al., 2012).

Besides the duration (within, inter, trans-generational), plastic responses can also be differentiated based upon the underlying mechanisms as genome-associated (transfer of small RNAs, methylation or histone modifications) and genome-independent (microbiome transfer) (Skinner 2008). They can be also differentiated upon the trigger of arising variation. It can be triggered either by environmental conditions ('acquired traits') or spontaneously (Lachmann and Jablonka 1996), for example due to random changes in DNA-methylation (Richards 2006). As it will be shown later, both, within and trans-generational plasticity can alter rate and direction of adaptation (Lachmann and Jablonka 1996) with the effect being either neutral, positive or in other cases negative for the rate of adaptation. While within-generational plasticity is well studied in an evolutionary context, most studies of especially trans-generational plasticity look on the effects from a perspective of disease (Burggren 2016) with other effects being relatively unexplored. This is true particularly for positive effects which can potentially have a big impact for medicine (Vaiserman 2008; Felling and Song 2015) and agriculture (Goddard and Whitelaw 2014; López-Arredondo et al. 2015; Bilichak and Kovalchuk 2016) and therefore study of plastic responses still require more attention.

Overview of plastic responses

Within-generation phenotypic plasticity

Phenotypic plasticity (within-generational plasticity) is commonly described as an ability of an organism to express different phenotypes depending on the environment (West-Eberhard 2003). A classic example of phenotypic plasticity are life history and morphological changes related to presence (versus absence) of chemical cues from predators in *Daphnia*. In a

presence of large predators such as fish (Stibor 1992) *Daphnia* decreases in size but increases in size in a presence of small predators such as phantom midge *Chaoborus* (Stibor and Lüning 1994). Presence of predators can also result in helmet enlargement (Tollrian 1990) or formation of neckteeth (Tollrian 1993). Phenotypic plasticity may allow organisms to respond to new conditions in a rate significantly higher than adaptations (Kingsolver and Buckley 2017). Some degree of plasticity may be already present in the population; in fact, plasticity is rather a common strategy. For example, research on climate change implies that most of what we consider an evolutionary adaptation to climate change might instead result from plastic responses (for review see Merilä and Hendry, 2014). Phenotypic plasticity is mostly adaptive in a fluctuating environment that changes fast relative to a generation time and that offers reliable (accurately predicting the future) cues about the future environment (Uller et al. 2013).

Phenotypic plasticity and evolution

Despite a large number of studies on plastic responses, there is a general disagreement weather plasticity constrains or facilitate adaptive evolution (Ancel 2000; Price et al. 2003; Ghalambor et al. 2007, 2015; Lande 2009; Chevin et al. 2010). Some argue that, by moving the phenotype closer to the new fitness peak, plasticity weakens the strength of selection, which might hinder adaptation (Falconer, 1981). In an extreme case, when the plastically induced phenotype matches the optimal phenotype, there is no need for a genetic adaptation (Price et al. 2003). On the other hand, plasticity can help organisms to persist under new environmental conditions and prevent them from extinction, in which case it would aid an adaptation as populations have more time to respond genetically (Simpson 1953). If the phenotype resulting from a plastic response is in the same direction as the phenotype favored by selection, we talk about adaptive plasticity (Ghalambor et al. 2007). However, plasticity is not always adaptive. If the resulting phenotype is further away from the optimal value, plasticity is considered non-adaptive (Ghalambor et al. 2007). Counterintuitively, also non-adaptive plasticity can eventually result in (Rutherford and Lindquist 1998; Ruden et al. 2003) or speed up (Ab Ghani and Merilä 2015; Ghalambor et al. 2015; Schmid and Guillaume 2017) adaptation; for example by increasing distance of a trait from a phenotypic optimum and thus increasing the strength of selection (Ghalambor et al. 2015). If the plastic response is antagonistic to the genetic response, it can lead to an apparent stasis when the trait does

respond to selection but phenotype remains unchanged (Merilä et al. 2001). Another possible outcome is a hyper-plasticity, when plasticity is in the same direction as the optimal phenotype, but the response is steeper than would be optimal and the new phenotype appears on the other side of the fitness peak, "overshooting" the optimum (King and Hadfield 2019). From this we can see that whether plasticity is adaptive depends on the environment (Ghalambor et al. 2007) and the relationship between phenotypic plasticity and adaptation is very complex and context dependent.

Costs and limits of plasticity

If plasticity helps organisms to adjust to current conditions, why are all organisms not maximally plastic? The reason could be that plasticity comes with costs or limitations. While the costs of plasticity result in decreased fitness even when an organism reaches the optimal phenotype (compared to the fitness of a non-plastic individual expressing the same phenotype), limits are the inability to express the optimal phenotype at all (Pigliucci 2005). Plasticity can be helpful within a certain range of environments, beyond which it might reach its limit and become insufficient or even maladaptive (Chevin et al. 2010). Physiological limits of phenotypic plasticity may be more easily reached in extreme environments (Rocha et al. 2009). One of the limitations is the reliance on environmental cues (Moran 1992; Simons 2011). In the case when the reliability of cues decreases, plasticity is predicted to decrease (Gavrilets and Scheiner 1993). If it results in mismatched phenotype, the cost could be, a decrease in fitness or, in an extreme case, a population extinction due to increasing distance of phenotype from a fitness optimum (Reed et al. 2010; Chevin et al. 2013). Especially with human induced changes we do observe a decoupling of environmental cues and optimal responses. For example, the yellow-bellied marmot (*Marmota flaviventris)* is emerging from hibernation earlier, as a response to warmer air temperature early in the spring. However, the day of snowmelt has remained unchanged, leading to decreased foraging opportunities (Inouye et al. 2000). Another example is decoupling of a day length, that remains constant, and temperature that is increasing earlier in the year. Other potential costs are costs of physiological machinery that allows organisms to acquire and interpret cues about environment or deleterious pleiotropy of genes that are encoding plasticity (Dewitt et al. 1998). Despite costs of plasticity being well developed on a theoretical level, studies in the

field suggest that costs may not be as important as previously thought (Buskirk and Steiner 2009).

