2014

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Citation/Publisher Attribution
Salomaki, Eric D.; Lane, Christopher E. (2014). "Are all red algal parasites cut from the same cloth?" *Acta Societatis Botanicorum Poloniae*. 83(4), 369-375.
Available at: http://dx.doi.org/10.5586/asbp.2014.047

Acta Societatis Botanicorum Poloniae

INVITED REVIEW Acta Soc Bot Pol 83(4):369–375 DOI: 10.5586/asbp.2014.047
Received: 2014-11-21 Accepted: 2014-12-12 Published electronically: 2014-12-31

Are all red algal parasites cut from the same cloth?

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Abstract

Parasitism is a common life strategy throughout the eukaryotic tree of life. Many devastating human pathogens, including the causative agents of malaria and toxoplasmosis, have evolved from a photosynthetic ancestor. However, how an organism transitions from a photosynthetic to a parasitic life history strategy remains mostly unknown. This is largely because few systems present the opportunity to make meaningful comparisons between a parasite and a close free-living relative. Parasites have independently evolved dozens of times throughout the Florideophyceae (Rhodophyta), and often infect close relatives. The accepted evolutionary paradigm proposes that red algal parasites arise by first infecting a close relative and over time diversify and infect more distantly related species. This provides a natural evolutionary gradient of relationships between hosts and parasites that share a photosynthetic common ancestor. Elegant microscopic work in the late 20th century provided detailed insight into the infection cycle of red algal parasites and the cellular interactions between parasites and their hosts. Those studies led to the use of molecular work to further investigate the origins of the parasite organelles and reveal the evolutionary relationships between hosts and their parasites. Here we synthesize the research detailing the infection methods and cellular interactions between red algal parasites and their hosts. We offer an alternative hypothesis to the current dogma of red algal parasite evolution and propose that red algae can adopt a parasitic life strategy through multiple evolutionary pathways, including direct infection of distant relatives. Furthermore, we highlight potential directions for future research to further evaluate parasite evolution in red algae.

Keywords: Rhodophyta; parasite evolution; pit connections; Florideophyceae, organelles

Introduction

Parasitism has evolved innumerable times throughout the eukaryotic tree of life [1]. Some of the more virulent parasites have transitioned from a once photosynthetic ancestor, including the causative agents of malaria and related mammalian diseases [2,3]. Therefore, understanding the evolutionary trajectory between photosynthesis and abandoning autotrophy for a parasitic strategy, is of particular importance. Red algal parasites are uniquely valuable to study this path because they have independently evolved many times, providing literally dozens of discrete events to compare [4–7]. This system may provide novel insights into the evolution of parasitism, especially with regard to the early stages of transitioning from a photosynthetic past.

Red algal parasites exclusively infect other red algae, typically ones with which they share a recent common ancestor [5–8]. The relationship between host and parasite was first recognized using morphological similarities in the life-cycles of parasites and their hosts [9]. More recently, molecular data have confirmed this hypothesis [4–6,10]. Traditionally red algal parasites have been placed into two different groups, based on their phylogenetic relationships with their hosts [6]. Adelphoparasites (adelpho is Greek for "kin") are closely related to their host and often infect only one host, whereas alloparasites are more divergent from their host(s) [4,6]. Currently, adelphoparasites are believed to make up roughly 90% of all red algal parasites [6].

Among the Florideophyceae, parasites belonging to at least 66 different red algal genera have evolved independently over 100 times (Tab. 1) [11]. The accepted evolutionary paradigm proposes that adelphoparasitism is the initial state, followed by parasite diversification, which leads to the development of alloparasites (Fig. 1). These "older" parasites can infect more distantly related taxa, and make up roughly 10% of red algal parasites [5,7]. Due to their rarity, alloparasites are relatively unstudied, with the exceptions of *Choreocolax polysiphoniae* and *Harveyella mirabilis*.

The importance of red algal pit connections

One of the defining characteristics of the florideophycean red algae is the ability of cells to form connections with their adjacent cells [12]. There are two distinct forms of these "pit connections" formed by red algae. Primary pit connections

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Handling Editor: Andrzej Bodył

Tab. 1 Taxonomic summary of red algal parasites including number of described species and type of parasite by family. The shaded boxes are adelphoparasite numbers, highlighted to indicate their abundance, relative to alloparasites.

Order	Family	Type and number of parasites
Ceramiales	Ceramiaceae	Adelphoparasites = 1
		Alloparasites = 2
	Delesseriaceae	Adelphoparasites = 14
	Rhodomelaceae	Adelphoparasites = 37
		Alloparasites = 8
	Spyridiaceae	Adelphoparasites = 1
	Wrangeliaceae	Adelphoparasites = 1
Corallinales	Corallinaceae	Adelphoparasites = 2
		Alloparasites = 1
	Hapalidiaceae	Adelphoparasites = 5
Gigartinales	Cystocloniaceae	Adelphoparasites = 2
		Alloparasites = 1
	Kallymeniaceae	Adelphoparasites = 3
	Phyllophoraceae	Adelphoparasites = 1
	Solieriaceae	Adelphoparasites = 1
Gracilariales	Gracilariaceae	Adelphoparasites = 2
	Pterocladiophilaceae	Adelphoparasites = 1
		Alloparasites = 12
Halymeniales	Halymeniaceae	Adelphoparasites = 1
Palmariales	Palmariaceae	Adelphoparasites = 1
	Rhodophysemataceae	Adelphoparasites = 1
Plocamiales	Plocamiaceae	Adelphoparasites = 2
Rhodymeniales	Faucheaceae	Adelphoparasites = 2
	Rhodymeniaceae	Adelphoparasites = 2
Incertae sedis	Incertae sedis	Adelphoparasites = 1
		Alloparasites = 6

