A War We Need: A Synthesis of Coccolithophore–Virus Interaction

Liz Koifman

Introduction

Emiliania huxleyi (E. huxleyi) is a globally abundant coccolithophore whose massive coastal and open-ocean blooms play a central role in marine carbon cycle, particularly through the ability to both photosynthetically fix CO■ into particulate organic carbon (POC) and biomineralize particulate inorganic carbon (PIC) as calcium carbonate (CaCO■) coccoliths. Research shows coccolithophores presently account for at least half of the 80–120 Tmol of annual PIC production in the pelagic ocean and coccolith-associated calcite produced in the surface waters and exported into the deep ocean account for around 83% of global, ballasted POC fluxes (Johns et al., 2019). Coccolith-associated calcite makes up about half of calcite raining down on to the marine seafloor, with the other half being derived from foraminifera (Broecker & Clark, 2009) E. huxleyi blooms are routinely terminated by large double-stranded DNA viruses known as coccolithoviruses, which infect and lyse populations on basin-wide scales (Vardi et al., 2012). The interaction between E. huxleyi and its viruses is now understood to extend far beyond a simple host–pathogen relationship. Rather it represents a closely coupled system in which host biomineralization, viral genetic innovation, strain-specific biochemical defenses, and chemically mediated infection pathways intersect to shape bloom dynamics and global carbon export. Recent genomic, physiological, and mesocosm studies have revealed that coccolithovirus infection engages host lipid signaling pathways, manipulates calcification, and restructures plankton communities in ways that have cascading ecological effects.

Coccolithophore Physiology and Calcification

Calcification is a defining feature of E. huxleyi. Each cell is surrounded by a coccosphere composed of intricately patterned calcium carbonate plates, whose abundance and thickness vary widely among strains. Research has demonstrated that strains with higher degrees of calcification consistently exhibit lower infection rates and reduced viral replication compared to naked strains, those without coccoliths, indicating that the coccosphere provides a meaningful protective barrier. Experimentally reducing environmental calcium prevents calcification and converts previously resistant strains into highly susceptible ones, demonstrating that the coccosphere acts directly as a defensive interface against coccolithoviruses (Johns et al., 2019).

Strain-level physiology further modifies this interaction. Some E. huxleyi genotypes with high dimethylsulphoniopropionate lyase (DMSP-lyase) activity are resistant to infection, suggesting that sulfur metabolic pathways may generate antiviral chemical conditions that interact with the degree of calcification (Schroeder et al., 2002). These observations indicate that calcification does not operate in isolation but is part of a broader suite of physiological traits that collectively structure viral outcomes in natural populations.

Calcification also shapes the biochemical landscape of the cell surface. Calcified cells demonstrate elevated concentrations of sialic-acid glycosphingolipids (sGSLs), membrane lipids involved in viral adsorption and surface charge regulation. Further analysis showed when Ca^{2}■ availability is lowered, both PIC and sGSL levels decline, revealing a direct link between biomineral formation and the lipid-signaling pathways that later govern infection. Reduced calcification is therefore associated with both diminished sGSL abundance and heightened viral susceptibility (Johns et al., 2019).

The relationship between calcification and infection is not strictly protective, however. Although calcified cells and their detached coccoliths exhibit significantly higher viral adsorption coefficients than naked cells, reduced calcification still leads to greater viral production. Similarly, E. huxleyi viruses (EhVs) actively disrupt calcification by triggering massive coccolith shedding, even in the absence of cell lysis, and these effects can be reproduced by exposing healthy calcified cells to virus-conditioned but particle free media (Johns et al., 2019). Together, these findings establish calcification as a central component of the arms race between E. huxleyi and its viruses. It shapes adsorption, susceptibility, and chemical signaling while also providing a physical barrier to prevent infection.

