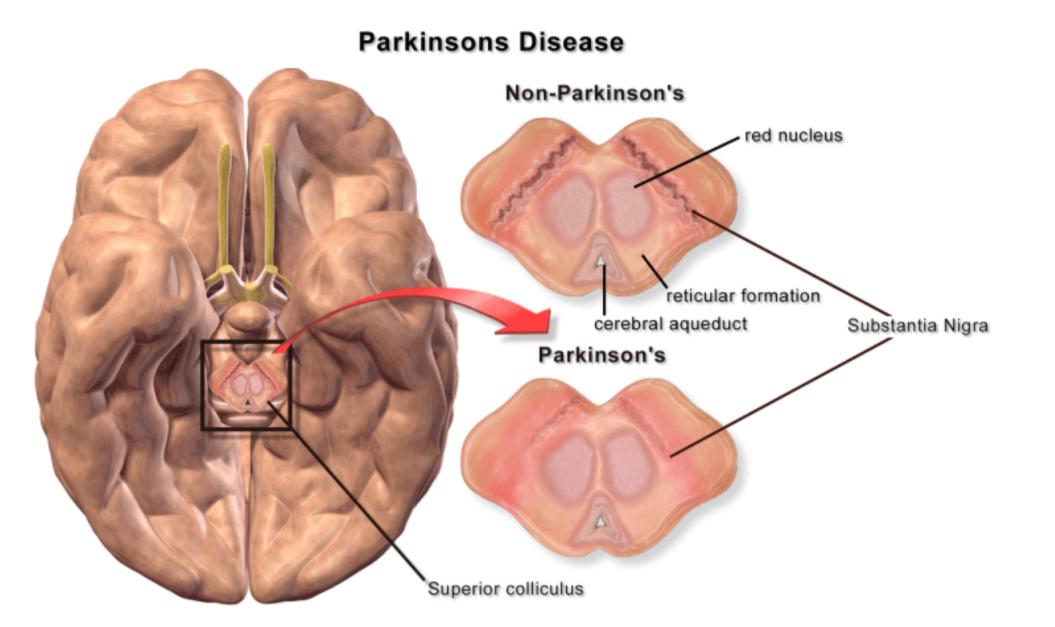


facilitate the formation of hydroxyl radicals. On the other hand, the selective loss of dopaminergic neurons containing neuromelanin is associated with Parkinson's disease (Mann and Yates 1983; Marsden 1983). The oxidized metabolites of dopamine, dopaminequinone derivatives, have been considered to contribute to

the degeneration of dopaminergic neurons. It should be noted that albino subjects manifest no neurological abnormality, except for the retinal abnormality mainly due to the impaired extension of the optic nerve fibers." (Takeda et al., 2007:205-206)

Italian researcher Luigi Zecca has written prolifically on this subject over the past two decades:

"The most highly pigmented cells in the human brain are the dopaminergic neurons of the substantia nigra and the noradrenergic neurons of the locus coeruleus [1,2]. The pigment, which is present in primates including chimpanzee, gibbon and baboon (and in their more distant relatives, such as horse and sheep [3,4]), is composed of neuromelanin (NM). This electron-dense substance is located in organelles surrounded by a double membrane in the neuronal perikaryon [5] that are known as NM granules. Parkinson's disease (PD) is characterized by preferential loss of those dopaminergic neurons that contain NM" (Zecca et al., 2003:578).



A lack of neuromelanin in the substantia migration is associated with Parkinson's Disease

Zecca and his colleagues have established that NM provides protection against damage to and destruction of neurons by binding to substances that cause this damage – a process known as chelation.

"The ability of NM to act as a 'black hole' capable of chelating redox-active metals [13,25] and a wide variety of drugs suggests that it could be a high capacity storage trapping system, and as such might prevent neuronal damage (Figure 1). It has also been suggested that the accumulation of toxic compounds by NM might be followed by a slow release of the toxin. However, this is unlikely because we have observed a high storage capacity of NM for toxic metals and have never found a saturation of this capacity in NM of human substantia nigra." (Zecca et al., 2003:579)

However, they have also found that in people with Parkinson's disease, extraneuronal NM, or NM that is no longer bound within a neuron, actually exacerbates neuron damage and destruction:

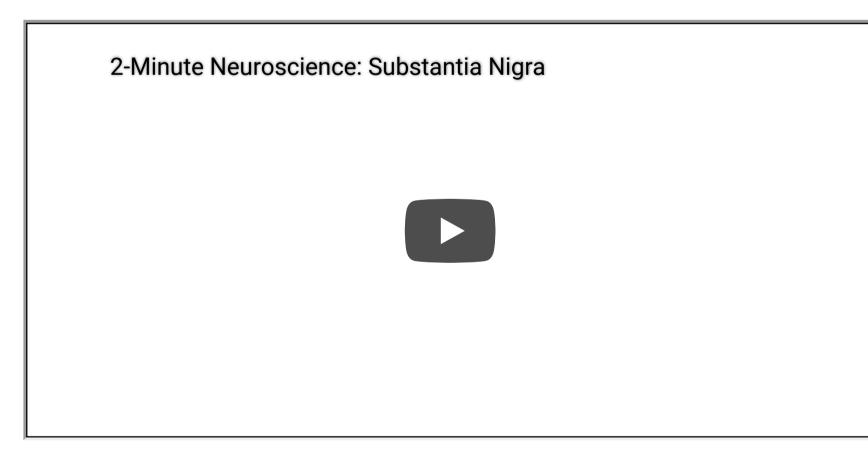
"Thus, in PD, although different mechanisms such as environmental toxins and genetic factors can initiate neuronal damage in the substantia nigra and striatum, NM released from dying neurons induces release of neurotoxic microglial factors, potentially leading to a subsequent aggravation of neuro-degeneration." (Zecca et al., 2003:579)

There is some indication that levels of neuromelanin may differ by ethnicity. For example, an American study in the early 00s found that the age- and gender-adjusted incidence rates of Parkinson's Disease were highest among Hispanics, followed by non-Hispanic Whites, Asians, and Blacks. (Van Den Eeden et al., 2003)

The same pattern (Hispanic highest, then non-Hispanic White with Black the lowest) was found by a 2005 review of cases of spina bifida anencephaly. Indeed, "the prevalence ratio for non-Hispanic black births was of borderline significance for spina bifida and was not significant for anencephaly." (Williams et al., 2005)

And Stewart (1996) reviewed several studies showing African and African-descended populations across the globe have much lower rates of central nervous system malformations (such as ancephalcy and spina bifada) and diseases (such as Parkinson's) than white people.

However, a major review of the incidence of brain and central nervous system tumours found that African American rates were only slightly lower than White American rates and were higher than the rates of other groups. (Quinn et al., 2015)



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