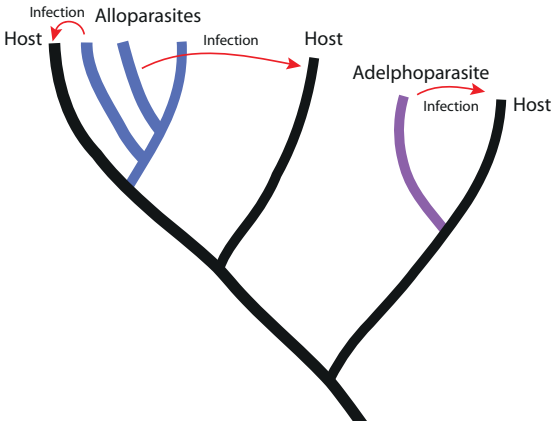

Fig. 1 Red algal parasites often evolve from a recent common ancestor with their host. These "adelphoparasites" (purple branch) are usually host-specific. However, parasites that have diversified into more than one species and/or infect distantly-related hosts, are known as "alloparasites" (blue branches).

arise between a mother and daughter cell during apical growth [13]. These connections result from a seemingly incomplete cell division where the septum begins to develop from the cell walls growing inward to separate the daughter nuclei [13]. However, cytokinesis is incomplete and the septum does not fuse, leaving an opening that connects the two cells [13]. A pit plug composed of a polysaccharide-protein complex then forms sealing the pit connection and separating the two cells [13,14]. Though pit connections result in an aperture that is not entirely sealed by a septum, the pit plug prevents the transfer of cellular contents and photosynthate between adjacent cells [15].

In addition to the primary pit connections, florideophytes also form secondary pit connections between adjacent non-daughter cells. These secondary pit connections are known to occur in a wide range of Florideophyceae and form between two genetically similar red algal cells [8,16]. Red algal parasite spores utilize secondary pit connections as a way to enter the cells of the host [8,17,18]. As evidence for the importance of secondary pit connections in parasitic infections, parasites are not known from red algal orders where secondary pit connections do not occur [19]. An advantage of this strategy is that the similarity between host and parasite at the genetic level allows parasite spores to simply deposit their organelles into the host cell and take over, spreading through primary or secondary connections [8,20]. The widespread existence of secondary pit connections among red algae is undoubtedly a primary factor in the promiscuous nature of parasitism as a life history strategy in the lineage.

Differences in adelphoparasite and alloparasite infection cycles

Spore germination and host infection

Rhodophytes lack flagella in all stages of their life cycle, making the initial stages of locating a host a passive process. Once a parasite spore lands upon a susceptible host, the parasite carpospore (2N) or tetraspore (1N) will germinate and undergo an initial cell division [21]. Adelphoparasite cells will divide between 1 and 3 more times before one of the cells forms a rhizoid that penetrates the surface of the host, growing into the wall of a host epidermal cell [21]. The tip of the rhizoid swells isolating a single parasite nucleus along with its organelles into a conjunctor cell which divides from the infection rhizoid [21,22]. This conjunctor cell then fuses, via a secondary pit connection, with the adjacent host cell (Fig. 2a). The contents of the conjunctor cell, which include the parasite nucleus and organelles, are deposited into the host cell, thus forming a heterokaryotic cell (containing both parasite and host nuclei) [8,17,21]. This connection between the parasite infection rhizoid and the transformed host cell is sealed by a pit plug that was formed previously during the initial fusion between the conjunctor cell and the parasite infection rhizoid [8,21].

The differences between adelpho- and alloparasites become evident at the initial infection of the host cell. In alloparasites, rather than going through a few cell divisions before penetrating the surface, the parasite spore attaches to a suitable host, penetrating and forming a hyphae-like

