**Week 7 – Group summaries**

**Summary:** Groups discussed papers about expansion load during range expansion (Gilbert et al. 2017, American Naturalist) and phenotype-environment mismatches in the sea (Marshall et al. 2009, Ecology Letters).

15 groups; 83+ participants

**1. What’s the relationships between phenotype-environment mismatches and genetic load?**

**Rutgers:**

* Hard to parse the connection. Most assumptions based on well-mixed populations.
* Might expect both maladaptation and expansion load to reduce fitness and limit range edges, but there is a lot of nuance.
* Expansion load could be reduced in environments under strong selection if alleles don’t surf to fixation. Selection would have to be stronger than what is in the Marshall paper.

**WSU:**

* No direct relationship
* There are other individuals of your type at the “new” environment, but what really matters is if one individual happens to get to this environment. As long as someone can get there, it’s still a random likelihood of high/low genetic load because of assumed connectivity.

**UMASS Amherst:**

* Including expansion load had smaller effect on expansion speed than difference in dispersal types.
* Discussed problem that models assume abundant center distribution which isn’t always the case

**LSU:**

* Genetic load is any deleterious allele that puts an individual at a fitness disadvantage (in specific environment or generally).
* 2 types of genetic load: expansion load where deleterious alleles get fixed from expanding population (allele surfing), and phenotypic environment mismatches, in which alleles are at a fitness disadvantage if they are in an environment the organism is not adapted to.

**NEU:**

* Can be interplay of pem and genetic load, they aren’t consistently linked in predictable fashion.
* Gilbert suggests increase in expansion load specifically could slow range expansion to point of overcoming local maladaptation, which could reduce instances of PEM. But loaded term was uniquely defined and not generally applicable to discussions of genetic load.
* Challenging to measure genetic load without strong contextual clarification. In absence of knowing true optimum, criteria for defining genetic load may be tricky.

**UC Davis:**

* Genetic load results from accumulation of maladaptive alleles in a population that reduces overall fitness relative to an optimum genotype. PEM occurs when individuals move from an environment in which they are well adapted to one in which they are poorly adapted. In these new environments, poorly adapted migrants have greater genetic load, resulting in high mortality rate.
* Genetic load relative to environment. Poorly adapted individual moves to another environment, previously maladaptive alleles may become useful phenotypic traits if genetic load was caused by outbreeding depression. Unlikely if genetic load is result of inbreeding depression.

**Virtual Group:**

* Complex. Marshall paper defines PEM very broadly, and Gilbert finds that maladaptation and load interact so each is less effective than expected when alone.
* Difference in outcome between a case where an allele is everywhere maladaptive vs allele that is adaptive in the core but maladaptive in the edge. Depend on demographics, but latter would correspond more closely with PEM.

**Uchic/UGA:**

* PEM could be selective agent shaping larval behavior, dispersal, and life history strategy. (produce behaviors that promote larval retention)
* Expansion load, local maladaptation, and potential interaction between two important for understanding impact of range expansion on genetic load.
* Challenging to test assumptions and hypotheses of model.

**USC:**

* During range expansion, PEM would slow expansion load and allow populations more time to adapt.
* Different environmental gradients would affect this though.
* With decreased environmental gradient, populations could expand further geographically and may lead to decreased PEM, but increase in genetic load.
* Multiple environmental gradients should also affect. Would there be more PEM causing slower range expansions?

