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Corrigendum

Corrigendum to "Discovery of novel interacting partners of PSMD9, a proteasomal chaperone: Role of an Atypical and versatile PDZ-domain motif interaction and identification of putative functional modules" [FEBS Open Bio 4 (2014) 571–583]

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The hnRNPA1 construct used in this paper was the short form (F320G) and not the long form (F372G) as previously stated.

A corrected version of Fig. 1 and Supplementary Tables S4 and S6 is provided here.

The figure legend to Fig. 1 should read: "(B) Recombinant WT hnRNPA1 or hnRNPA1 C-terminal mutant (F320G or CΔ7)".

In addition, the text on p. 573, Results, 3.1, 11 lines from bottom should read: "... we cloned and expressed the shorter isoform of hnRNAP1 as a GST fusion protein ..."

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.fob.2015.06.010.

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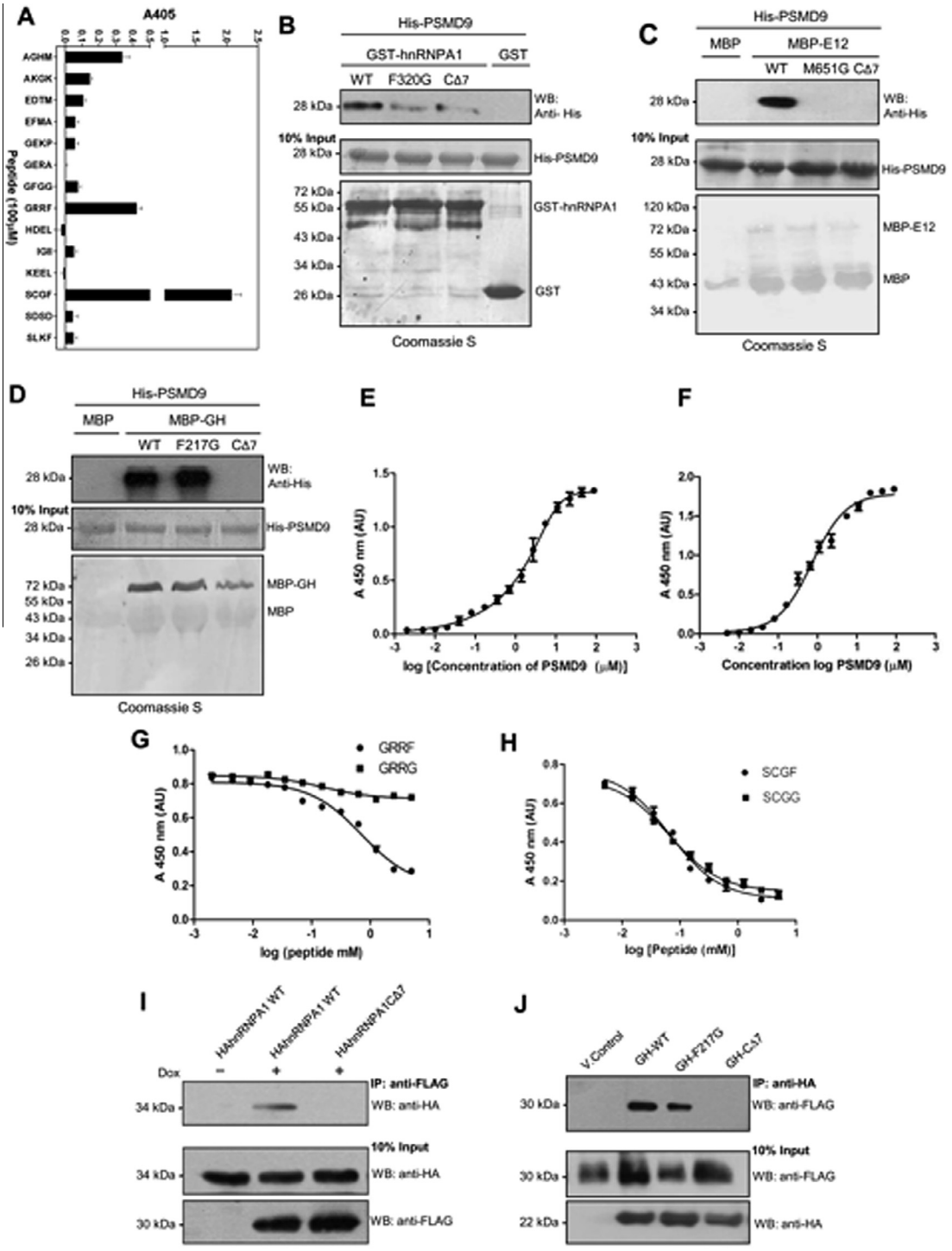

Fig. 1. Identification of putative interacting partners of PSMD9, and the importance of C-terminal residues in interaction. (A) Conserved C-terminal motif s in the form of tetra peptides were tested for binding to PSMD9 using ELISA (see Section 2 for details). Values represent mean ± SEM (Standard Error of Mean) from three different experiments performed in duplicates.(B) Recombinant WT hnRNPA1 or hnRNPA1 C-terminal mutant (F320G or CD7) bound to GST served as baits to pull down PSMD9. (C) Interaction of recombinant E12 and its C-terminal mutants (MBP-fusions) with PSMD9 (His-tag) were tested by *in vitro* affinity pull-down using MBP-agarose (see Section 2 for details). (D) Interaction of recombinant GH and its C-terminal mutants (MBP fusions) with PSMD9 was tested by in vitro affinity pull-down using MBP-agarose (see Section 2 for details). (E) Interaction of PSMD9 with hnRNPA1 was monitored by ELISA (see Section 2 for details). Data were best fit to one site specific binding using GraphPad Prism (commercial software, www.graphpad.com). The dissociation constant (K_d) for the interaction was found to be 1.33 ± 0.04 μM for hnRNPA1. Data from two independent experiments each done in duplicates is represented as mean ± SD (SD-standard deviation). (F) Interaction of PSMD9 with growth hormone. Data were fit to one site specific binding using PRISM. The dissociation constant (K_d) for the interaction was found to be 0.84 ± 0.07 μM for growth hormone. Measurements were done in duplicates and data is represented as mean ± SD (SD-standard deviation) for two independent experiments. (G) C-terminal peptide GRRF inhibits hnRNPA1-PSMD9 interaction. Prior to its incubation with hnRNPA1 coated plates, PSMD9 (0.65 μM) was incubated with GRRF or GRRG peptides. (H) C-terminal peptide SCGF and SCGG inhibit interaction of growth hormone with PSMD9. Prior to incubation with growth hormone, PSMD9 (0.65 μM) was incubated with SCGF or SCGG peptides. Ki for SCGF was calculated to be 36.7 ± 0.29 μM and for SCGG, it was 35.6 ± 0.24 μM. Data from two independent experiments each done in duplicates is represented as mean ± SD. (I) Interaction of hnRNPA1 and PSMD9 in mammalian cells. FLAG-tagged PSMD9 or its C-terminal mutant and HA- tagged hnRNPA1 were co-expressed in HEK293 cells. FLAG-PSMD9 was immunoprecipitated using M2-Agarose beads, followed by Western blot with anti-HA antibody. (J) Growth hormone and PSMD9 interact upon co-expression in mammalian cells. HA-Growth hormone or its C-terminal mutants and FLAG-PSMD9 were co-expressed in HEK293 cells and interaction was monitored by Co-IP as described in Supplementary methods.