Catch-up growth

Adverse conditions experienced by individuals in early stages of their lives, such as limited food intake or low temperature, can result in decreased growth. If the conditions later improve those individuals may react by accelerating their growth, sometimes in a rate even higher than would be normal, to 'catch-up' on weight of individuals with a standard growth rate (Figure 1.). This form of within-generational plasticity is called catch-up (or compensatory) growth (Metcalfe and Monaghan 2001). Examples of catch-up growth come from various organisms such as mice (Ozanne and Hales 2004), zebra finches (Fisher et al. 2006) and fish such as sticklebacks (Ab Ghani and Merilä 2015), green swordtails (Royle et al. 2006) and salmon (Morgan and Metcalfe 2001). The ability to increase growth rate relatively to the normal points to that organisms don't always grow in a maximum possible rate. This is interesting, taking into account that a large body size and a short development often increases fitness (Chown and Gaston 1997). Possible explanations to why organisms are not generally growing at their maximum rate could be trade off with other functions, such as lifespan (Ozanne and Hales 2004) or reduced physical (Royle et al. 2006) and cognitive (Fisher et al. 2006) function. However, sometimes no trade-off is found which may point out to the importance to search for a trade off in a trans-generational context. For example, in an experiment conducted on nematode worms *C. elegans* juvenile worms were exposed to a cold temperature, which resulted in decreased growth when compared to individuals from a control treatment. After return to the standard temperature, worms increased their growth and grew into size comparable with controls. Interestingly, cold-exposed individuals have higher total reproduction and fitness and lifespan comparable to standard treatment individuals, there was no apparent trade-off. Possible explanation could be trade-off in the form of decreased offspring quality (Rosa et al. 2020). Catch-up growth is predicted to be adaptive in an environment that is rapidly changing (Frézal and Félix 2015) or where the development and reproduction are under time constrains (Dahl et al. 2012). It can be quantified by a compensatory index, a ratio of the difference between weight of the animal at the end of a restricted period and a compensatory growth period relative to the growth at the end of the restricted growth alone, so that for example, value of 100% would indicate a full catch-up

(Wilson and Osbourn 1960). In some cases, however, the rate can be higher than 100%, which results in overcompensation so that individuals originating from restricted conditions grow bigger than unrestricted individuals (Ab Ghani and Merilä 2015).

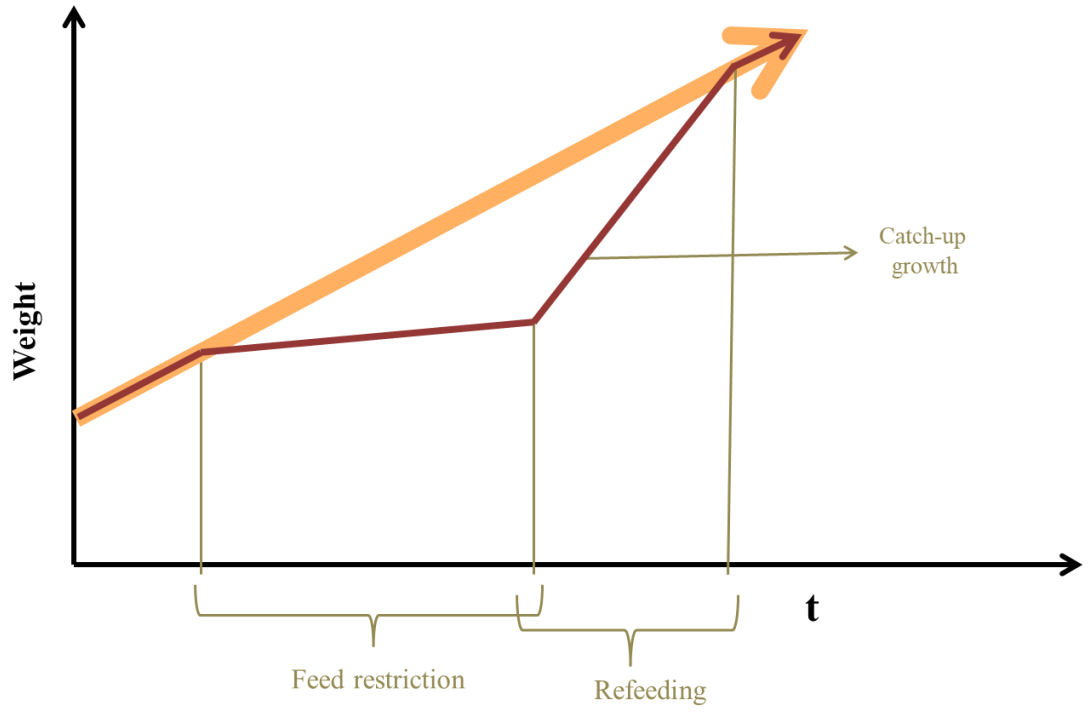

Figure 1. After restricted food intake individuals may decrease their growth rate and consequently increase the growth rate if the food availability improves. The rate of the growth (slope) is, in this case even higher than in unrestricted individuals (Catch-up growth). At the end of the growth period, both restricted and unrestricted individuals reach the same size (adapted from Nicieza and Wootton 2003).