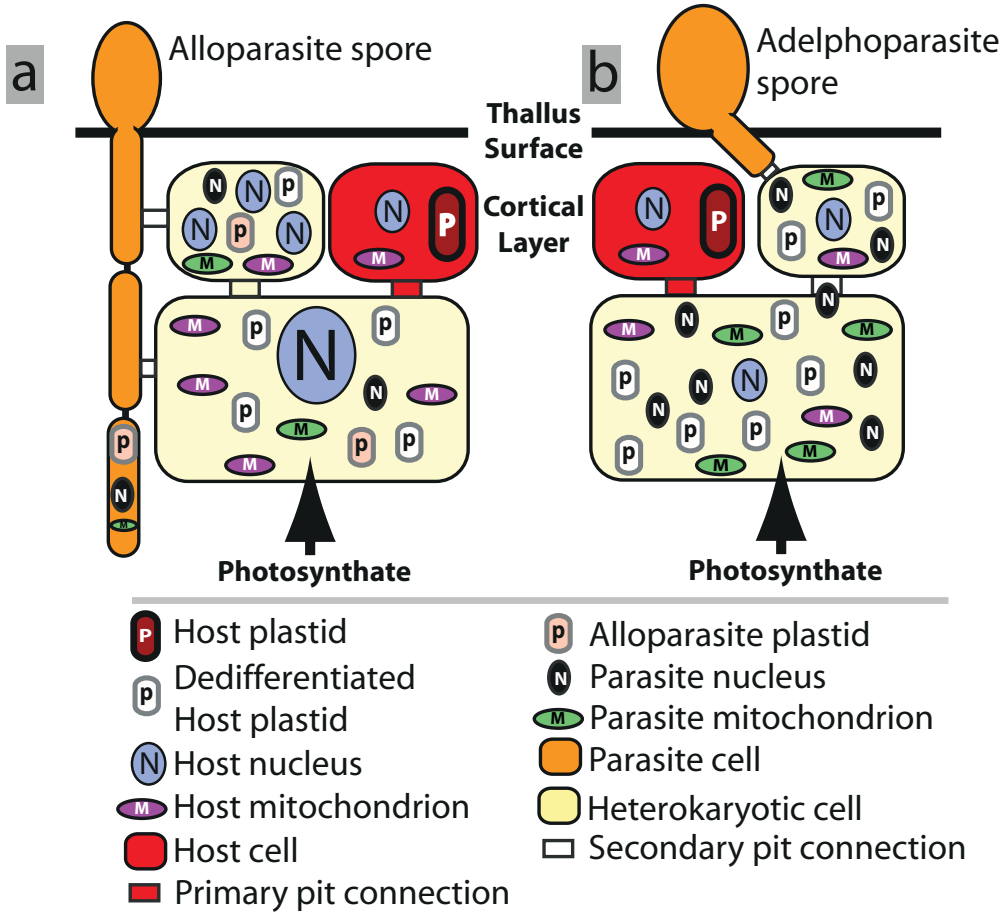

Fig. 2 Infection strategies of red algal alloparasites and adelphoparasites. **a** Alloparasites penetrate the host thallus and grow a network of filamentous cells into the host. Each cell is capable of fusing to a host cell via secondary pit connection and depositing its contents. Once inside the cell, the alloparasite nucleus does not divide, but causes the host nucleus to enlarge, or in the case of cortical cells, multiply. It is currently unclear whether the alloparasite plastid is derived from the host or parasite. **b** Adelphoparasite spores fuse with a cortical cell and inject their contents. These parasite nuclei can multiply within the host, however no nuclear DNA synthesis has been observed by the host after infection. Parasite nuclei and organelles spread via the host primary pit connections. The host organelles also multiply in response to infection.

network of multicellular filaments between the host cells [8,23,24]. These filaments enable the parasite to spread numerous cells deep into the host away from the initial site of infection (Fig. 2b). Each alloparasite cell in these filaments contains a single nuclei and can form a conjunctor cell and secondary pit connection through which the parasite deposits its cellular contents into the host, creating a heterokaryotic cell [8,23,24].

Inside the heterokaryon

After the host cell becomes heterokaryotic, both adelpho- and alloparasites take control of the host cellular machinery [8,21]. Almost immediately upon infection, the hosts' central vacuole tonoplast is lysed, allowing cytoplasm to spread throughout the space previously occupied by the vacuole [4,25]. Subsequently, the number of organelles including plastids, mitochondria, and ribosomes increases throughout the cytoplasm causing it to appear denser [8,21,23,25]. Along with the increase in cytoplasmic organelles comes an increase in cell size (hypertrophy), a process in which the cell can grow to 40 times its original size [8,22,26]. In addition to the increased organelles, either the parasite or host nuclei also increase in size and/or number [8,23,24].

Goff and Coleman used 4′,6-diamidino-2-phenylindole (DAPI) staining and microspectrofluorometry to examine the interactions between the alloparasite, *Choreocolax polysiphoniae* and its host *Vertebrata lanosa* [8]. Their study showed that infected *V. lanosa* central cells will become enlarged and the nuclei will undergo DNA synthesis but not nuclear division resulting in polyploid host nuclei [8]. Alternatively, in infected *V. lanosa* pericentral cells, the host nuclei will either increase in size, increase in number, or some combination of both [8]. An increase in the number of host nuclei is the most common response [8]. Host cells that are adjacent to infected cells and connected by pit connections will not show any cytological transformation [8,23].

Conversely, no DNA synthesis or nuclear division has been observed from the host nucleus after infection by the adelphoparasite *Janczewskia gardneri* [24]. Instead, the adelphoparasite nucleus rapidly undergoes DNA synthesis, generating numerous parasite nuclei inside a single host cell (Fig. 2) [24]. The adelphoparasite subsequently spreads to

additional host cells through the formation of conjunctor cells that can infect adjacent host cells [21]. Adelphoparasites can also form rhizoidal infection cells, which are multinucleate and contain large numbers of mitochondria, ribosomes, and dedifferentiated host-derived proplastids and can fuse with more distant host cells [21].

