

oyster mortality in Galveston Bay rose from 11% to 48%, reaching 100% at some reefs; mortality was significantly correlated with the duration of bottom salinity below 5 psu, while storm-induced sediment deposition showed no such relationship, implicating extended low-salinity exposure as the main driver (Du et al., 2021). A similar pattern was observed when a series of atmospheric rivers produced extreme freshwater discharge into San Francisco Bay, causing sustained salinities below ~6.3 and a near-100% mass mortality of wild oysters, closely matching critical salinity tolerances and highlighting how rare events can wipe out already depleted populations.

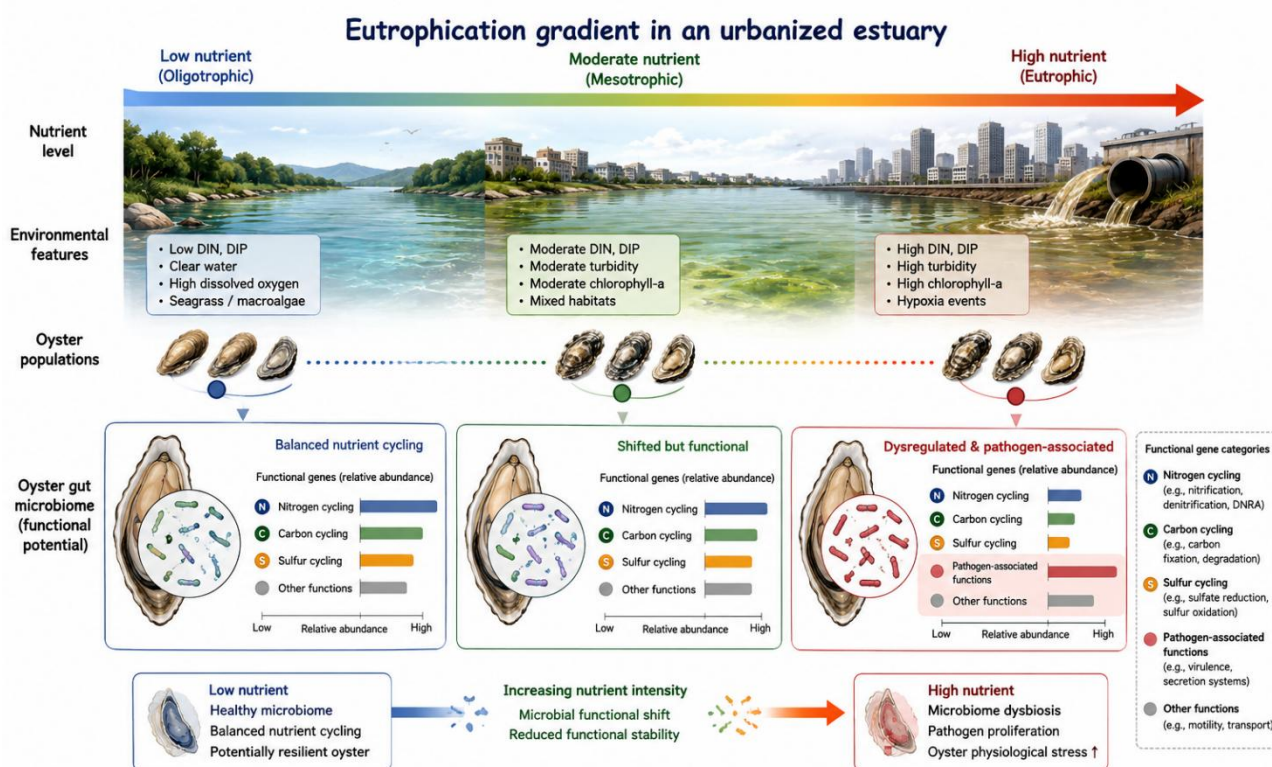


Figure 1 Changes in oyster gut microbiome functional profiles along an estuarine eutrophication gradient. Nutrient enrichment alters microbial functional genes associated with nutrient cycling, suggesting a link between eutrophication intensity, microbial dysbiosis, and potential host physiological stress

5 Disease Transmission and Epidemiological Characteristics

5.1 Waterborne transmission and density-dependent spread mechanisms

Experimental and modeling work for *Vibrio aestuarianus* shows clear waterborne transmission, with a basic reproduction number (R_0) around 2.9 and a generation time of ~5.5 days in small, closed oyster populations. (Lupo et al., 2020). Transmission is dose-dependent, driven by bacterial shedding and the concentration in seawater, implying threshold exposure levels needed to initiate an epidemic. At bay scale, hydrodynamic connectivity and temperature control the spatial spread of waterborne bacteria, allowing long-distance dispersal between distant farms over months. Theoretical models of marine infectious diseases further emphasize that highly infected oysters and the balance between pathogen release and removal in the water column determine outbreak thresholds and the existence (or not) of low-abundance refuges.

5.2 Transmission via aquaculture facilities and biological vectors

Recirculating and hatchery-like systems modify but do not eliminate waterborne viral circulation: OsHV-1 persists in seawater despite biofiltration and UV, and detection odds are higher where treatment is interrupted, showing facility design directly affects pathogen loads. Cohabitation experiments show higher donor biomass and feeding (algal addition) markedly increase OsHV-1 transmission and mortality, indicating facility management of stocking and feeding influences effective dose exposure. Field observations in Woollooware Bay show clustered OsHV-1 mortalities within farms and vertical stratification in the water column, consistent with infection from common