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CORRIGENDUM: Ubiquinone-quantum dot bioconjugates for *in vitro* and intracellular complex I sensing

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The authors have noticed that Figure 1 was published incorrectly in the original version of this Article. The correct Figure 1 appears below.

Figure 1 | Schematic of ubiquinone-CdSe/ZnS QDs as redox fluorescence biosensor for Parkinson's disease diagnosis. (a), Ubiquinone-terminated disulphides (QnNS) synthesis and self-assembly of QnNS on to CdSe/ZnS QDs. (b), Conceptual visualisation of QnNS-QDs as complex I sensor *in vitro*. Under oxidized state (QnNS), ubiquinone functions as a favorable electron acceptor, this results in effective QDs' fluorescence quenching. Addition of complex I to QnNS-QDs solution in the presence of NADH, ubiquinone coupled electron transfer and proton translocation from NADH, producing reduced ubiquinol (HQnNS) form on the surface of QDs to mimic the initial stages of the respiratory chain. Ubiquinol when in close proximity to the QDs produces fluorescence enhancement. (c), Energetic diagram of the QDs bioconjugates between QDs and QnNS/HQnNS. (d), Fluorescence spectra of ubiquinone/ubiquinol- functionalised CdSe/ZnS QDs. (e), Cyclic voltammetry of QnNS-CdSe/ZnS QDs. (f), Visualisation of QnNS-CdSe/ZnS QDs as an intracellular complex I sensor. The mitochondrial-specific neurotoxin, rotenone, inhibits complex I and leads to Parkinson's-like pathogenesis. Parkinson's disease is characterised by impaired activity of complex I in mitochondria.