Alloparasites are capable of mitotic divisions to create the multicellular filaments that spread between host cells [23,24]. However, the alloparasite nucleus does not undergo DNA synthesis inside the host cell [8,23,24]. Therefore, parasite nuclei remain at a 1:1 ratio with the number of secondary pit connections between parasite and host cells [8,23,24]. Once inside a heterokaryon, the adelphoparasite continues spreading to adjacent host cells via pit connections, while utilizing the host to progress through its lifecycle and reproduce [21].

Formation of reproductive structures

As the adelphoparasite spreads throughout the host, a gall or "erumpent pustule" begins to form as host cells continually expand upon infection by the parasite. Eventually the adelphoparasite will start to form reproductive structures [21]. If the original infecting spore was haploid the parasite will form caprosporangia that can be fertilized by a spermatia from another parasite forming a diploid carposporophyte that will eventually release carpospores [21,27]. If the original infection was from a diploid carpospores, the parasite will undergo meiosis forming haploid tetraspores that will be released from the erumpent pustule [21,27].

An alloparasite does not spread through the host like the adelphoparasite. Rather than forming a gall from invaded cells, a host pericentral cell containing a parasite nuclei and many host nuclei will form a protuberance [8]. This protuberance will become isolated from the original host cell and undergo mitotic divisions, which produces the mature parasite pustule containing reproductive cells similar to that of adelphoparasites [8]. After the reproductive cells are released, the former host tissue that made the erumpent pustule becomes necrotic [8]. While the fate of future adelphoparasites lies in the released spores, alloparasites are able to continue their infection of the same host as parasite filaments continue to grow into uninfected areas [8].

Organelles

Studies of cellular organelles have yielded particularly interesting findings during investigations of red algal parasite biology. Early studies established the role of secondary pit connections between the parasite and host cells and demonstrated their role in transferring the parasite nucleus to the host cell [8,17,21]. However, it was unclear whether the parasite maintained its own mitochondrion and plastid or if it utilized the host organelles once the parasite nucleus was transferred into the host cell. Cytoplasmic organelles support many major metabolic pathways as well as play major roles in cellular energy and carbohydrate production. It has been well established that purifying selection is relaxed on parasite organellar genes that become unnecessary, leading to genome reduction in parasites as they increasingly rely on a host for energy and carbohydrates [28–32]. Therefore, it seems likely that some red algal parasite mitochondrion and plastid genes would be truncated or even lost over time. The origin and roles of mitochondria and plastids in the parasite-host interaction may reveal key information regarding red algal parasite biology and their ability to infect and control the host cellular machinery.

First using the alloparasite *Choreocolax polysiphoniae*, and later the adelphoparasites *Gracilariophila oryzoides* and *Gardneriella tuberifera*, researchers observed that in addition to the parasite nuclei, organelles are also transferred to the host cell via the conjunctor cell upon infection [21,24]. Once molecular tools became more widely available, Goff and Coleman utilized restriction fragment length polymorphisms (RFLPs) to investigate the origin of mitochondria in the adelphoparasites *Plocamiocolax pulvinata*, *Gracilariophila oryzoides*, and *Gardneriella tuberifera* and their respective hosts, *Plocamium cartilagineum*, *Gracilariopsis lemaneiformis*, and *Sarcodiotheca gaudichaudii* [22]. This work revealed that *P. pulvinata* and *G. oryzoides* maintain a genetically unique mitochondrion and that both the parasite and host mitochondria are present within the heterokaryotic cells [22]. The study was unable to conclusively demonstrate that the mitochondrion from *Gardneriella tuberifera* is unique from that of *Sarcodiotheca gaudichaudii*, due to the extremely close relationship between the two species [22].

The mitochondrion genomes of the adelphoparasites *Plocamiocolax pulvinata* and *Gracilariophila oryzoides*, as well as its host *Gracilariopsis andersonii*, were recently sequenced. These data paved the way for a new level of fine-scaled investigations of red algal parasites, elucidating details of the organellar genome architecture that was previously unattainable [33]. When comparing the mitochondrion genome sequences of the parasites with the free-living host, the *atp8* and *sdhC* genes from *G. oryzoides* were determined to be pseudogenes [33]. Furthermore, *atp8* was determined to be absent from *P. pulvinata*. However, the authors noted that according to sequenced cDNA libraries, the genes were still transcribed [33]. Recent second generation sequencing of additional samples of these species has revealed that the "missing" genes are all present and lack frameshift mutations (Salomaki and Lane, unpublished). These data suggest that purifying selection is maintained on red algal parasite mitochondria and that even though much of the parasite life cycle exists inside a host cell, red algal parasites still require their own mitochondrion for their survival.