**UCSB:**

* Genetic load can contribute to PEM at population levels, but maladaptive allele frequencies may not wholly determine PEM since plasticity may also contribute to PEM when transgenerational plasticity is mismatched with offspring experience.
* PEM linked to genetic variation can contribute to genetic load and other factors may contribute.
* Locally adapted population exhibiting mitochondrial traits that are maladaptive in a foreign environment (home to pop b) may increase genetic load to pop B following settlement and reproduction not only through PEM but potentially through mitochondrial incompatibility as well.
* Genetic load may increase after multiple generations- introgression of pop A into pop B could reduce offspring fitness via simultaneous PEM and hybrid breakdown.
* How do authors define PEM? Say that individuals that disperse suffer heightened mortality before reproduction relative to locally derived individuals, where others say it’s a reduction in fitness when an organism is specialized to one environment finds itself in another. Important because reduction in fitness may not necessitate mortality prior to reproduction. May simply be reduced reproductive success. Immigrant individuals capable of successful, even if reduced, reproduction will contribute to genetic load.
* Temporal env. Heterogeneity in which we might expect temporally variable environments to harbor genotypes associated with increased plasticity. Role of adaptive plasticity mentioned, but could allow propagules to survive settlement and eventual reproduction in foreign environments in spite of having suboptimal genotypes.

**Laval:**

* Genetic load is composed of many different loads. PEM cause one kind of load, but this is argued in Marshall that this shouldn’t happen in marine environment where consequences of PEM will appear before the reproduction of migrants. Suggest that PEM will cause reduction of connectivity in marine environments but no migration load
* Surfing alleles is another type of load, a drift load that appears when frequency of unfavorable or deleterious alleles increases due to stochasticity in small populations.
* Conclusion of Gilbert is that increased maladaptation due to environmental gradient from the center to the edges reduces expansion load and vice versa, increased expansion load will cause that locally adapted populations to have better chance of survival. Maladaptation due to PEM and expansion load at edges will appear negatively correlated.
* Genetic load and PEM relationship will be affected for all kinds of variables. Strength of connectivity between patches, sampling and time scales, kinds of dispersion, as well as environmental change and dynamics adding stress.
* PEM will cause migration load (=kind of genetic load) in terrestrial environments. In marine environments, PEM will reduce connectivity increasing the expansion load due to less migrants and drift load.

**Cornell:**

* Since PEM increase mortality in foreign migrant larval, gene flow is reduced. This could lessen genetic load by allowing local adaptation and inhibiting maladaptive alleles from continually being reintroduced by migrants (migrant load). Reduced gene flow leads to lower Ne, there may be less selection to purge linked deleterious variants.
* Important factor is migration at larval stage. Migration at adult stage could worsen genetic load by inhibiting local adaptation by swamping the migrant load.
* With MPAs, important to think of size and placement of MPAs. If MPAs are smaller than scale of environmental heterogeneity, may be ineffective due to PEM or may increase genetic load from migrant load. May be better to have many smaller MPAs for greater diversity of protected phenotypes.
* If goal is to reduce genetic load, MPA placement should be chosen according to environmental heterogeneity.

**MIT/WHOI:**

* PEM on broad scale would decrease genetic load as deleterious alleles are actively chosen against. Also produces bottleneck effect that could in the longer term allow for increases in the accumulation of deleterious alleles as drift occurs.
* Also consider innate patchiness of marine environment spatially and temporally – might encourage recruitment of organisms in other conditions would display a phenotype-environment mismatch. This would allow for an increase in allelic diversity, which might help act against genetic load accumulated from genetic drift.

**UQ:**

* Genetic load includes mutation load, expansion load, and migration load – latter two have geographic aspect.
* Expansion load could possibly contribute to PEM but main mismatch would most likely stem from maladaptation of migrants or colonizers.
* There are other sources of mismatch especially plastic phenotypes that match source of origin. Physiological debt from long or difficult larval phase could reduce fitness.

**MSC:**

* Equate PEM to post-settlement selection in marine systems and agree that migration load may be more prominent in terrestrial systems given the life history stage at which dispersal occurs.
* For long lived organisms that experience environmental change, end may be genetic load via a different process.