Bet-hedging

If there are no reliable cues about the future in a variable environment, bet-hedging will be favored by selection over phenotypic plasticity (Moran 1992; Simons 2011). Bet-hedging is based on lowering variance in fitness between events (arithmetic mean fitness) to maximize long-term fitness (geometric mean fitness) (Slatkin 1974). There are two different ways to lower variation in fitness: conservative bet-hedging ("always play it safe") when individuals

produce offspring of generalist phenotype (sometimes referred to as a generalist strategy), and diversified bet-hedging ("don't put all your eggs in one basket") when individuals produce offspring of diverse phenotypes, so that at least some of them will match the environment (Philippi and Seger 1989). In contrast to conservative bet-hedging that results in a low variance in fitness (and in trait values) among offspring originating from the same parents, diversified bet-hedging may increase variance in fitness (and in trait values) among the offspring. Parents can further diversify offspring of the same cohort or offspring among different cohorts. In the second case, offspring originating from one cohort will be similar compared to their same-cohort siblings but different from siblings from different cohorts (characteristics of each cohort are determined stochastically) (Childs et al. 2010). This strategy is sometimes referred to as "adaptive coin flipping" (Cooper and Kaplan 1982). This would be for example, equivalent to flipping a coin every year and based on outcome deciding, weather to produce phenotype suitable for a wet year or a dry year (Olofsson et al. 2009). The optimal bet-hedging strategy will depend on a likelihood of extreme events (Olofsson et al. 2009).

Parental effects

Transfer of hormones (Groothuis and Schwabl 2008), nutrients (Wells 2003) or antibodies (Boulinier and Staszewski 2008) from parents to the offspring shaping offspring phenotype falls into the category of parental effects; a form of trans-generational plasticity that lasts only one generation (Uller 2008). Parental effects can be described as effect of parental phenotype on the phenotype of the offspring without direct alteration of offspring DNA (Wolf and Wade 2009). Such effects can be divided into maternal and paternal effects, which are very similar in principle, and analogous conclusions can be drawn for paternal and maternal effects. An example of maternal effects would be a placement of antibodies against specific pathogens into the egg yolks, as occurring in some species of birds (Gasparini et al. 2001) or the egg or propagule size itself (Parsons 1970; Grant 1991; Williams 1994). Whereas an example of a paternal effect would be a seminal fluid containing various nutrients and influencing development when passed to the offspring (Crean et al. 2016). Parental effects are diverse and can influence offspring in many different ways. In some cases, offspring directly inherits the parental phenotype. Compared to within-generational plasticity which relies on environmental cues reflecting the future state of an environment, parental effects will then be based on

relying on parental phenotype matching and representing the environment (Uller 2008; Shea et al. 2011). In other cases, parents can anticipate the future environment and prepare the offspring according the expectations, regardless on whether their own phenotype matches the environment. For example, offspring can develop traits opposite to those of their parents, if the parent expects the traits will increase offspring fitness and the traits can be influenced by parental effects (Dey et al. 2016). In other words, direction of parental effects will depend on the environmental correlation; if the correlation is positive, this will result in evolution of positive parental effects when offspring phenotype follows parental phenotype. However, if the correlation is negative (environment of parents always differs from environment of the offspring) result will be negative parental effects (Dey et al. 2016; Lind et al. 2020). Another type of parental effects is a condition transfer. High condition is connected with higher amount of metabolic resources and individuals in a high condition may be able to convert more resources into fitness-enhancing traits and therefore have higher fitness than low condition individuals (Bonduriansky and Crean 2018). High condition parents may therefore be able to provide more resources to the offspring (Parker 2002; Pooley et al. 2014), resulting in resemblance between parent and offspring because of transfer of parental condition (Bonduriansky and Crean 2018). Yet, another category is selfish parental effects. Those are usually the result of parent-offspring conflict which causes the optimal level of plasticity between parents (usually mother) and offspring to be mismatched (Uller 2008). As a result, parental effects act to increase parental rather than offspring fitness which sometimes comes with a cost of decreased offspring fitness (Marshall and Uller, 2007). For example, a mother that mate with males of lower quality may decrease investment into the offspring with those males, which will result in lower offspring fitness (Cunningham and Russell 2000). Parental effects don't always increase fitness of parents or their offspring, for example, mothers sometimes transfer pathogens (Bernardo 1996) to the next generation. Marshall and Uller call this type of parental effects 'transmissive maternal effects' as the effect of maternal phenotype is transmitted to the offspring despite being maladaptive (Marshall and Uller 2007). The direction of the maternal effects can also vary and will depend on the character of the environment; sometimes decreasing and sometimes increasing phenotypic variance (Kuijper and Hoyle 2015). In constant environments, negative maternal effects will help to reduce deviations from a mean phenotypic value, thus keeping phenotype of the population within a narrow range. This is because in a constant environment, the population is already likely close to the fitness peak and most of the variation will be caused by random perturbations. Keeping phenotype close to the population mean will thus keep it close to the phenotypic optimum

(Uller 2008). In contrast, in a population experiencing rapid environmental change, maternal effects will reach positive value (change in the trait value of the offspring will be in the same direction as the parental phenotype), helping offspring to adjust their phenotype to cope with the new stressor. Individuals that survived and reproduced despite a stressor are likely to be closer to the new phenotypic optimum and their offspring will thus benefit from resembling parental phenotype (Kuijper and Hoyle 2015). Yet, another scenario is a periodically changing environment in which equilibrium is never reached. Under such conditions evolution of parental effects will be much more complex and will depend on the strength of selection and presence versus absence of phenotypic plasticity (for more information see Kuijper and Hoyle 2015).

