PLOS ONE

CORRECTION

Correction: S-Nitrosylation of G protein-coupled receptor kinase 6 and Casein kinase 2 alpha modulates their kinase activity toward alpha-synuclein phosphorylation in an animal model of Parkinson's disease

Weiwei Wu, Chun Chau Sung, Peichun Yu, Jiahua Li, Kenny K. K. Chung

In Figs 3–7, there are incorrect symbols in the labels. Please view the correct figures here.

Citation: Wu W, Sung CC, Yu P, Li J, Chung KKK (2020) Correction: S-Nitrosylation of G protein-coupled receptor kinase 6 and Casein kinase 2 alpha modulates their kinase activity toward alpha-synuclein phosphorylation in an animal model of Parkinson's disease. PLoS ONE 15(6): e0235296. https://doi.org/10.1371/journal.pone.0235296

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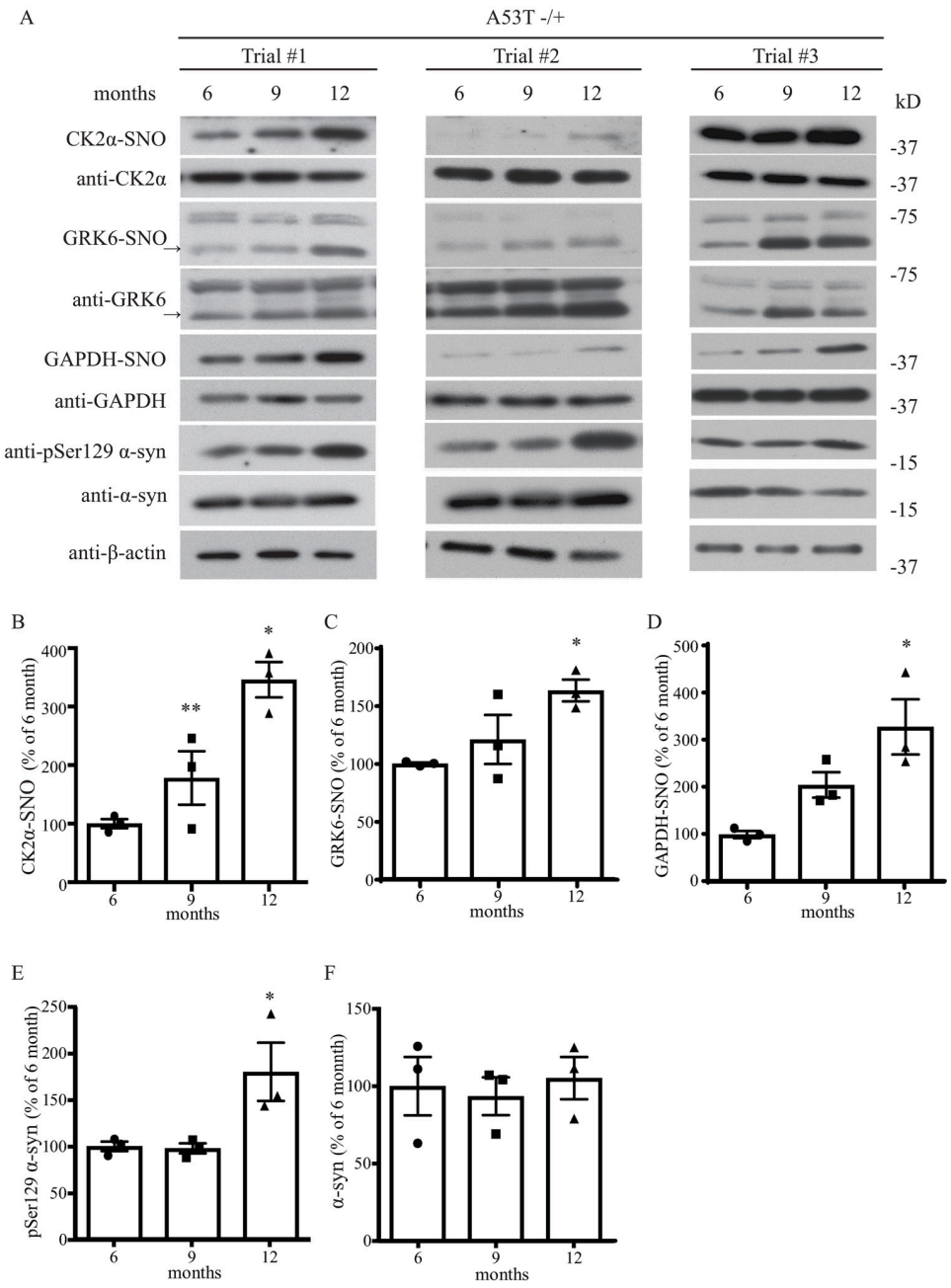

