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Discussion Paper: Epigenetics and Phenotypic Plasticity

Epigenetics may drive phenotypic plasticity and facilitate adaptive evolution. These central problems rooted in basic biological research help us predict the future magnitude and variability of responses that individuals and populations have to environmental stressors. While Ghalambor et al. (2015) tested a fundamental assumption of phenotypic plasticity and adaptive evolution, Ryu et al. (2018) and Liew et al. (2018) added an epigenetic component to their long-term studies to better understand the mechanism behind thermal acclimation under climate change in their native ranges.

One assumption that Ryu et al. (2018) and Liew et al. (2018) operate under is that epigenetic mechanisms facilitate adaptive phenotypic plasticity and will increase the performance and fitness of organisms reared under future ocean conditions. Ghalambor et al. (2015) challenges this assumption by ultimately showing that plasticity in gene expression is generally in the opposite direction of adaptive evolution. Rather than genetic assimilation, plasticity that is non-adaptive allows for rapid adaptive evolution via strong selection against it. Ghalambor et al. (2015) demonstrate that plasticity was non-adaptive by showing that differentially expressed transcripts in descendent translocated populations were evolving in the same direction as native populations in the translocation environment and that for those transcripts, plasticity was reduced in descendent introduction populations. However, Ghalambor et al. (2015) was not able to provide an ecological justification for why expressing non-adaptive plasticity against the optimum would be advantageous for an organism, making it hard to come up with a reason why this would occur as a mechanism for directional selection. In addition, adaptive plasticity could lead to disruptive selection, which would also cause descendent populations to lowered plasticity.

Ghalambor et al. (2015)’s results provide an explanation to Ryu et al. (2018) and Liew et al. (2018) results who both were not able to definitively identify epigenetic mechanisms that underlying phenotypic responses and differential transcriptomic expression they observed in response to changes in temperature and pH (respectively). If we follow the logic in Ghalambor et al. (2015), this may have occurred because they were trying to find relationships between adaptive transcriptomic and epigenetic responses rather than identifying the direction of the response towards or away from an optimum, which would help reveal non-adaptive plastic responses that might be driving acclimation instead. Although it is also important to take note that vertebrates and invertebrates have different epigenetic mechanisms and cannot be directly compared.

Ryu et al. (2018) found an association between methylation and transgenerational acclimation to climate change, but a weak correlation between methylation and gene expression. They attribute this to an additional epigenetic layer (epigenetic control) that does not directly translate to thermal acclimation and show a relationship between differentially methylated regions and metabolic reprogramming (e.g., angiogenesis, oxygen consumption, and insulin responses). In order to test non-adaptive versus adaptive epigenetic control like Ghalambor et al. (2015) did, Ryu et al. (2018) would have had to see a change in plasticity from the control groups and temperature treatment groups in the same direction as fish that were reared in those treatment groups for a longer amount of time.

Similarly, Liew et al. (2018) found fewer than expected genes related to biomineralization in response to more acidic pH conditions and found instead differentially methylated rates for processes related to general growth and stress responses. This result was in contrast to their physical measurements of coral skeleton structure, which indicated that under lower pH, calyxes were stronger and the skeleton was more porous. The authors were unable to link the physical change they were observing with the differentially expressed transcripts or methylated positions that came from sequencing those coral individuals. In the end, they conclude that epigenetic mechanisms behindcoral species were reducing spurious transcription and noise and served as a cross-talk between the transcriptional machinery and histone modifications. One reason that I thought of to explain why there weren’t more epigenetic/genomic signals of biomineralization within stressed out corals was because maybe the corals’ algal symbionts played a larger role than previously expected in skeletal morphology. Perhaps comparing the epigenome of a coral with its algal community intact with the ones in this study (which had its algal symbionts removed) could show how symbioses could facilitate epigenetic control and plasticity. It is also possible that the complicated epigenetic mechanisms of invertebrates cause there to be so little empirical understanding of how they actually operate on a whole-genome scale.

In conclusion, while Ghalambor et al. (2015) challenges us to think about plasticity and epigenetics in the opposite direction that Ryu et al. (2018) and Liew et al. (2018) did, the latter two papers provided a more ecological/applied justification for their results, making them more contextualized and realistic. The major challenge moving forward in epigenetics research will be attempting to quantify the magnitude and direction of epigenetic control rather than simply identifying the associations between the epigenome and transcriptome.