Parental effects and evolution

Parental effects could potentially influence the evolution in ways similar to within-generational plasticity. Even though parental effects can alter response to selection (Kirkpatrick and Lande 1989; Hoyle and Ezard 2012) and play an important role in parent-offspring co-adaptations (Kölliker 2005) their role for adaptation is still under question. For parental effects to be adaptive, parental phenotype needs to have an informative value about the future. However, this may not be very common in natural environments (Uller 2008). For example, parents may compensate for the adverse conditions by plastically altering their own phenotype in which case the cues passed to the offspring will be buffered and won't contain information about the adversity (Uller and Pen 2011). Thus, compared to within-generational plasticity, parental effects have relatively weak effect on adaptation (Kuijper and Hoyle 2015) and weak effect on offspring phenotype (Uller et al. 2013). However, some authors speculate that because of an early effect on individuals some of parental effects can be mistakenly assigned to genetic effects (Danchin et al. 2011). Similarly for within-generational plasticity, an important factor is also whether parental effects are costly. For example, maternal investment in a large egg size that increases fitness of the offspring may come with a cost of reduced offspring number (Marshall and Uller 2007). Costs can be also present in the offspring; for example exhibiting costly behavior in birds may reduce energy sources that could be converted into growth (Mock and Parker 1997).

Trans-generational epigenetic inheritance

The term epigenetics (epigenetic inheritance) is in the literature used in two different contexts; epigenetics in a broad sense that includes any kind of non-genetic inheritance (including maternal effects, phenotypic plasticity, etc.) and epigenetics in a narrow sense representing transmission of epigenetic markers (see below) across generations. Throughout this essay, the term epigenetics is used in a narrow sense.

Epigenetic markers can be divided into three categories: DNA methylation, histone modifications and small RNAs. A relatively common mechanism of epigenetic alteration is DNA methylation: that is the bonding of methyl groups to DNA bases. Methylation is known to interfere with translation of DNA sequences into RNA, which results in methylated bases being differentially expressed. DNA methylation can have some dramatic consequences for the organism, for example in mice, simple methylation of a specific retrotransposon results in complete change of coat color (Wolff 1978). Another mechanism is histone modifications; changes in three-dimensional structure of chromosomes. If modifications of histone results in tightly packed regions of chromosomes those regions will be less exposed to transcription factors and therefore gene expression will be repressed and vice versa. The last mechanism is transfer of small non-coding RNAs including piwi-interacting RNAs, micro-RNAs and transfer RNA-derived RNAs. As the name suggests, they are not translated into proteins, instead they have functions in the regulation of gene expression. For example, some small non-coding RNAs can bind to mRNAs and disrupt their translation into peptides, which is known as RNA interference or RNAi, and others can affect a three-dimensional structure of chromosomes and influence a gene expression as described above.

By determining how DNA is read they have an important role in cell differentiation, enabling initially identical cells to carry out different functions. Presence (or absence) of epigenetic markers in the DNA is influenced by various environmental factors (Jablonka and Raz 2009). Changes in epigenetic markers are usually ‘reset’ in each generation, preventing their transmission across generations. In mammals, for example, embryonic and primordial cells are first de-methylated, to be subsequently re-methylated and start with a clean state (Seisenberger et al. 2013). However, there has been multiple studies showing incomplete epigenetic resetting, leading to transmission of the markers from the parental generation to the offspring, resulting in trans-generational epigenetic inheritance (Chandler and Stam 2004;

Vastenhouw et al. 2006; Jablonka and Raz 2009); a form of trans-generational plasticity. Detailed evidence for epigenetic inheritance comes, for example, from the nematode worms *Caenorhabditis elegans.* In one study, worms were injected with a double-stranded RNA resulting in a gene silencing via RNA interference (RNAi) and the epigenetically modified phenotype caused by a single episode of RNAi persisted over as many as 80 generations (Vastenhouw et al. 2006). In another experiment, also conducted in *C. elegans*, RNAi caused by manipulation of piwi-interacting RNA lasted over 20 generations (Ashe et al. 2012). Environmentally induced trans-generational epigenetic inheritance was demonstrated by Rechavi et al. (2014) on *C. elegans* worms; experimentally showing that starvation-induced changes were transferred over at least three generations (Rechavi et al. 2014).

Because epigenetic markers determine whether a gene will be expressed or not, some researchers imagine epigenetic markers as on/off switches with the effect being either present or absent. Yet, evidence suggest that, at least in some cases, epigenetically inherited trait must be under environmental influences for several generations to be fully expressed – 'wash in' effect and few generations in the absence of the environmental factor to fully disappear – 'wash out' effect (Corrales et al. 2014) which may influence a magnitude of the effect they have on the phenotype.

Epigenetic inheritance and evolution

Despite the mounting evidence for trans-generational transfer of epigenetic markers (Vastenhouw et al. 2006, Rechavi et al. 2014), researchers cannot agree weather it is a result of a mere error or has an adaptive role. Some argue that epigenetic inheritance is, indeed, adaptive with the beneficial role in heterogeneous environment. For example, Uller et al. (2015) showed that epigenetic inheritance would be adaptive in an environment that changes infrequently relative to a generation time. As Uller et al. (2015) explains, if an individual can respond to the environment by epigenetic alterations of the phenotype, and if the epigenetic markers are not passed to another generation, only the first generation after the change is going to be negatively impacted by the changed environment. However, in a case of epigenetic transmission to the next generations, the phenotype of two or more consecutive generations may be mismatched (as their phenotype is still "following" the original environment). Therefore epigenetic inheritance is only adaptive in highly auto-correlated environment; that is an environment in which environmental conditions for one generation are

correlated with conditions of the next generation. Another factor important for the evolution of epigenetic inheritance is the absence of reliable parenta or environmental cues (Uller et al. 2015). If the cues are reliable, it may be better for an individual to adjust its phenotype according to those (Uller et al. 2013). If environment is changing slowly, the environmental correlation between generations is high and the offspring is likely to experience the same conditions that their parents and thus maternal environment will have a high informative value for the offspring (Uller et al. 2015). On the other hand, if the environment is changing rapidly and randomly, then the parental environment doesn't represent a good cue for the offspring and within-generational plasticity would be selected for instead (Moran 1992; Simons 2011).