Fig 3. Aging increases GRK6 and CK2α S-nitrosylation in an A53T α-syn transgenic mouse model of PD. (A) Hemizygous A53T α-syn transgenic mouse brain samples of 6, 9 and 12 months old were analyzed with in vivo biotin-switch assay for GRK6, CK2α and GAPDH. The samples were also subject to Western blot analysis of GRK6, CK2α, GAPDH, pSer129 α-syn and α-syn. (→: GRK6 band) (B) Quantification of hemizygous A53T α-syn transgenic mouse brain samples for in vivo CK2α S-nitrosylation as in (A) (* $p < 0.05$, ** $p < 0.01$; # of animals = 3 for each time point; one-way ANOVA with Bonferroni post-hoc test). (C) Quantification of hemizygous A53T α-syn transgenic mouse brain samples for in vivo GRK6 S-nitrosylation as in (A) (* $p < 0.05$; # of animals = 3 for each time point; one-way ANOVA with Bonferroni post-hoc test). (D) Quantification of hemizygous A53T α-syn transgenic mouse brain samples for in vivo GAPDH S-nitrosylation as in (A) (* $p < 0.05$; # of animals = 3 for each time point; one-way ANOVA with Bonferroni post-hoc test). (E) Quantification of hemizygous A53T α-syn transgenic mouse brain samples for protein levels of pSer129 α-syn as in (A) (# of animals = 3 for each time point). (F) Quantification of hemizygous A53T α-syn transgenic mouse brain samples for protein levels of α-syn as in (A) (# of animals = 3 for each time point).

