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Commentary

Evolutionary Change in Meristematic cell Lineages *via* Intra Organismal Selection

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DESCRIPTION

Somatic mutations are the only source of genetic variation in clonally reproducing plants in the absence of sexual recombination. They examine the likelihood that these somatic mutations in the shoot apical meristem will be stabilized in succeeding generations of meristems. A meristem cell dynamics model for the stratified shoot apical meristem is provided. For a two and three celled shoot apical meristem, the fate of a single mutant initial is investigated. The number of apical initials, the intervals between selection cycles, the total number of selection cycles, and the cell viability of the mutant genotype make up the model's major parameters.

The chimeric state disappears when the number of selection cycles and mitotic divisions each cycle rises, and the likelihood of mutation fixation gets closer to an asymptote. The value of this fixation asymptote is mostly determined by cell viability, but the time required to attain it is primarily determined by the total number of initials and mitotic divisions. The chimeric condition may present a chance for harmful mutations to be eradicated through intra organismal selection or random drift, in contrast to how Muller's Ratchet (MR) is thought to function in plants.

They conclude that intra organismal selection has the capacity to impose an evolutionary shift through a meristematic cell lineage alone, in addition to being a significant factor for the eradication of harmful mutations. Meristems are undifferentiated cell lineages that are specific to vascular plants and are derived through mitosis. The activity in these cell lineages causes initial growth, which develops into the basic plant shape as well as all differentiated plant structures. Meristems also give rise to vegetative produced progeny, known as ramets, in plant species with facultative or obligate clonal reproduction, connecting lineages of mitotically derived descendant individuals.

When the mutant cell is fit than the wild type, there is a chance that it will displace the ancestral cells, either randomly through drift or, more frequently, through intra organismal selection. Although many authors have highlighted the potential significance of intra organismal selection, it has primarily been viewed as a method of removing harmful somatic mutations. The situation in which a cell lineage with a beneficial mutation undergoes fixation by intra organismal selection has received little consideration.

Fundamentally interesting is the replacement of the wild type by a mutant in a mitotic cell lineage because it may be seen as an example of evolution: a descendent individual is created with a new (and homogeneous) genotype. The purpose of this work is to examine the circumstances in which mutant cell lineages may replace ancestral genotypes within ramet lineages and the implications of this for current beliefs about vegetative propagating plants.

They demonstrated that intra organismal selection may have a significant impact on the fate of somatic mutations by simulating the long-term retention of distinct categories of somatic mutations for various types of shoot-apical meristems. One of their findings was that many apical meristem groups in higher plants struggle to lose harmful somatic mutations, and some specifically, stratified meristems actually encourage the long-term accumulation of mutations. In order to account for ramet development in vegetative propagated plants, they modify their model of an un-stratified shoot apical meristem.

Despite the fact that their model only takes a single mutation into account and does not account for the accumulation of recurrent mutations, it is nevertheless. It is improbable that more than one mutation will accumulate in the shoot apex before the meristem is homogenized, either by fixing the mutant or returning to the wild type condition, at low mutation rates (per cell generation). Their findings demonstrated that, under the assumption that a high mutation load entails an increased probability of population extinction, even weak selection among cell lineages, slightly deleterious mutations, and acts strongly to reduce the mutation load, decreasing both the frequency of deleterious mutants and the observable mutation rate.

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