While evident that parasites maintain their native mitochondrion, microscopy and molecular studies have demonstrated that red algal parasites do not maintain their own plastid [21,22]. Microscopy shows that the spores of adelphoparasites *Gracilariophila oryzoides*, and *Gardneriella tuberifera* contain proplastids lacking photosynthetic pigments, phycobilisomes, and thylakoids [21]. Once the parasite injects its nuclei and organelles into a host cell, the host plastids transform and their light harvesting pycobilisomes disappear from the thylakoids [21]. After rapid dedifferentiation, simple proplastids that are similar to the infecting parasite plastid bud off the host plastid [21]. As the parasite nuclei, mitochondrion, and host derived proplastids spread to adjacent cells through pit connections, plastids from the newly infected host cells also rapidly dedifferentiate

into proplastids [21]. Eventually cells emerge from the heterokaryotic host cell containing only parasite nuclei, parasite mitochondrion and a host-derived proplastid [21]. RFLP analysis was utilized to investigate whether this plastid was a genetically unique parasite plastid, or instead, the parasite was incorporating a host-derived proplastid [22]. This study revealed that adelphoparasites *Plocamiocolax pulvinata*, *Gracilariophila oryzoides*, and *Gardneriella tuberifera* and their hosts had identical banding patterns [22]. Subsequent DNA sequencing of the variable plastid *rbc*L-*rbc*S spacer region revealed that the plastid from both hosts and parasites were genetically identical, confirming that the parasite plastid is a dedifferentiated host plastid [22].

Interestingly, two studies suggest that the proplastid may be capable of differentiating again into a photosynthetic plastid. While investigating the photosynthetic rates and C^{14} transfer from *Polysiphonia* (=*Vertebrata*) *lanosa* to the alloparasite *Choreocolax polysiphoniae*, Callow et al. examined the alloparasite pustules after dissection from their host [34]. The authors note that many of the parasite pustules had a pinkish hue and incorporated radioactively labeled C^{14} into their thallus [34]. Furthermore, the carbon fixation rate increased over time (up to 66 hours) leading the authors to conclude that *C. polysiphoniae* is capable of photosynthesis on its own. However, the source of photosynthetic activity in dissected *C. polysiphoniae* pustules may be from host cells that have been incorporated into the pustule as observed in that study, and independently, by Kugrens and West, and Goff examining *Janczewskia gardneri* [21,26,34]. Additionally, the status of *J. gardneri* as a parasite or obligate epiphyte has been debated due to its pigmentation [24,35,36]. Most recently, it was noted that during the early stages of the interaction between *J. gardneri* and its host, *Laurencia spectabilis*, *J. gardneri* exists as colorless cells and "infects" host cells in the same manner as other adelphoparasites. As *J. gardneri* cells erupt from the host they remain colorless but the cells become pigmented once the adelphoparasite becomes reproductively mature [24]. Whether this pigmentation originates from host cells in the pustule matrix, or if the proplastid differentiates back into a photosynthetic plastid remains unknown.

Nutrient transfer

With the exception of a few adelphoparasites that gain pigmentation upon reproductive maturity, red algal parasites are not capable of photosynthesis on their own and must obtain carbohydrates and other nutrients from a host. After parasite infection, the host (now heterokaryotic) cell loses the ability to photosynthesize as a result of plastid dedifferentiation [21]. This leads to a differential gradient of carbon between the heterokaryotic cell and the adjacent normally functioning host cells [21,22,25]. To account for the loss of carbon fixation, uninfected host cells direct photosynthate to heterokaryon and parasite cells that they are connected to via pit connections [24,37].

The first studies investigating carbon transfer between a red algal host and its parasite found three products of photosynthesis (floridoside, isofloridoside, and manitol) were transferred from the host to its parasite via a concentration gradient [34,38]. Later five different sugar species were identified to be assimilated by the host *Rhodomela confervoides* and translocated to its parasite *Harveyella mirabilis* [39]. Investigations into carbon translocation in *H. mirabilis* demonstrated the localization of carbon, from being fixed by the photosynthetic host through its movement into the parasite cells and revealed that heterokaryon cells incorporated more C^{14} than neighboring uninfected host cells [37]. Furthermore, it was determined that starch was not distributed evenly throughout the parasite cells as might be expected, but instead was being directed preferentially to parasite reproductive cells [8,37]. Given the capabilities of parasites for obtaining carbon from the host, the role of the maintained proplastid in parasite cells remains in question.

Host specificity and parasite resistance

Red algal parasites are known to be extremely host specific, usually infecting one to a few, closely related host species [7,40,41]. A study using the adelphoparasite *Janczewskia morimotoi* tested its ability to infect 15 other species including close relatives of its natural host, *Laurencia nipponica*, as well as members of different genera [42]. While *J. morimotoi* was capable of infecting two close relatives of its natural host, the more distantly related potential hosts prevented parasite infections [42]. Additionally, the host specificity of *Leachiella pacifica* was assessed through culture studies attempting to use parasites isolated from *Polysiphonia paniculata* to infect *Pterocladia bipinnata* and vice-versa [41]. Parasites isolated from *P. paniculata* could infect other populations of the same species as well as some other *Polysiphonia* species, however they could not infect *Pt. bipinnata* populations that were susceptible to parasites isolated from other *Pt. bipinnata* specimens [41]. These *L. pacifica* isolates showed strong genus-level host specificity. However, due to the greatly reduced morphology of red algal parasites, it cannot be ruled out that parasites isolated from different genera are, in fact, different host-specific species. Revisiting this study with molecular data would strengthen our understanding of host specificity and potentially reveal cryptic parasite species.