Coccolithovirus Biology and Infection Mechanisms

Early experiments conducted on natural viral isolates collected from collapsing blooms in the English Channel have established that the E. huxleyi specific viruses (EhVs) are large icosahedral dsDNA viruses, 170–200 nm in diameter (Wilson et al., 2002). Morphological consistency across ten isolates, paired with phylogenetic analysis of their DNA polymerase genes, confirmed that they form a distinct lineage within the family Phycodnaviridae, now recognized as the genus Coccolithovirus (Schroeder et al., 2002). The first Coccolithovirus genome to be fully sequenced was Eh V-86, with analysis revealing an unexpectedly large and functionally novel dsDNA genome with a length of over 407 kbp and 472 genes predicted [Figure 1]. Research found that of these genes, only a fraction match known proteins, emphasizing how genetically distinct Coccolithoviruses are relative to other Phycodnaviridae. They discovered virus-specific promoter regions, introns, and a suite of RNA polymerase subunits that allow EhV-86 to conduct at least part of its own transcription independently of the host nucleus (Wilson et al., 2005). This transcriptional autonomy, combined with a highly dense coding architecture, supports the idea that coccolithoviruses form a distinct, sophisticated lineage within the nucleocytoplasmic large DNA viruses.

Figure 1. Circular representation of the 407,339-bp EhV-86 genome (Wilson et al., 2005)

The most transformative insight from the EhV-86 genome is the discovery of a viral sphingolipid biosynthesis pathway, including genes responsible for encoding serine palmitoyltransferase, and dihydroceramide desaturase. These enzymes enable the synthesis of viral glycosphingolipids (vGSLs), which act as signaling molecules within infected cells. Sphingolipids are membrane lipids present in all eukaryotes and some prokaryotes and also play a key role in several processes, particularly signal transduction (Wilson et al., 2005). A sphingolipid biosynthesis pathway has not previously been discovered on a virus genome. Sphingolipid biosynthesis leads to the formation of ceramide (Merril Jr., 2002), which is an intracellular signal known to suppress cell growth and trigger apoptosis (Futerman & Hannun, 2004).

Their ecological relevance was demonstrated in mesocosm experiments linking laboratory results to natural bloom conditions which revealed that coccolithovirus infection of E. huxleyi is driven by a coordinated set of lipid-based and oxidative signals. These vGSLs act as potent signaling molecules that trigger a cascade of host cellular responses, including elevated reactive oxygen species (ROS), caspase-specific enzymatic activity, and increased metacaspase expression [Figure 2]. Together, these markers indicate the activation of programmed cell death, which not only kills infected cells but can induce death in nearby uninfected neighbors, accelerating bloom collapse (Vardi et al., 2012). This mechanism highlights a sophisticated viral strategy in which the virus reprograms host lipid metabolism to generate biochemical signals that actively terminate the population it infects. This provides a rare mechanistic bridge between subcellular processes and ecosystem-scale bloom dynamics

Figure 2. E. huxleyi Cellular Response and Infection Signals (Vardi et al., 2012).

Biogeochemical Consequences: Viral Shunt and Carbon Export

A key ecological insight from the research discussed is that PCD and lipid signaling do not only terminate blooms, they also reshape carbon cycling. As infection progresses, virus-induced ROS and caspase activity are accompanied by a marked increase in the production of transparent exopolymer particles (TEP), sticky polysaccharides that promote cell aggregation [Figure 3]. Microscopy from the mesocosms revealed E. huxleyi cells encased within larger TEP-rich matrices, indicating accelerated formation of “marine snow” (Vardi et al., 2012). This aggregation enhances the downward transport of particulate organic and inorganic carbon, effectively strengthening marine carbon cycling. Thus, coccolithovirus infection of E. huxleyi couples cell death with enhanced sinking, providing a mechanistic explanation for how viral infection influences the biological carbon pump.

Figure 3. Coccolithophore TEP Production (Vardi et al., 2012).

The inorganic carbon cycle is similarly affected. Viral infection triggers widespread coccolith shedding, releasing dense CaCO■ platelets that serve as an effective ballast, thereby increasing the sinking velocity of aggregates. Detached coccoliths are also highly adsorptive, which reduces the number of free infectious virions even as they contribute to enhanced PIC export (Johns et al., 2019). Thus, infection simultaneously alters both the organic and inorganic branches of carbon cycling, transforming a bloom from an actively growing biomass population into a source of sinking mineral organic aggregates.