**2. How does dispersal scale and the scale of environmental heterogeneity contribute to genetic load?**

**Rutgers:**

* Marine species don’t have migration load or less if migrants aren’t able to establish and reproduce. But other components still apply.
* Components of genetic load still possible for marine species in the case where they are establishing new populations at the edge of their range (assuming they don’t go so far that there is no possibility of survival to reproduction).
* Higher load will impede success of high dispersers – if environment changes they will disperse further.
* Expansion load could be limiting the edge of a species’ range expansion, not dispersal, and slower expansions might allow enough time for selection to act enabling adaptation to the new environment.
* Depends on rate of migrant alleles from core, which could hinder local adaptation – would need to see how migration from core influences fitness at the edge.

**WSU:**

* Disparity in papers assuming linearity in environmental gradients (heterogeneity) and dispersal gradients – we know these factors aren’t linear, but models assume they are.
* Can we apply these concepts knowing this disconnect?
* Gibert argues increased slope of an assumed linear environmental gradient reduces genetic load. Means that selection takes a bigger role in the observed effects on the population on a range edge when you have steeper environmental gradient.
* Life history characteristics (broadcast), how can there be relationship with genetic load in adults if selection is happening in a completely different life stage with different selective pressures during larval dispersal?
* MPAs – should think about scale of dispersal and environments when designing ocean spatial management.
* Temporal considerations matter! Can’t just consider snapshots in time.

**UMASS Amherst:**

* Longer distance dispersal strategies may affect expansion load (not just expansion speed).
* Marine species relatively free of migration load due to mortality prior to reproduction.
* Marine species likely to suffer expansion load due to severe bottlenecking that occurs during sweepstakes and selection might outweigh expansion load in these taxa since they are able to persist despite the load.
* Many habitats follow this patchy pattern and apparent lack of connectivity across habitats might be the rule rather than the exception in aquatic environments. May compare with wind-dispersed terrestrial organisms.

**LSU:**

* Depends on organism and scale of dispersal relative to scale of environmental heterogeneity. Less genetic load if dispersal is large and environmental heterogeneity is small. If dispersal is small but env. Het. Is large, may be more genetic load.
* Steepness of environmental gradient slows range expansion, but more time for the core to adapt to the range edge and ameliorate the high genetic load.
* Assumes environmental heterogeneity in continuous landscape, not mosaic. In mosaic, if dispersal is short, high probability of staying in environment you’re adapted to.
* If you’re long range disperser, chance of getting to env. You’re adapted to is small.
* To link papers: local maladaptation and expansion may interact positively, each making the effects of the other more pronounced. Makes sense but model found opposite. They ameliorate each other, reducing genetic load.
* Could also be interpreted as PEM, all organisms are so maladapted at the range edge with such high genetic load that range edge populations quickly go extinct because they can be rescued by the core.
* Why do organisms disperse if PEM exists? Environments change through time, dispersal is not as random as we think, as way to increase genetic diversity?

**NEU:**

* Increased dispersal is associated with increased exposure to greater diversity of habitats, and migration load does impact local fitness of adaptive population, but evolutionary theory needed.
* Are orgs. Believed to be broadly dispersing more vagile or more plastic? Need to know fitness of genomes in different environments we won’t know the pressures that are driving them.
* Pressures could be represented in SNP data, but also indels and variation in genomic architecture.
* Discuss connection between genomic architecture and genetic load. Marine orgs have higher load than terrestrial as consequence of sweepstakes reproduction or increased germline mutation accumulation, possibly also due to PEM.
* Issue of MPAs – initial fecundity could be high but result in migration load. Load is context dependent, environmental heterogeneity can shift advantage/disadvantage of individual variants.
* What is feasibility in terms of planning MPAs? Systems aren’t static, heterogeneous environments shift with heterogeneous populations and it would be good to see more direct consideration of how managers can better account for this.