Dawsoniocolax bostrychiae and *Bostrychiocolax australis* are parasites that infect *Bostrychia radicans* [40]. A study on host range and specificity of these parasites on a variety of potential hosts, yielded similar results to the *J. morimotoi* study: the genetic distance between parasite and host has a strong negative correlation with susceptibility to parasite penetration and infection [40]. The authors note that they encountered hosts that are resistant to parasite infection, including some host populations that contained resistant and susceptible specimens [40]. In several cases the parasite was capable of forming an initial infection in a resistant host, however the host cell or cells adjacent to the infected cell died off, preventing the parasite from spreading further into the host [40]. However, subsequent molecular studies revealed phenotypic plasticity and cryptic diversity in *B. radicans* [43]. Therefore, the possibility remains that resistant and susceptible hosts from the host resistance study were actually different species. These findings emphasize the need

for ongoing taxonomic evaluation of red algal parasites and their hosts. Without the taxonomic framework, questions about whether or not the host is actually resisting parasite infection cannot be answered conclusively.

Many questions remain

Why does the parasite maintain a copy of the host plastid as it is forming its own reproductive cells and spores? Other parasites that have evolved from a plastid bearing ancestor, including the apicomplexans *Eimeria tenella* and *Plasmodium falciparum*, the parasitic plant *Epifagus virginiana* and many others, maintain a reduced plastid for cellular functions other than photosynthesis, such as fatty acid biosynthesis [2,30,31]. However, none of these plastid-bearing parasites steal a plastid from their host like the red algal adelphoparasites. Are adelphoparasites genetically similar enough to their hosts that they can target nuclear-encoded proteins to the host-derived proplastid and utilize those products for fatty acid biosynthesis? Genomic analyses of signaling and targeting peptides for plastid targeted nuclear genes in red algal parasites, combined with transcriptomic and proteomic approaches, will provide valuable insight into the role of the plastids in the infection mechanism and parasite life cycle. The use of additional molecular tools including in-situ hybridization would enable researchers to localize parasite nuclear-encoded proteins in the heterokaryotic cell.

Furthermore, the taxonomic range and multiple independent origins of red algal parasites makes it difficult to make generalizations based on a few observations. Thus far, the origin of red algal parasite plastids has only been investigated in adelphoparasites and the origin of the alloparasite plastid remains unknown. The assumption is that alloparasites first progress through an adelphoparasite stage and also maintain a co-opted host plastid. However, there are distinct developmental differences between adelpho- and alloparasites, including the initial steps of infection and alloparasites inability to synthesize DNA in heterokaryon cells (Fig. 2). Therefore, it seems plausible to propose an alternative hypothesis that, rather than passing through an adelphoparasite stage, alloparasites are capable of directly evolving infection mechanisms to parasitize distantly related hosts. In the proposed scenario, alloparasites would presumably maintain their own plastid, as they are likely incapable of utilizing such a genetically distant host plastid. Preliminary genomic data from *C. polysiphoniae* indicates this may, in fact, be the case (Salomaki and Lane, unpublished)

Future research investigating red algal parasite evolution will provide unique insight into the effects of transitioning from a free-living to a parasitic life strategy. Molecular data has supported morphological observations that red algal parasites share a recent common ancestor with their hosts [4–6,10]. However, further use of molecular tools is essential to provide a robust taxonomic framework of red algal parasites and their hosts. Only then can meaningful observations be made about host specificity and parasite resistance. With the technological advances of the past few decades and continually decreasing costs of DNA sequencing, information about the relationships between parasites and their hosts, unraveling the roles of parasite and host interactions, and the origins and function of organelles is within our grasp.

Acknowledgments

The authors express their thanks to two anonymous reviewers who provided helpful feedback in improving the manuscript. Funding for this work was provided by the National Science Foundation (grant #1257472).

Authors' contributions

The following declarations about authors' contributions to the research have been made: conceived of the topic for this review and wrote the initial draft: EDS; provided editorial feedback, conceptual guidance and figures for the final manuscript: CEL.

Competing interests

No competing interests have been declared.