These biogeochemical effects are shaped by ecological and evolutionary interactions within the bloom. Host–virus specificity means that only strains with low DMSP-lyase activity are susceptible to infection, whereas high-lyase strains which convert dimethylsulphoniopropionate (DMSP) into dimethylsulphide (DMS) and acrylic acid tend to be resistant. Because DMS is a key precursor of marine sulfur aerosols, the selective removal of low-lyase strains and survival of high-lyase strains has implications not only for bloom succession but also for sulfur cycling and potential atmospheric feedback. Viral diversity further amplifies these effects, with multiple coccolithovirus genotypes coexisting within a single bloom, each targeting different host subpopulations (Schroeder et al., 2002). This diversity contributes to rapid shifts in community composition as infection progresses, altering the balance of calcified and non-calcified cells and consequently influencing both PIC and POC export pathways.

Cumulative research therefore shows that coccolithovirus infection is a major driver of carbon export processes. Calcification dynamics, viral glycosphingolipid signaling, TEP production, and coccolith shedding operate as an integrated system that transforms living phytoplankton biomass into sinking material, restructuring bloom communities while redirecting carbon and sulfur flows. By terminating blooms, modulating the partitioning of organic and inorganic carbon, and interacting with host sulfur metabolism, coccolithoviruses exert a disproportionately large influence on marine biogeochemistry and atmospheric feedback (Schroeder et al., 2002).

Conclusion and Future Directions

The interaction between Emiliania huxleyi and coccolithoviruses illustrates how a single host-virus system can shape marine ecosystems far beyond the scale of individual cells. Viral manipulation of host lipid signaling, combined with the dismantling of calcification structures, enables coccolithoviruses to trigger rapid programmed cell death and bloom collapse. At the same time, host traits such as calcification, glycosphingolipid composition, and DMSP-lyase activity mediate resistance and structure infection patterns across natural populations. These tightly linked physiological and molecular processes translate directly into altered carbon flux as infection drives TEP production, coccolith shedding, and aggregation, accelerating the export of both organic and inorganic

carbon to the deep ocean. Although these studies reveal a sophisticated arms race with major biogeochemical consequences, key mechanisms remain unresolved. The identity of the viral infochemicals that induce decalcification, the details of host-viral sphingolipid interactions, and the ecological implications of strain-level diversity are still poorly understood. Future work integrating genomic, lipidomic, and field-based approaches will be essential for clarifying these pathways and quantifying how infection-driven carbon export varies under shifting ocean conditions. In summary, the interactive system between E. huxleyi and EhV provides a powerful model for understanding how microbial interactions regulate the biological carbon pump and influence the structure and function of marine ecosystems.

References

Broecker, W., and E. Clark. 2009. Ratio of coccolith CaCO3 to foraminifera CaCO3 in late Holocene deep sea sediments. Paleoceanogr. 24(3).

Futerman, A.H., and Y.A. Hannun. 2004. The complex life of simple sphingolipids. EMBO Rep. 5(8): 777-782.

Johns, C.T., A.R. Grubb, J.I. Nissimov, F. Natale, V. Knapp, et al. 2019. The mutual interplay between calcification and coccolithovirus infection. Environ. Microbiol. 21(6): 1896-1915.

Merrill Jr, A.H. 2002. De novo sphingolipid biosynthesis: a necessary, but dangerous, pathway. J. Biol. Chem. 277(29): 25843-25846.

Obeid, L.M., C.M. Linardic, L.A. Karolak, and Y.A. Hannun. 1993. Programmed cell death induced by ceramide. Sci. 259(5102): 1769-1771

Schroeder, D., J. Oke, G. Malin, and W.H. Wilson. 2002. Coccolithovirus (Phycodnaviridae): Characterisation of a new large dsDNA algal virus that infects Emiliana huxleyi. Arch. Virol. 147: 1685–1698.

Vardi, A., L. Haramaty, B.A.S. Van Mooy, H.F. Fredricks, S.A. Kimmance, et al. Host–virus dynamics and subcellular controls of cell fate in a natural coccolithophore population. 2012. Proc. Natl. Acad. Sci. U.S.A.. 109 (47): 19327-19332.

Wilson, W.H., D.C. Schroeder, M.J. Allen, M.T.G. Holden, J. Parkhill, et al. 2005. Complete Genome Sequence and Lytic Phase Transcription Profile of a Coccolithovirus. Sci. 309: 1090-1092.

Wilson, W.H., G.A. Tarran, D. Schroeder, M. Cox, J. Oke, and G. Malin. 2002. Isolation of a virus responsible for the demise of an Emiliania huxleyi bloom in the English Channel. J. Mar. Biol. Assoc. U.K.. 82(3): 369-377.