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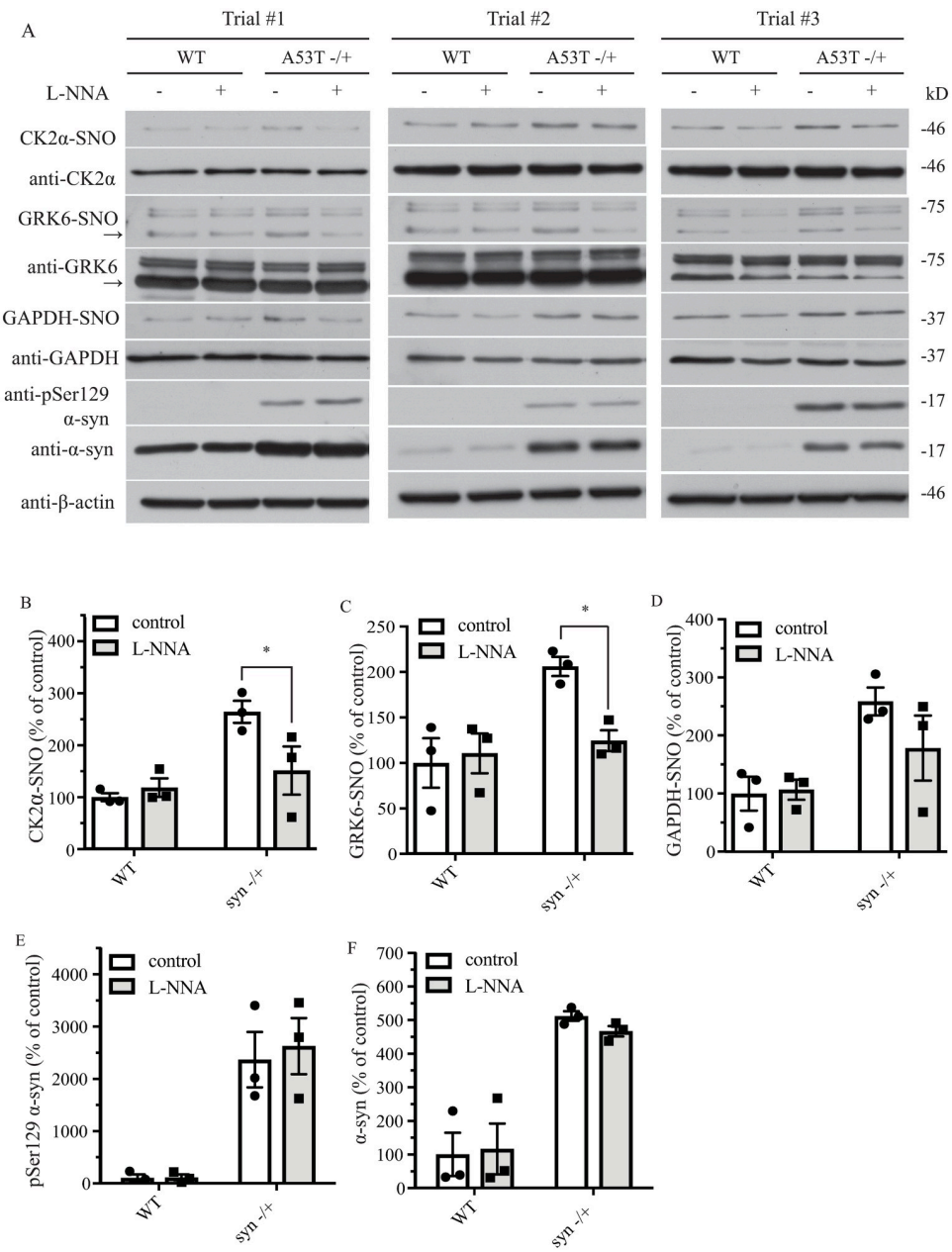

Fig 4. A53T α-syn transgenic expression increases S-nitrosylation of GRK6, CK2α and GAPDH in the mouse brain. (A) WT, and hemizygous A53T α-syn transgenic mouse brain samples of 9 months old treated with or without L-NNA were analyzed with in vivo biotin-switch assay for CK2α and GAPDH. The samples were also subject to Western blot analysis of CK2α, GAPDH, pSer129 α-syn and α-syn. (→: GRK6 band) (B) Quantification of WT and hemizygous A53T α-syn transgenic mouse brain samples for in vivo CK2α S-nitrosylation as in (A) (* $p < 0.05$; no. of animals = 3 in each group; two-way ANOVA with Bonferroni post-hoc test). (C) Quantification of WT and hemizygous A53T α-syn transgenic mouse brain samples for in vivo GRK6 S-nitrosylation as in (A) (* $p < 0.05$; # of animals = 3 in each group; two-way ANOVA with Bonferroni post-hoc test). (D) Quantification of WT and hemizygous A53T α-syn transgenic mouse brain samples for in vivo GAPDH S-nitrosylation as in (A). (E) Quantification of WT and hemizygous A53T α-syn transgenic mouse brain samples for protein levels of pSer129 α-syn as in (A) (# of animals = 3 in each group). (F) Quantification of WT and hemizygous A53T α-syn transgenic mouse brain samples for protein levels of α-syn as in (A) (# of animals = 3 in each group).