References

1. Lafferty KD, Allesina S, Arim M, Briggs CJ, de Leo G, Dobson AP, et al. Parasites in food webs: the ultimate missing links: parasites in food webs. Ecol Lett. 2008;11(6):533–546. http://dx.doi.org/10.1111/j.1461-0248.2008.01174.x
2. Wilson RJMI, Denny PW, Preiser PR, Rangachari K, Roberts K, Roy A, et al. Complete gene map of the plastid-like DNA of the malaria parasite *Plasmodium falciparum*. J Mol Biol. 1996;261(2):155–172. http://dx.doi.org/10.1006/jmbi.1996.0449
3. Köhler S, Delwiche CF, Denny PW, Tilney LG, Webster P, Wilson RJM, et al. A plastid of probable green algal origin in apicomplexan parasites. Science. 1997;275(5305):1485–1489. http://dx.doi.org/10.1126/science.275.5305.1485
4. Goff LJ, Moon DA, Nyvall P, Stache B, Mangin K, Zuccarello G. The evolution of parasitism in the red algae: molecular comparisons of adelphoparasites and their hosts. J Phycol. 1996;32(2):297–312. http://dx.doi.org/10.1111/j.0022-3646.1996.00297.x
5. Zuccarello GC, Moon D, Goff LJ. A phylogenetic study of parasitic genera placed in the family Choreocolacaceae (Rhodophyta). J Phycol. 2004;40(5):937–945. http://dx.doi.org/10.1111/j.1529-8817.2004.04029.x
6. Goff LJ, Ashen J, Moon D. The evolution of parasites from their hosts: a case study in the parasitic red algae. Evolution. 1997;51(4):1068. http://dx.doi.org/10.2307/2411036
7. Goff LJ. The biology of parasitic red algae. In: Round F, Chapman D, editors. Progress in phycological research. Amsterdam: Elsevier Biomedical Press; 1982. p. 289–369. (vol 1).
8. Goff LJ, Coleman AW. The role of secondary pit connections in red algal parasitism. J Phycol. 2004;21(3):483–508. http://dx.doi.org/10.1111/j.0022-3646.1985.00483.x
9. Setchell WA. Parasitism among red algae. Proc Am Philos Soc. 1918;57(2):155–172.
10. Kurihara A, Abe T, Tani M, Sherwood AR. Molecular phylogeny and evolution of red algal parasites: a case study of *Benzaitenia*, *Janczewskia*, and *Ululania* (Ceramiales). J Phycol. 2010;46(3):580–590. http://dx.doi.org/10.1111/j.1529-8817.2010.00834.x
11. Verbruggen H, Maggs CA, Saunders GW, Le Gall L, Yoon H, de Clerck O. Data mining approach identifies research priorities and data requirements for resolving the red algal tree of life. BMC Evol Biol. 2010;10(1):16. http://dx.doi.org/10.1186/1471-2148-10-16
12. Pueschel CM, Cole KM. Rhodophycean pit plugs: an ultrastructural survey with taxonomic implications. Am J Bot. 1982;69(5):703. http://dx.doi.org/10.2307/2442960
13. Ramus J. Pit connection formation in the red alga *Pseudogloiophloea*.

