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Opinion piece

Is more better? Polyploidy and parasite resistance

Ploidy-level variation is common and can drastically affect organismal fitness. We focus on the potential consequences of this variation for parasite resistance. First, we elucidate connections between ploidy variation and key factors determining resistance, including allelic diversity, gene expression and physiological condition. We then argue that systems featuring both natural and artificially manipulated ploidy variation should be used to evaluate whether ploidy level influences host–parasite interactions.

Keywords: polyploidy; host–parasite interactions; allelic diversity; gene expression; host condition

1. INTRODUCTION

Polyploidization[1] has generated variation in ploidy level within and across species [1,2], and new examples are continually being discovered, particularly in animals [3]. Intraspecific ploidy-level variation is associated with a suite of connections between polyploidy and biological phenomena [4,5], and has the potential to influence fitness-related traits [6,7]. We discuss whether ploidy variation could mediate resistance to a ubiquitous enemy—parasites (defined here as an organism that harms its host). Although current theory suggests ploidy level can profoundly influence infection dynamics and host–parasite evolution [8,9], data are scarce [10,11].

2. PLOIDY AND IMMUNE FUNCTION

Polyploidy (i.e. autopolyploidy) could directly influence immune response to a parasite attack in at least two ways. Firstly, the addition of a new genome may increase allelic diversity. Higher allelic diversity at immune genes could help hosts recognize a greater diversity of parasites [12]. Secondly, if the additional genome copies are expressed, then polyploids may generate higher amounts of gene products related to immune function.

(a) *Allelic diversity and immune function*

The high allelic diversity of immune genes is partly a consequence of parasite-mediated selection for rare genotypes and/or novel immune functions [13]. Parasite-mediated selection may generate such diversity through mechanisms such as heterozygote advantage [14] and negative frequency-dependence [13]. Heterozygote advantage could be influenced by ploidy level because the extra alleles present in polyploids may increase the probability of heterozygosity for an individual at a given locus. The presence of an extra genome could also increase the likelihood that an individual possesses a rare genotype at resistance loci; this would be advantageous if parasite-mediated negative frequency-dependent selection favours rare genotypes.

Studies from natural populations have documented connections between parasite-mediated selection and the maintenance of allelic variation at genes associated with immune function [12,15]. Genetic polymorphism can play an important role in generating variation in recognition molecules [16], and diversity is important for disease resistance in host individuals and populations [17,18]. Furthermore, measures of genome-wide genetic variation (e.g. microsatellite heterozygosity [19,20]) are often correlated with individual immune function and susceptibility [20–22]. Increased allelic diversity associated with polyploidy may thus enhance host ability to detect and fight off a variety of parasites. However, polyploidy might be irrelevant in generating functional diversity if increased allelic variation is dwarfed by variation generated at the protein level via somatic diversification of recognition molecules [23,24].

(b) *Expression levels and immune function*

Protein [25] and RNA content [26] often increase with ploidy level [27], suggesting that extra chromosome sets can increase gene expression. However, certain loci or even whole genomes (generally, in allopolyploids) are up- or downregulated (or even silenced) as ploidy increases [28]. This among-locus variation may be system specific [27,28], making it difficult to predict how polyploidy will affect particular genes.

Although the relationship between expression levels and immune function is not well characterized [29,30], some suggest that the two could be positively related [29]. Data consistent with this possibility come from comparisons of immune function in male and female mammals, which is typically higher in females. One explanation for this sexual dimorphism in immune function could be differences in sex chromosome number [29], although higher investment [31] by females cannot be excluded. Although females typically express only one of their X-chromosomes per cell (owing to inactivation), some X-chromosome genes (involved in immune function [29]) escape silencing. Given the difference in sex chromosome number, the dosage of these gene products may be higher in females [32].

3. THREE-WAY INTERACTION: PLOIDY, CONDITION AND RESISTANCE

Individuals suffering from environmental stressors are often more susceptible to infection [33,34]. This may be a consequence of weakened immune defences in hosts of poor condition [35], because immune functions are energetically costly to maintain and use [36]. Consequently, any effects of ploidy-level variation on host condition could indirectly influence parasite resistance.

Current knowledge regarding connections between body condition and ploidy level comes largely from studies that focused on reallocation to growth in artificially generated and sterile triploid fish and shellfish used for aquaculture [37–39]. While the applicability of these studies to natural, fertile autopolyploids is limited, they

[1]Polyploids can have either non-hybrid (autopolyploid) or hybrid (allopolyploid) origins. Because hybridization can influence phenotype and genotype, we focus on autopolyploids, unless stated otherwise.

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do suggest that ploidy level influences traits that can interact with condition. The few relevant studies from natural animal polyploids have also demonstrated that variation in body composition [26] and growth rate [40], among others, can be associated with ploidy level.

4. DIRECT EMPIRICAL CONNECTIONS

Studies directly addressing ploidy level and host immune function suggest that polyploidy may have no effect or be detrimental. In both the farmed Pacific oyster (*Crassostrea gigas*) [41] and field-collected New Zealand freshwater snails (*Potamopyrgus antipodarum*) [42], haemocyte concentration in the haemolymph of triploids is lower than in diploids. Similarly, nitroblue tetrazolium reaction and hypoferraemic response are reduced in triploid versus diploid goldfish (*Carassius auratus*) and salmon (*Salmo salar*), respectively [43,44]. Additionally, while the transcriptional responses of several immune-related genes to bacterial infection do not differ between diploid and triploid Chinook salmon (*Oncorhynchus tshawytscha*), some genes show reduced performance in triploids [45]. Polyploidy may thus negatively affect immune defence, or polyploid individuals may not need to mount a strong immune response.

Like many animal polyploids, these triploid fishes and molluscs are asexual, and so the effects of ploidy and reproductive mode may be confounded [1,46]. This problem can be circumvented by taking advantage of systems that feature both mating system and ploidy-level variation [47,48] and by comparing resistance in triploid and tetraploid asexuals.

5. CONCLUSION

Connections between ploidy and parasite resistance are certainly complex, and selection on resistance is not necessarily positively linear [49] (e.g. because of immune defence costs [36]). More is not inevitably better. In fact, higher ploidy may be harmful [50]. To determine if ploidy-level variation can affect disease spread and resistance evolution, we must first ask: Is increased ploidy associated with higher allelic diversity at resistance genes? Do organisms with higher ploidy levels have higher expression levels of immune genes? How is resistance affected by host conditions mediated by polyploidization?

An effective way to evaluate these connections between parasitism and ploidy will be focus on naturally existing, non-hybrid species or conspecifics that contain a mix of diploids and autopolyploids. These systems will also ideally be amenable to the artificial creation of neopolyploids [51], allowing the direct phenotypic effects of polyploidy and the long-term consequences of extra genomic copies to be decoupled.

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