Supplementary Information for:

Subcellular compartmentalisation of copper, iron, manganese, and zinc in the Parkinson’s disease brain

Sian Genoud¹, Blaine R. Roberts², Adam P. Gunn², Glenda Halliday¹,³,⁴, Simon Lewis¹, Helen Ball², Dominic J. Hare²,⁶,⁷,*, Kay L. Double¹, *

¹ Discipline of Biomedical Science and Brain and Mind Centre, Sydney Medical School, The University of Sydney, Camperdown, NSW 2006, Australia
² The Florey Institute of Neuroscience and Mental Health, The University of Melbourne, Parkville, VIC 3052, Australia
³ Neuroscience Research Australia, Randwick, NSW 2031, Australia
⁴ School of Medical Sciences, University of New South Wales, NSW 2052, Australia
⁵ Bosch Institute, University of Sydney, Camperdown, NSW 2006, Australia
⁶ Elemental Bio-imaging Facility, University of Technology Sydney, Broadway, NSW 2007, Australia
⁷ Department of Pathology, The University of Melbourne, Parkville, VIC 3052, Australia

* Correspondence to dominic.hare@florey.edu.au or kay.double@sydney.edu.au
Figure S1: Total metal ratios in occipital cortex (OCx) fusiform gyrus (FUS) and substantia nigra (SN) of healthy aged controls and Parkinson’s disease. Both (a) Cu:Zn and (b) Cu:Fe were higher in the SN of healthy brains, and was decreased in the SN when compared to control. (c) The Cu:Mn ratio was decreased in the Parkinson’s disease SN, and the (d) Fe:Mn and (e) Fe:Zn ratio increased. * p < 0.05, *** p < 0.001 (vs control regions). # p < 0.05; ## p < 0.01 (Parkinson’s disease SN vs control SN). All concentrations are µg g⁻¹ wet weight of tissue.
Figure S2: Comparison of Braak stage in the Parkinson’s disease OCx, FUS and SN showed no significant difference for (a) Cu, (b) Fe, (c) Zn and (d) Mn.