Supplementary Figure 1. Uncropped immunoblot examples. For each protein measured, four immunoblots were required for each brain region. These were cut into three strips to allow for immunoblotting of different sized proteins. Strips for each protein were identically processed and digitally imaged together, with cases from each group included on every strip. For each protein measured, one of the four strips are shown as an example of antibody specificity. For almost all antibodies used only a single band at the expected size was detected. In the case of other bands, * indicates the band that was quantified based on the expected molecular weight. Orange dots indicate control cases. Blue squares indicate idiopathic Parkinson's disease cases. Green filled and upturned unfilled triangles indicate G2019S and I2020T LRRK2 mutation cases respectively.

Supplementary Figure 2. Lack of α-synuclein Ser129 phosphorylation in the occipital cortex. Immunoblotting was used to measure α-synuclein Ser129 phosphorylation in the occipital cortex of the control and Parkinson's disease cases (**A**). Only two idiopathic Parkinson's disease cases had detectable α-synuclein phosphorylation (indicated by *). **B)** β-actin measured in the same immunoblots as A to demonstrate loading.

Supplementary Figure 3. LRRK2 phosphorylation in the occipital cortex. Multivariate analysis covarying for post-mortem delay was used to assess changes in levels of leucine-rich repeat kinase 2 (LRRK2) phosphorylation at serines 910 (**A**), 935 (**B**) and 973 (**C**) in the occipital cortex from controls (n=fourteen), idiopathic Parkinson's disease (n=thirteen) and LRRK2-associated Parkinson's disease cases (n=seventeen). The LRRK2 mutation cohort consisted of cases with the G2019S mutation (green filled triangles) and the I2020T mutation (upturned green unfilled triangles). Individual data points are shown on the graph as well as mean ± standard error of the mean. Representative immunoblots are shown with uncropped examples available in the supplementary material. Phosphorylated immunoblots were corrected to levels of total LRRK2.

Supplementary Figure 4. Braak staging correlates with α-synuclein Ser129 phosphorylation in the frontal cortex. Spearman analysis shows a significant correlation between Braak Lewy body stage and α-synuclein Ser129 phosphorylation in the frontal cortex

of the cases with Parkinson's disease. Both idiopathic and LRRK2-associated Parkinson's disease cases are included in the analysis.

Supplementary Figure 5. Cathepsin D and ATP13A2 in frontal cortex. Multivariate analysis covarying for post-mortem delay was used to assess changes in levels of cathepsin D (CatD) (**A,B**) and cation transporting ATPase 13A2 (ATP13A2) (**C**) in the occipital cortex from controls (n=fourteen), idiopathic Parkinson's disease (n=thirteen) and LRRK2-associated Parkinson's disease cases (n=seventeen). The LRRK2 mutation cohort consisted of cases with the G2019S mutation (green filled triangles) and the I2020T mutation (upturned green unfilled triangles). Individual data points are shown on the graph as well as mean ± standard error of the mean. Representative immunoblots are shown with uncropped examples available in the supplementary material.

Supplementary Figure 6. Levels of Cathepsin D negatively correlate with storage time. Spearman analysis revealed a significant negative correlation between levels of TBS soluble cathepsin D and the storage time of the post-mortem brain samples.