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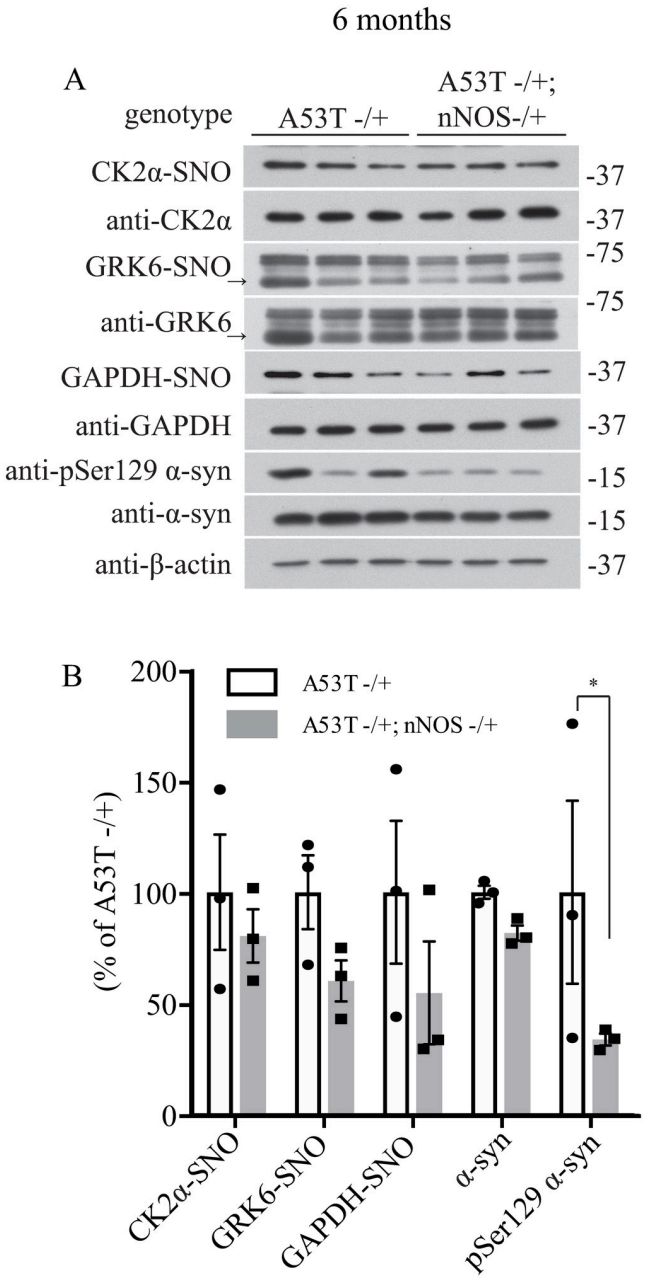

Fig 5. Deletion of neuronal NOS (nNOS) reduces the accumulation of pSer129 α-syn in 6-month-old A53T α-syn transgenic mice. (A) Hemizygous A53T α-syn transgenic mouse brain (A53T -/+), and hemizygous A53T α-syn transgenic and nNOS heterozygous knockout (A53T -/+; nNOS -/+) double mutant mouse brain samples at 6-month-old were analyzed with in vivo biotin-switch assay for CK2α and GAPDH. The samples were also subject to Western blot analysis of CK2α, GAPDH, pSer129 α-syn and α-syn. (→: GRK6 band) (B) Quantification of CK2α, GRK6 and GAPDH S-nitrosylation and protein levels of pSer129 α-syn and α-syn as in (A) (* $p<0.05$; # of animals = 3 in each group; Student's t-test).