When adapting to new conditions, an important factor is the speed of the response. Both genetic mutations and epigenetic inheritance can lead to an altered phenotype that may increase fitness in the new environment. However, epigenetic modifications enable rapid change that can't be reached via genetic mutations (Rando and Verstrepen 2007). While it typically may take hundreds or thousands of generations for genetic mutations to have a substantial effect on a population performance, epigenetic inheritance can have an immediate effect (Rando and Verstrepen 2007). The reason is a genetic mutation, even if positive, will spread throughout the population slowly, being expressed in perhaps only few more individuals each generation. The same epigenetic change may, however, arise among many individuals (even the entire population) simultaneously. This is because many individuals in a population may possess the same epigenetic markers and will likely be exposed to the same environmental stressors (Burggren 2016). Following the same logic, after an environmental stressor disappears, it may take many generations for a new, genetically determined, phenotype, to disappear from the population. This is reached by individuals carrying the mutation being replaced by individuals with a new mutation, beneficial in the original environment. Meanwhile, changes via epigenetic markers are easily reversible; the phenotype will rapidly 'switch' back to the original stage, within one, or, in a case of 'wash out' effect, a few generations (Klironomos et al. 2013; Burggren 2016). This is true not only in temporally changing environment, but also in a spatially variable environment (Schrey and Richards 2012). Moreover, epigenetic mutations, at least those caused by methylation, are much faster to occur (in several orders of magnitude) than mutations in DNA (Klironomos et al. 2013). This may result in more readily response to selection. All of this supports an idea of epigenetic inheritance being beneficial in heterogeneous environment.

What does it mean for evolutionary theory?

As we have seen, non-genetic inheritance can have some important consequences for evolution. However, some forms of non-genetic inheritance (trans-generational plasticity) has some important implication also for the evolutionary theory, some of them so significant, that portion of scientific community call for revising evolutionary synthesis that would, among others, include effect of non-genetic inheritance (Danchin et al. 2011). For example, the fact that many non-genetic factors can be influenced by external effects results in "inheritance of acquired traits" which was, since Darwin, considered impossible (Bonduriansky and Day 2018). Moreover, non-genetic inheritance can respond to selection; environmentally induced traits increase variation among individuals and as Bonduriansky and Day (2018) argue, any variation that is heritable can respond to natural selection. This opposes a standard definition of evolution that states that evolution sources solely from changes in allele frequencies across generations. All the factors stated above add on importance of better understanding of plastic responses.

Summary

As we have seen, plastic responses can help organisms to cope with variable environments where genetic adaptations may not be sufficient (Berteaux 2004) and by interacting with genetic adaptations they may influence rate and direction of evolutionary response (Ancel 2000; Price et al. 2003; Ghalambor et al. 2007, 2015; Lande 2009; Chevin et al. 2010). The effects of the plastic responses vary and depend on type of response but also on specific characteristics of the environment. For example, parental effects can sometimes increase (Dey et al. 2016) and in other cases decrease (Bernardo 1996; Marshall and Uller 2007) parental and offspring fitness. Despite increasing interest in plastic responses, trans-generational plasticity remains relatively unexplored and more research is needed for us to fully understand impact of plastic responses on organismal survival in heterogeneous environments and ability to adapt. This is especially important nowadays, with increasing rate of climate change that besides changes in mean measures also results in higher occurrence and magnitude of extreme events and consequent increase in environmental variability (Easterling 2000; Palmer and Räisänen 2002; Van Aalst 2006).

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References

Ab Ghani, N. I., and J. Merilä. 2015. Population divergence in compensatory growth responses and their costs in sticklebacks. Ecology and Evolution 5:7–23.

Ancel, L. W. 2000. Undermining the Baldwin expediting effect: does phenotypic plasticity accelerate evolution? Theoretical Population Biology 58:307–319.

Ashe, A., A. Sapetschnig, E.-M. Weick, J. Mitchell, M. P. Bagijn, A. C. Cording, A.-L. Doebley, L. D. Goldstein, N. J. Lehrbach, J. Le Pen, G. Pintacuda, A. Sakaguchi, P. Sarkies, S. Ahmed, and E. A. Miska. 2012. piRNAs can trigger a multigenerational epigenetic memory in the germline of *C. elegans*. Cell 150:88–99.

Bernardo, J. 1996. Maternal effects in animal ecology. American Zoologist 36:83–105.

Berteaux, D. 2004. Keeping pace with fast climate change: can arctic life count on evolution? Integrative and Comparative Biology 44:140–151.

Bilichak, A., and I. Kovalchuk. 2016. Transgenerational response to stress in plants and its application for breeding. Journal of Experimental Botany 67:2081–2092.

Bonduriansky, R., A. J. Crean, and T. Day. 2012. The implications of nongenetic inheritance for evolution in changing environments. Evololutionary Applications 5:192–201.

Bonduriansky, R., and T. Day. 2018. Extended Heredity: A new understanding of inheritance and evolution. Princeton University Press.

Bonduriansky, R., and T. Day. 2009. Nongenetic inheritance and its evolutionary implications. Annual Review of Ecology, Evolution, and Systematics 40:103–125.

Boulinier, T., and V. Staszewski. 2008. Maternal transfer of antibodies: raising immuno-ecology issues. Trends in Ecology & Evolution 23:282–288.

Burggren, W. 2016. Epigenetic inheritance and its role in evolutionary biology: re-evaluation and new perspectives. Biology 5:24. doi:10.3390/biology5020024

Buskirk, J. V., and U. K. Steiner. 2009. The fitness costs of developmental canalization and plasticity. Journal of Evolutionary Biology 22:852–860.

