# White Plague Disease | Enfermedad de Plaga Blanca

*These paragraphs were written by Emma Korein, with contributions from Tania Metz Estrella, Melissa Gonzalez, Dr. Ernesto Weil and Miguel Figuerola. These paragraphs were last updated in July 2021.*

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White plague disease (WPD) has two variants, type I (WPD-I) and type II (WPD-II), both of which were first described in Florida in the 1970s. Compared with WPD-I which predominantly affects *Mycetophyllia ferox* and the *Orbicella spp.* complex, WPD-II has a higher virulence and affects more species (it is estimated that over 40 species have been impacted, *Montastraea* and *Orbicella* species being the most prominently affected).[1–4] WPD is characterized by rapidly progressing rates of tissue mortality (1-10 cm/day) that leave behind exposed coral skeleton, usually starting at the colony base and moving upwards.[2,5] WPD was first detected in Puerto Rico in La Parguera in 1995 and has since been observed throughout the archipelago, including in mesophotic reefs.[6,7] WPD prevalence and subsequent coral loss greatly increased in Puerto Rico and the wider Caribbean in 2005 and 2010 in correlation with the two highest thermal anomalies in recorded history, which also induced the two most extensive and intensive bleaching events.[6,8–11] This event led to massive declines in key reef building species, which was further exacerbated by bleaching, other coral diseases, hurricanes, and other impacts.[6,8,12] This epizootic had long-term ecosystem-wide impacts on coral communities, including a loss of large reef building species.[12–15] The etiology of all WPD types is unknown and therefore precise diagnosis is difficult, especially given visual similarities with other white syndromes that also do not have an identified putative pathogen. Multiple studies have found microbial shifts between healthy and WPD affected coral tissue, indicating a bacterial pathogen could play a causative role.[16–19] Early research isolated *Aurantimonas coralicida* as the pathogen of WPD-II,[20] however research since has taken WPD samples completely absent of this bacterium and there is no current consensus on a single pathogen associated with WPD symptoms.[16–19] Viruses have also been considered as a possible causative agent,[21] and certain environmental factors may also play a role in WBD prevalence, including water depth, temperature, light intensity, season, water turbidity, salinity, and levels of oxygen.[6,10,22–24] WPD susceptibility varies significantly by coral species, possibly as a result of varying thresholds for microbial dysbiosis, or a microbial imbalance in the coral host that can result in disease.[25,26] WPD is highly transmissible and known to spread through water, direct contact, and animal vectors, including the corallivorous worm *Hermodice carunculate* and the corallivorous snail *Coralliophila abbreviate.*[1,15,27] While treatment is not widely used, research has found phage therapy,[28,29] shading[23] and epoxy putty[30] to be effective techniques at reducing WPD progression.

La enfermedad de plaga blanca (WPD, por sus siglas en inglés) tiene dos variantes, tipo I (WPD-I) y tipo II (WPD-II), ambas descritas por primera vez en Florida en la década de 1970. En comparación con la WPD-I que afecta predominantemente a la especie *Mycetophyllia ferox* y el complejo de especies de *Orbicella*, la WPD-II tiene una mayor virulencia y afecta más especies (es estimado que más de 40 especies están afectadas, siendo que las especies *Montastraea* y *Orbicella* son las más afectadas).[1–4] La WPD se caracteriza por mortalidad tisular que progresa rápidamente (1 - 10 cm/día) que deja el esqueleto de coral expuesto, generalmente comenzando en la base de la colonia con progresión hacia arriba.[2,5] La WPD se detectó por primera vez en Puerto Rico en La Parguera en 1995 y desde entonces se ha observado la enfermedad a través del archipiélago, incluida en los arrecifes mesofóticos.[6,7] La prevalencia de la WPD y

la y perdido de coral vivo aumentó considerablemente en el Caribe en 2005 y 2010 en correlación con las dos anomalías térmicas más altas en la historia registrada, que también indujeron los dos eventos de blanqueamiento más extensos e intensos.[6,8–11] Este evento provocó una disminución masiva de especies formadoras del arrecife, que se exasperó aún más por el blanqueamiento, otras enfermedades de corales y huracanes, entre otros.[6,8,12] Esta epizootia tuvo impactos a largo plazo en el ecosistema de coral, incluido la pérdida de grandes especies formadoras del arrecife.[12–15] Se desconoce la etiología de todos los tipos de WPD y, por lo tanto, es difícil realizar un diagnóstico preciso, especialmente con las similitudes visuales de otros síndromes blancos que tampoco tienen un patógeno putativo identificado. Varios estudios han encontrado cambios microbianos entre el tejido sano y el tejido afectado por WPD, lo que indica que un patógeno bacteriano podría causar la enfermedad.[16–19] Las primeras investigaciones encontraron *Aurantimonas coralicida* como el patógeno de WPD-II.[20] Sin embargo, en investigaciones posteriores se encontró muestras de WPD sin esta bacteria, y no existe un consenso actual sobre un patógeno asociado con los síntomas de WPD.[16–19] Además, se ha considerado un virus como un posible agente causal,[21] y ciertos factores ambientales también podrían tener un rol importante en la prevalencia de la WBD, incluido la profundidad del agua, la temperatura, la intensidad de la luz, la temporada, la turbidez, la salinidad y los niveles de oxígeno.[6,10,22–24] La susceptibilidad a la WPD varía significativamente según la especie de coral, posiblemente como resultado de distintos umbrales de disbiosis microbiana, o un desequilibrio microbiano en el coral que puede resultar en enfermedad.[25,26] La WPD es altamente transmisible y se propaga a través del agua, el contacto directo y vectores animales, incluido el gusano coralívoro *Hermodice carunculate* y el caracol coralívoro *Coralliophila abreviado*.[1,15,27] Aunque no usado ampliamente, algunas investigaciones han encontrado que la sombra,[23] "phage therapy"[28,29] y la masilla epoxi[30] son técnicas efectivas para reducir la progresión de la WPD.

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