J Phycol. 1969;5(1):57–63. http://dx.doi.org/10.1111/j.1529-8817.1969.tb02577.x

14. Ramus J. Properties of septal plugs from the red alga *Griffithsia pacifica*. Phycologia. 1971;10(1):99–103. http://dx.doi.org/10.2216/i0031-8884-10-1-99.1
15. Turner CHC, Evans LV. Translocation of photoassimilated ^{14}C in the red alga *Polysiphonia lanosa*. Br Phycol J. 1978;13(1):51–55. http://dx.doi.org/10.1080/00071617800650061
16. Hawkins EK. Observations on the developmental morphology and fine structure of pit connections in red algae. Cytologia. 1972;37(4):759–768. http://dx.doi.org/10.1508/cytologia.37.759
17. Wetherbee R, Quirk H. The fine structure of secondary pit connection formation between the red algal alloparasite *Holmsella australis* and its red algal host *Gracilaria furcellata*. Protoplasma. 1982;176:166–176. http://dx.doi.org/10.1007/BF01283319
18. Wetherbee R, Quirk HM, Mallett JE, Ricker RW. The structure and formation of host-parasite pit connections between the red algal alloparasite *Harveyella mirabilis* and its red algal host *Odonthalia floccosa*. Protoplasma. 1984;119(1–2):62–73. http://dx.doi.org/10.1007/BF01287818
19. Blouin NA, Lane CE. Red algal parasites: models for a life history evolution that leaves photosynthesis behind again and again. Bioessays. 2012;34(3):226–235. http://dx.doi.org/10.1002/bies.201100139
20. Wetherbee R, Quirk HM. The fine structure and cytology of the association between the parasitic red alga *Holmsella australis* and its red algal host *Gracilaria furcellata*. Protoplasma. 1982;110(3):153–165. http://dx.doi.org/10.1007/BF01283318
21. Goff LJ, Zuccarello G. The evolution of parasitism in red algae: cellular interactions of adelphoparasites and their hosts. J Phycol. 1994;30(4):695–720. http://dx.doi.org/10.1111/j.0022-3646.1994.00695.x
22. Goff LJ, Coleman AW. Fate of parasite and host organelle DNA during cellular transformation of red algae by their parasites. Plant Cell. 1995;7(11):1899. http://dx.doi.org/10.2307/3870197
23. Goff LJ, Coleman AW. Transfer of nuclei from a parasite to its host. Proc Natl Acad Sci USA. 1984;81(17):5420–5424. http://dx.doi.org/10.1073/pnas.81.17.5420
24. Goff LJ, Coleman AW. Nuclear transfer from parasite to host: a new regulatory mechanism of parasitism. Ann NY Acad Sci. 1987;503(1):402–423. http://dx.doi.org/10.1111/j.1749-6632.1987.tb40626.x
25. Goff LJ. The biology of *Harveyella mirabilis* (Cryptonemiales, Rhodophyceae). V. Host responses to parasite infection. J Phycol. 1976;12(3):313–328. http://dx.doi.org/10.1111/j.1529-8817.1976.tb02850.x
26. Kugrens P, West JA. The ultrastructure of an alloparasitic red alga *Choreocolax polysiphoniae*. Phycologia. 1973;12(3–4):175–186. http://dx.doi.org/10.2216/i0031-8884-12-3-175.1
27. Goff LJ, Coleman AW. Elucidation of fertilization and development in a red alga by quantitative DNA microspectrofluorometry. Dev Biol. 1984;102(1):173–194. http://dx.doi.org/10.1016/0012-1606(84)90183-0
28. Borza T, Popescu CE, Lee RW. Multiple metabolic roles for the nonphotosynthetic plastid of the green alga *Prototheca wickerhamii*. Eukaryot Cell. 2005;4(2):253–261. http://dx.doi.org/10.1128/EC.4.2.253-261.2005
29. de Koning AP, Keeling PJ. The complete plastid genome sequence of the parasitic green alga *Helicosporidium* sp. is highly reduced and structured. BMC Biol. 2006;4(1):12. http://dx.doi.org/10.1186/1741-7007-4-12
30. Wolfe KH, Morden CW, Palmer JD. Function and evolution of a minimal plastid genome from a nonphotosynthetic parasitic plant. Proc Natl Acad Sci USA. 1992;89(22):10648–10652. http://dx.doi.org/10.1073/pnas.89.22.10648
31. Cai X, Fuller AL, McDougald LR, Zhu G. Apicoplast genome of the coccidian *Eimeria tenella*. Gene. 2003;321:39–46. http://dx.doi.org/10.1016/j.gene.2003.08.008
32. Vaidya AB, Mather MW. Mitochondrial evolution and functions in malaria parasites. Annu Rev Microbiol. 2009;63(1):249–267. http://dx.doi.org/10.1146/annurev.micro.091208.073424
33. Hancock L, Goff L, Lane C. Red algae lose key mitochondrial genes in response to becoming parasitic. Genome Biol Evol. 2010;2:897–910. http://dx.doi.org/10.1093/gbe/evq075
34. Callow JA, Callow ME, Evans LV. Nutritional studies on the parasitic red alga *Choreocolax polysiphoniae*. New Phytol. 1979;83(2):451–462. http://dx.doi.org/10.1111/j.1469-8137.1979.tb07470.x
35. Setchell WA. Parasitic florideae. I. Berkeley, CA: University of California Press; 1914. (University of California publications in botany; vol 6).
36. Court GJ. Photosynthesis and translocation studies of *Laurencia spectabilis* and its symbiont *Janczewskia gardneri* (Rhodophyceae). J Phycol. 1980;16(2):270–279. http://dx.doi.org/10.1111/j.0022-3646.1980.00270.x
37. Goff LJ. The biology of *Harveyella mirabilis* (Cryptonemiales, Rhodophyceae). VI. Translocation of photoassimilated ^{14}C. J Phycol. 1979;15(1):82–87. http://dx.doi.org/10.1111/j.1529-8817.1979.tb02966.x
38. Evans LV, Callow JA, Callow ME. Structural and physiological studies on the parasitic red alga *Holmsella*. New Phytol. 1973;72(2):393–402. http://dx.doi.org/10.1111/j.1469-8137.1973.tb02047.x
39. Kremer BP. Carbon economy and nutrition of the alloparasitic red alga *Harveyella mirabilis*. Mar Biol. 1983;76(3):231–239. http://dx.doi.org/10.1007/BF00393022
40. Zuccarello GC, West JA. Host specificity in the red algal parasites *Bostrychiocolax australis* and *Dawsoniocolax bostrychiae* (Choreocolacaceae, Rhodophyta). J Phycol. 1994;30(3):462–473. http://dx.doi.org/10.1111/j.0022-3646.1994.00462.x
41. Zuccarello GC, West JA. Genus and race specificity in the red algal parasite *Leachiella pacifica* (Choreocolacaceae, Rhodophyta). Phycologia. 1994;33(3):213–218. http://dx.doi.org/10.2216/i0031-8884-33-3-213.1
42. Nonomura AM, West JA. Host specificity of *Janczewskia* (Ceramiales, Rhodophyta). Phycologia. 1981;20(3):251–258. http://dx.doi.org/10.2216/i0031-8884-20-3-251.1
43. Zuccarello GC, West JA. Multiple cryptic species: molecular diversity and reproductive isolation in the *Bostrychia radicans*/*B. moritziana* complex (Rhodomelaceae, Rhodophyta) with focus on North American isolates. J Phycol. 2003;39(5):948–959. http://dx.doi.org/10.1046/j.1529-8817.2003.02171.x