Cellular Physiology	Cell Physiol Biochem 2020;54:798	
and Biochemistry	DOI: 10.33594/000000260	© 2020 The Author(s) Published by Cell Physiol Biochem Press GmbH&Co. KG, Duesseldorf www.cellphysiolbiochem.com

Erratum

In the original article by Centrone, et al., entitled "AQP2 Abundance is Regulated by the E3-Ligase CHIP Via HSP70" [Cell Physiol Biochem 2017;44(2):515-531, DOI: 10.1159/000485088], Fig. 1 does not correspond exactly to the description reported in the result section and in its figure legend. Specifically, while the data regarding the coimmunoprecipitation in MCD4 cells is described, this data is missing in Figure 1. The published Figure 1 contains only the results obtained in renal tissue.

The correct Fig. 1 and its correct legend are displayed below. The authors confirm that all of the results and conclusions of the article remain unchanged.

Additionally, there has been a mistake in the Discussion section on page 526, which should be corrected as follows: "In renal cells, vasopressin stimulation causes ERK activation [59,60] as ERK inhibition with U0126 impaired the vasopressin-induced AQP2 expression [59]. In contrast, more recent data showed that vasopressin reduces the level of ERK phosphorylation which has been shown to phosphorylate AQP2 at S261 [42]."

The authors sincerely apologize for the mistakes.

Fig. 1. AQP2 and CHIP co-immunoprecipitation in MCD4 cells and renal tissue. AQP2 was immunoprecipitated (I.P. AQP2) using pre-C-tail antibodies from MCD4 cells (A) and from fresh mouse kidney slices (B). Whole cell lysates were also subjected to precipitation with only protein A-coupled Sepharose (ProtA) or with nonspecific IgG. Immunocomplexes were analyzed by immunoblotting with antibodies specific for CHIP; anti-AOP2 antibodies were used as control of the immunoprecipitation experiments. The obtained results indicate that AQP2 can complex with CHIP in MCD4 cells and in renal tissue.