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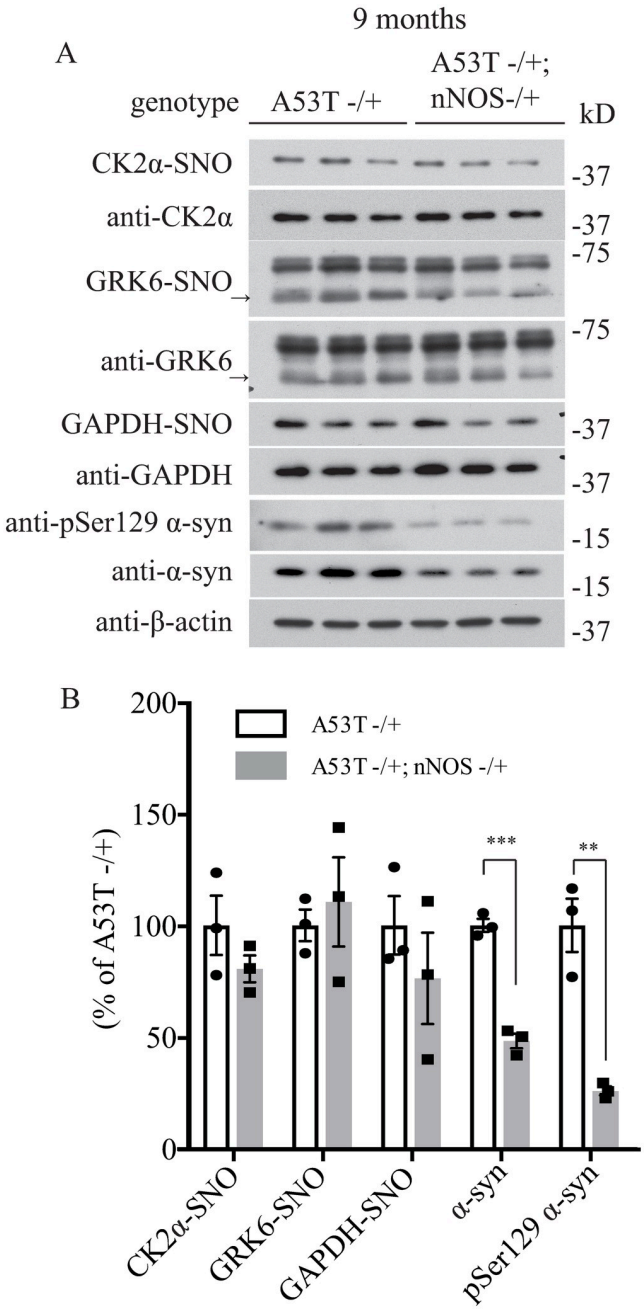

Fig 6. Deletion of neuronal NOS (nNOS) reduces the accumulation of pSer129 α-syn and total α-syn in 9-month-old A53T α-syn transgenic mice. (A) Hemizygous A53T α-syn transgenic mouse brain (A53T -/+), and hemizygous A53T α-syn transgenic and nNOS heterozygous knockout (A53T -/+; nNOS -/+) double mutant mouse brain samples at 9-month-old were analyzed with in vivo biotin-switch assay for CK2α and GAPDH. The samples were also subject to Western blot analysis of CK2α, GAPDH, pSer129 α-syn and α-syn. (→: GRK6 band) (B) Quantification of CK2α, GRK6 and GAPDH S-nitrosylation and protein levels of pSer129 α-syn and α-syn as in (A) (*** $p<0.001$; ** $P<0.01$; # of animals = 3 in each group; Student's t-test).