Chandler, V. L., and M. Stam. 2004. Chromatin conversations: mechanisms and implications of paramutation. Nature Reviews Genetics 5:532–544. doi: 10.1038/nrg1378

Chevin, L.-M., R. Gallet, R. Gomulkiewicz, R. D. Holt, and S. Fellous. 2013. Phenotypic plasticity in evolutionary rescue experiments. Philosophical Transactions of the Royal Society Londdon B Biological Sciences 368(1610), 20120089. doi: 10.1098/rstb.2012.0089

Chevin, L.-M., R. Lande, and G. M. Mace. 2010. Adaptation, plasticity, and extinction in a changing environment: towards a predictive theory. Public Library of Science Biology 8(4):e1000357.

Childs, D. Z., C. J. E. Metcalf, and M. Rees. 2010. Evolutionary bet-hedging in the real world: empirical evidence and challenges revealed by plants. Proceedings of the Royal Society B: Biological Sciences 277:3055–3064.

Chown, S. L., and K. J. Gaston. 1997. The species–body size distribution: energy, fitness and optimality. Functional Ecology 11:365–375.

Cooper, W. S., and R. H. Kaplan. 1982. Adaptive “coin-flipping”: a decision-theoretic examination of natural selection for random individual variation. Journal of Theoretical Biology 94:135–151.

Corrales, J., C. Thornton, M. White, and K. L. Willett. 2014. Multigenerational effects of benzo[a]pyrene exposure on survival and developmental deformities in zebrafish larvae. Aquatic Toxicology 148:16–26.

Crean, A. J., M. I. Adler, and R. Bonduriansky. 2016. Seminal fluid and mate choice: new predictions. Trends in Ecology & Evolution 31:253–255.

Cunningham, E. J. A., and A. F. Russell. 2000. Egg investment is influenced by male attractiveness in the mallard. Nature 404:74–77.

Dahl, E., G. Orizaola, A. G. Nicieza, and A. Laurila. 2012. Time constraints and flexibility of growth strategies: geographic variation in catch-up growth responses in amphibian larvae. Journal of Animal Ecology 81:1233–1243.

Danchin, É., A. Charmantier, F. A. Champagne, A. Mesoudi, B. Pujol, and S. Blanchet. 2011. Beyond DNA: integrating inclusive inheritance into an extended theory of evolution. Nature Reviews Genetics 12:475–486.

Dewitt, T. J., A. Sih, and D. S. Wilson. 1998. Costs and limits of phenotypic plasticity. Trends in Ecology & Evolution 13:77–81.

Dey, S., S. R. Proulx, and H. Teotónio. 2016. Adaptation to temporally fluctuating environments by the evolution of maternal effects. Public Library ofSciences Biology 14(2):e1002388.

Easterling, D. R. 2000. Climate extremes: observations, modeling, and impacts. Science 289:2068–2074.

Felling, R. J., and H. Song. 2015. Epigenetic mechanisms of neuroplascitic and the implications for stroke recovery. Experimental Neurology 268:37–45.

Fisher, M. O., R. G. Nager, and P. Monaghan. 2006. Compensatory growth impairs adult cognitive performance. Public Library of Sciences Biology 4(8):e251.

Frézal, L., and M.-A. Félix. 2015. *C. elegans* outside the Petri dish. eLife 4:e05849.

Fridmann-Sirkis, Y., S. Stern, M. Elgart, M. Galili, A. Zeisel, N. Shental, and Y. Soen. 2014. Delayed development induced by toxicity to the host can be inherited by a bacterial-dependent, transgenerational effect. Frontiers in Genetics 5:27

Gasparini, J., K. D. McCoy, C. Haussy, T. Tveraa, and T. Boulinier. 2001. Induced maternal response to the Lyme disease spirochaete *Borrelia burgdorferi sensu lato* in a colonial seabird, the kittiwake *Rissa tridactyla*. Proceedings of the RoyalSociety B: Biological Sciences. 268:647–650.

Gavrilets, S., and S. M. Scheiner. 1993. The genetics of phenotypic plasticity. V. Evolution of reaction norm shape. Journal of Evolutionary Biology 6:31–48.

Ghalambor, C. K., K. L. Hoke, E. W. Ruell, E. K. Fischer, D. N. Reznick, and K. A. Hughes. 2015. Non-adaptive plasticity potentiates rapid adaptive evolution of gene expression in nature. Nature 525:372–375.

Ghalambor, C. K., J. K. McKay, S. P. Carroll, and D. N. Reznick. 2007. Adaptive versus non-adaptive phenotypic plasticity and the potential for contemporary adaptation in new environments. Functional Ecology 21:394–407.

Goddard, M. E., and E. Whitelaw. 2014. The use of epigenetic phenomena for the improvement of sheep and cattle. Frontiers in Genetics 5.(247). doi:10.3389/fgene.2014.00247

Grant, M. C. 1991. Relationships between egg size, chick size at hatching, and chick survival in the Whimbrel Numenius phaeopus. Ibis 133:127–133.

Groothuis, T. G. G., and H. Schwabl. 2008. Hormone-mediated maternal effects in birds: mechanisms matter but what do we know of them? Philosophical Transactions of the Royal Society of London. B: Biological Sciences. 363:1647–1661.

Hoyle, R. B., and T. H. G. Ezard. 2012. The benefits of maternal effects in novel and in stable environments. Journal of the Royal Society Interface 9:2403–2413.

Inouye, D. W., B. Barr, K. B. Armitage, and B. D. Inouye. 2000. Climate change is affecting altitudinal migrants and hibernating species. Proceedings of Natlional Acadademy of Sciences of the United States of America. 97:1630–1633.

Jablonka, E., and G. Raz. 2009. Transgenerational epigenetic inheritance: prevalence, mechanisms, and implications for the study of heredity and evolution. The Quarterly Review of Biology 84:131–176.