**UC Davis:**

* Environmental heterogeneity can be structured as continuous or discretely patchy. May affect genetic load differently depending on dispersal scale.
* When dispersal scales are large, cline will result in high genetic load because farther individuals disperse the more poorly adapted to their environment they are.
* In contrast, in patchy structure, long distance dispersal need not necessarily result in high genetic load bc far flung habitat patches are as likely to be suitable for dispersing individuals as sites near natal locations.
* Higher genetic load is more likely in a patchy structure where nearby sites are just as likely to be good habitats as they are to be poor habitat.
* Example is porcelain crab, which disperses across patchy environment of cobbles and mussel beds. Expect this species to have high genetic load as they are not thought to disperse far. No structure at Fst, maybe structure at Qst?

**Virtual Group:**

* Expect when scale of dispersal is larger than scale of heterogeneity, PEM (migrant load) will increase.

**USC:**

* If scale of dispersal is greater than scale of environmental heterogeneity, there will be increase in PEM which will slow down range expansions and decrease genetic load, as only those organisms with highest fitness in that environment are surviving to range edge.
* If dispersal is smaller than scale of environmental heterogeneity, genetic diversity will be maintained and there will be little or no genetic load.
* Need clarification of what environmental heterogeneity means.

**UCSB:**

* Increasing scale of dispersion increases the probability that an individual will encounter an environment in which its phenotype is mismatched, which could reduce genetic load by propagules onto new populations.
* This depends on environmental heterogeneity, because long distance dispersers in homogenous environments may contribute to genetic load compared to short distance dispersers settling somewhere across a more heterogenous environment.

**Laval:**

* When scale of dispersal is high, probabilistically fewer individuals will arrive (in and species with feeding larvae those who arrive will be disadvantageous) therefore decreasing likelihood of carrying deleterious alleles that can act as mutation load.
* Due to surfing alleles, deleterious mutations that arrive to range edge will increase frequency.
* Increased env. Heterogeneity will increase maladaptation due to PEM, maladaptation reduces the pace of expansion producing a reduction in expansion load and a number of migrants.
* In marine environments, the presence of environmental heterogeneity will reduce connectivity (due to PEM) could mean that expansion load will increase in this case too, because in small populations or edge populations drift load (surfing) will increase.

**Cornell:**

* Relationship of dispersal scale to scale of environmental heterogeneity is more important than the individual scales.
* Greater environmental heterogeneity, the lower we expect genetic load and expansion load, but less connected populations become.
* If dispersal scale is less than environmental heterogeneity we don’t expect migrant load. Clear that life history of species is important for management and evolutionary consequences of genetic load.

**MIT/WHOI:**

* With larger scale of dispersal, increased likelihood of mismatch between the arriving organisms and environment which would potentially contribute to a bottlenecking of survivors leading to increased genetic load.
* Takes broad scale view of environmental heterogeneity. On smaller scales, more likely that organism would encounter environment that matches its phenotype thus increasing diversity and decreasing genetic load.
* Then important to consider how fine-tuned the organisms have to be in order to for them to experience significant die-offs to result in a bottle neck.
* If dispersal is greater than scale of environmental heterogeneity, would result in decreased connectivity in ocean which favors local adaptation being predominant. Also favors increased genetic load as genetic drift occurs.

**UQ:**

* If organisms dispersal scale includes heterogeneous environments, then there will be migration load, unless phenotypic plasticity allows environmental matching post-settlement.
* Interesting situation would occur where environment changes rapidly (steep gradient) and therefore gene flow may be reduced due to post settlement mortality.

**MSC:**

* Environmental heterogeneity is primarily considered a gradient. But can also be patchiness. Patchiness could affect interaction of maladaptation and expansion load, as well as persistence of locally adapted populations.
* Non overlapping generations may affect predictions. How common are species with non-overlapping generations? May alter predictions especially for long lived species if individuals survive initial post-settlement selection and are more likely to reproduce.
* Interesting that gibert model included selfing, but what affect would this have on species that don’t self.
* Suggestion to follow Qst and Fst of cohort through life history, but wonder about feasibility. Anyone done this?