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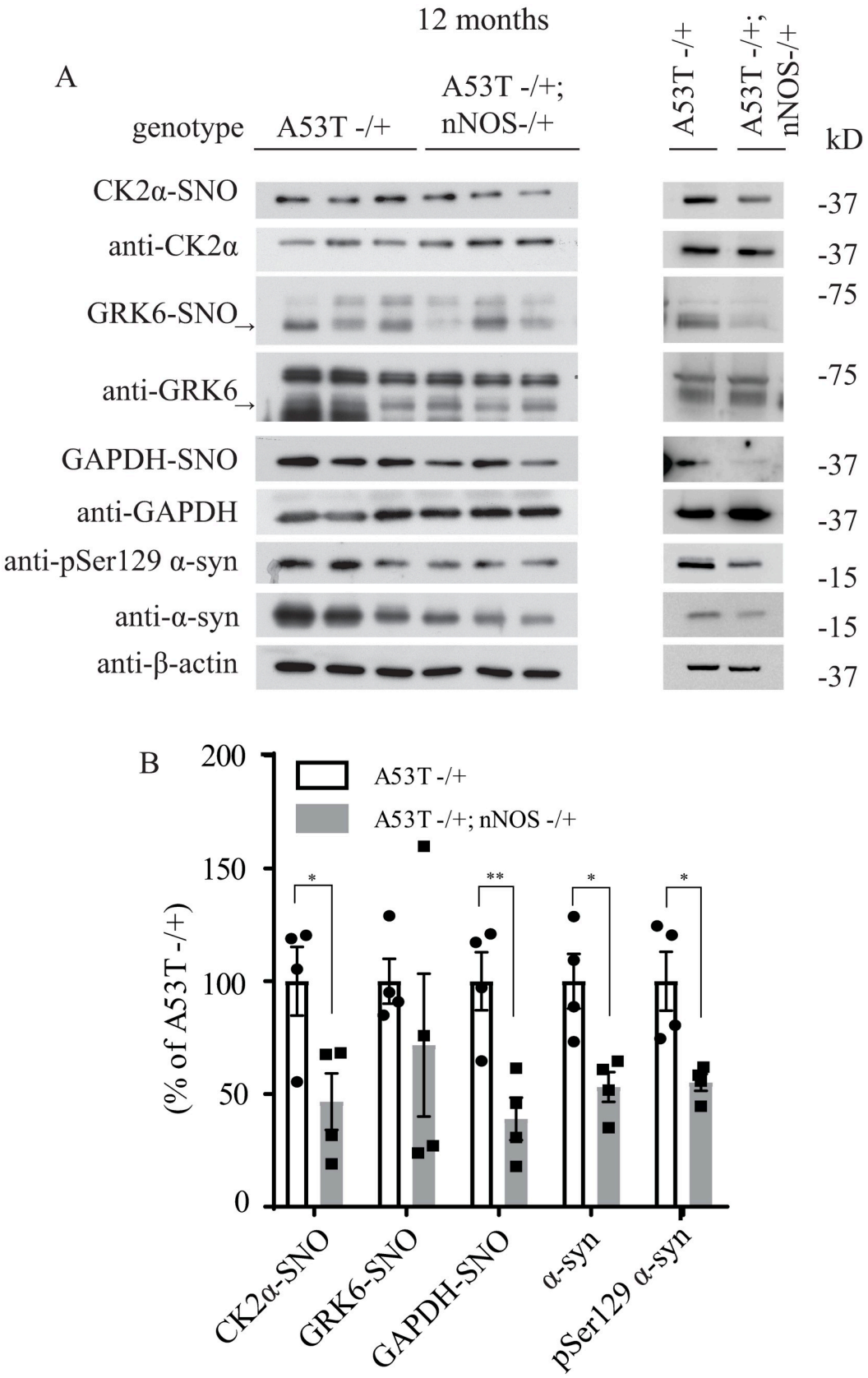

Fig 7. Deletion of neuronal NOS (nNOS) reduces the accumulation of pSer129 α-syn and total α-syn in 12-month-old A53T α-syn transgenic mice. (A) Hemizygous A53T α-syn transgenic mouse brain (A53T -/+), and hemizygous A53T α-syn transgenic and nNOS heterozygous knockout (A53T -/+; nNOS -/+) double mutant mouse brain samples at 12-month-

old were analyzed with in vivo biotin-switch assay for CK2α and GAPDH. The samples were also subject to Western blot analysis of CK2α, GAPDH, pSer129 α-syn and α-syn. (→: GRK6 band) (B) Quantification of CK2α, GRK6 and GAPDH S-nitrosylation and protein levels of pSer129 α-syn and α-syn as in (A) (* $p<0.05$; ** $p<0.01$; # of animals = 4 in each group; Student's t-test).

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Reference

1. Wu W, Sung CC, Yu P, Li J, Chung KKK (2020) S-Nitrosylation of G protein-coupled receptor kinase 6 and Casein kinase 2 alpha modulates their kinase activity toward alpha-synuclein phosphorylation in an animal model of Parkinson's disease. PLoS ONE 15(4): e0232019. https://doi.org/10.1371/journal.pone.0232019 PMID: 32343709