King, J. G., and J. D. Hadfield. 2019. The evolution of phenotypic plasticity when environments fluctuate in time and space. Evolution Letters 3:15–27.

Kingsolver, J. G., and L. B. Buckley. 2017. Quantifying thermal extremes and biological variation to predict evolutionary responses to changing climate. Philosophical Transactions of the Royal Society of Lond B: Biological Sciences 372:1471-2970.

Kirkpatrick, M., and R. Lande. 1989. The evolution of maternal characters. Evolution 43:485–503.

Klironomos, F. D., J. Berg, and S. Collins. 2013. How epigenetic mutations can affect genetic evolution: Model and mechanism. BioEssays 35:571–578.

Knopik, V. S., M. A. Maccani, S. Francazio, and J. E. McGeary. 2012. The epigenetics of maternal cigarette smoking during pregnancy and effects on child development. Dev. Psychopathol. 24:1377–1390.

Kölliker, M. 2005. Ontogeny in the family. Behav. Genet. 35:7–18.

Kuijper, B., and R. B. Hoyle. 2015. When to rely on maternal effects and when on phenotypic plasticity? Evolution 69:950–968.

Lachmann, M., and E. Jablonka. 1996. The inheritance of phenotypes: an adaptation to fluctuating environments. Journal of theoretical biology 181:1–9.

Lande, R. 2009. Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation. Journal of Evolutionary Biology 22:1435–1446.

Leimar, O., and J. M. McNamara. 2015. The evolution of transgenerational integration of information in heterogeneous environments. The American Naturalist 185:55–69.

Lind, M. I., M. K. Zwoinska, J. Andersson, H. Carlsson, T. Krieg, T. Larva, and A. A. Maklakov. 2020. Environmental variation mediates the evolution of anticipatory parental effects. Evolution Letters 606103.

Lind, M. I., M. K. Zwoinska, J. Andersson, H. Carlsson, T. Krieg, T. Larva, and A. A. Maklakov. 2019. Environmental variation mediates the evolution of anticipatory parental effects. bioRxiv, doi: 10.1101/606103.

López-Arredondo, D., S. I. González-Morales, E. Bello-Bello, G. Alejo-Jacuinde, and L. Herrera. 2015. Engineering food crops to grow in harsh environments. 2(4):651. doi:10.12688/f1000research.6538.1

Marshall, D., and T. Uller. 2007. When is a maternal effect adaptive? Oikos 116:1957–1963.

Merilä, J., and A. P. Hendry. 2014. Climate change, adaptation, and phenotypic plasticity: the problem and the evidence. Evolutionary Applications 7:1–14.

Merilä, J., B. C. Sheldon, and L. E. B. Kruuk. 2001. Explaining stasis: Microevolutionary studies in natural populations. Pp. 199–222 *in* A. P. Hendry and M. T. Kinnison, eds. Microevolution Rate, Pattern, Process. Springer Netherlands, Dordrecht.

Metcalfe, N. B., and P. Monaghan. 2001. Compensation for a bad start: grow now, pay later? Trends in Ecology & Evolution 16:254–260.

Mock, D. W., and G. A. Parker. 1997. The evolution of sibling rivalry. Oxford University Press, USA.

Moran, N. A. 1992. The evolutionary maintenance of alternative phenotypes. The American Naturalist 139:971–989.

Morgan, I. J., and N. B. Metcalfe. 2001. Deferred costs of compensatory growth after autumnal food shortage in juvenile salmon. Proceedings of the Royal Society of London B: Biological Sciences 268:295–301.

Olofsson, H., J. Ripa, and N. Jonzén. 2009. Bet-hedging as an evolutionary game: the trade-off between egg size and number. Proceedings of the Royal Society of London B: Biological Sciences 276:2963–2969.

Ozanne, S. E., and C. N. Hales. 2004. Catch-up growth and obesity in male mice. Nature 427:411–412.

Palmer, T. N., and J. Räisänen. 2002. Quantifying the risk of extreme seasonal precipitation events in a changing climate. Nature 415:512–514.

Parker, T. H. 2002. Maternal condition, reproductive investment, and offspring sex ratio in captive red junglefowl (*Gallus gallus*). The Auk 119:840–845.

Parsons, J. 1970. Relationship between egg size and post-hatching chick mortality in the herring gull (*Larus argentatus*). Nature 228:1221–1222.

Pembrey, M., R. Saffery, L. O. Bygren, Network in Epigenetic Epidemiology, and Network in Epigenetic Epidemiology. 2014. Human transgenerational responses to early-life experience: potential impact on development, health and biomedical research. Journal of Medeical Genetetics. 51:563–572.

Perez, M. F., and B. Lehner. 2019. Intergenerational and transgenerational epigenetic inheritance in animals. Nature Cell Biology 21:143–151.

Philippi, T., and J. Seger. 1989. Hedging one's evolutionary bets, revisited. Trends in Ecology & Evolution 4:41–44.

Pigliucci, M. 2005. Evolution of phenotypic plasticity: where are we going now? Trends in Ecology & Evolution 20:481–486.

Pooley, E. L., M. W. Kennedy, and R. G. Nager. 2014. Maternal inbreeding reduces parental care in the zebra finch, *Taeniopygia guttata*. Animal Behaviour 97:153–163.

Price, T. D., A. Qvarnström, and D. E. Irwin. 2003. The role of phenotypic plasticity in driving genetic evolution. Proceedings of the Royal Society of London B: Biological Sciences 270:1433–1440.

Rando, O. J., and K. J. Verstrepen. 2007. Timescales of genetic and epigenetic inheritance. Cell 128:655–668.

