# Rethinking what causes Parkinson's: Studies suggest we may be looking in the wrong place

During her time as a postdoc at the University of Basel in Switzerland, Sarah Shahmoradian decided to study the abnormal aggregates of protein that develop inside nerve cells and contribute to Parkinson's disease.

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"We were originally looking for fibrils," Shahmoradian says, "but unexpectedly, we found an abundance of . . . mitochondria, other organelles, and lipid membranes [in the Lewy bodies]." The researchers also found evidence of lysosomes, organelles that facilitate cellular waste removal.

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The study is one of many that raise questions about the prevailing idea that ?-synuclein accumulation is the underlying cause of the neurodegeneration in Parkinson's disease. Rather, ?-synuclein buildup may be just one symptom of a more fundamental problem: the cells' inability to break down excess lipids and proteins, including ?-synuclein. Some Parkinson's patients carry mutations in genes associated with lysosomal function, and studies in mice have revealed that natural aging leads to the build-up of lipids associated with Parkinson's disease. These findings have led a small but growing set of scientists to propose that for a vast majority of Parkinson's patients, the disease is fundamentally a cellular machinery problem, not a protein problem.

**Read full, original post:** Is It Time to Rethink Parkinson's Pathology?