Rechavi, O., L. Houri-Ze'evi, S. Anava, W. S. Sho Goh, S. Y. Kerk, G. J. Hannon, and O. Hobert. 2014. Starvation-induced transgenerational inheritance of small RNAs in *C. elegans.* Cell 158:277–287.

Reed, T. E., R. S. Waples, D. E. Schindler, J. J. Hard, and M. T. Kinnison. 2010. Phenotypic plasticity and population viability: the importance of environmental predictability. Proceedings of the Royal Society of London B: Biological Scienes 277:3391–3400.

Richards, E. J. 2006. Inherited epigenetic variation--revisiting soft inheritance. Nature Reviews Genetics. 7:395–401.

Rocha, F., H. F. Medeiros, and L. B. Klaczko. 2009. The reaction norm for abdominal pigmentation and its curve in *Drosophila mediopunctata* depend on the mean phenotypic value. Evolution 63:280–287.

Rosa, E., Z. Sekajova, F. Spagopoulou, P. I. Zervakis, and M. Lind. 2020. Compensatory growth plasticity in *C. elegans* is regulated by thermosensitive TRP channel and increases fitness. Manuscript.

Royle, N. J., J. Lindström, and N. B. Metcalfe. 2006. Effect of growth compensation on subsequent physical fitness in green swordtails *Xiphophorus helleri*. Biology Letters 2:39–42.

Ruden, D. M., M. D. Garfinkel, V. E. Sollars, and X. Lu. 2003. Waddington's widget: Hsp90 and the inheritance of acquired characters. Seminars in Cell & Developmental Biology 14:301–310.

Rutherford, S. L., and S. Lindquist. 1998. Hsp90 as a capacitor for morphological evolution. Nature 396:336–342.

Schmid, M., and F. Guillaume. 2017. The role of phenotypic plasticity on population differentiation. Heredity 119:214–225.

Schrey, A. W., and C. L. Richards. 2012. Within-genotype epigenetic variation enables broad niche width in a flower living yeast. Molecular Ecology 21:2559–2561.

Seisenberger, S., J. R. Peat, T. A. Hore, F. Santos, W. Dean, and W. Reik. 2013. Reprogramming DNA methylation in the mammalian life cycle: building and breaking epigenetic barriers. Philosophical Transactions of the Royal Society of London B: Biological Sciences. 368:20110330.

Shea, N., I. Pen, and T. Uller. 2011. Three epigenetic information channels and their different roles in evolution: Epigenetic mechanisms and evolution. Journal of Evolutionary Biology 24:1178–1187.

Simons, A. M. 2011. Modes of response to environmental change and the elusive empirical evidence for bet hedging. Proceedings of the Royal Society B: Biological Sciences 278:1601–1609.

Simpson, G. G. 1953. The Baldwin Effect. Evolution 7:110–117.

Skinner, M. K. 2008. What is an epigenetic transgenerational phenotype? F3 or F2. Reprod Toxicol 25:2–6.

Slatkin, M. 1974. Hedging one's evolutionary bets. Nature 250:704–705.

Stibor, H. 1992. Predator induced life-history shifts in a freshwater cladoceran. Oecologia 92:162–165.

Stibor, H., and J. Lüning. 1994. Predator-induced phenotypic variation in the pattern of growth and reproduction in *Daphnia hyalina* (Crustacea: Cladocera). Functional Ecology 8:97–101

Tollrian, R. 1993. Neckteeth formation in *Daphnia pulex* as an example of continuous phenotypic plasticity: morphological effects of *Chaoborus kairomone* concentration and their quantification. Journal of Plankton Research 15:1309–1318.

Tollrian, R. 1990. Predator-induced helmet formation in *Daphnia cucullata* (Sars). Archiv für Hydrobiologie 119:191–196.

Uller, T. 2008. Developmental plasticity and the evolution of parental effects. Trends in Ecology & Evolution 23:432–438.

Uller, T., S. English, and I. Pen. 2015. When is incomplete epigenetic resetting in germ cells favoured by natural selection? Proceedings of the Royal Society B: Biological Sciences 282:20150682.

Uller, T., S. Nakagawa, and S. English. 2013. Weak evidence for anticipatory parental effects in plants and animals. Journal of Evolutionary Biology 26:2161–2170.

Uller, T., and I. Pen. 2011. A theoretical model of the evolution of maternal effects under parent–offspring conflict. Evolution 65:2075–2084.

Vaiserman, A. M. 2008. Epigenetic engineering and its possible role in anti-aging Intervention. Rejuvenation Research 11:39–42.

Van Aalst, M. K. 2006. The impacts of climate change on the risk of natural disasters: The Impacts of climate change on the risk of natural disasters. Disasters 30:5–18.

Vastenhouw, N. L., K. Brunschwig, K. L. Okihara, F. Müller, M. Tijsterman, and R. H. A. Plasterk. 2006. Long-term gene silencing by RNAi. Nature 442:882–882.

Wells, J. C. K. 2003. The thrifty phenotype hypothesis: thrifty offspring or thrifty mother? Journal of Theoretical Biology 221:143–161.

West-Eberhard, M. J. 2003. Developmental plasticity and evolution. Oxford University Press.

Williams, T. D. 1994. Intraspecific variation in egg size and egg composition in birds: effects on offspring fitness. Biological Reviews 69:35–59.

Wilson, P. N., and D. F. Osbourn. 1960. Compensatory growth after undernutrition in mammals and birds. Biological Reviews 35:324–361.

Wolf, J. B., and M. J. Wade. 2009. What are maternal effects (and what are they not)? Philosophical Transactions of the Royal Society of London B: Biological Sciences 364:1107–1115.

Wolff, G. L. 1978. Influence of maternal phenotype on metabolic differentiation of agouti locus mutants in the mouse. Genetics 88:529–539.

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