CORRIGENDUM

DOI: 10.3892/mmr.2018.9479

Neurotoxin β-N-methylamino-L-alanine induces endoplasmic reticulum stress-mediated neuronal apoptosis

HAIYING SHEN, KIYOON KIM, YOOJUNG OH, KYUNG SIK YOON, HYUNG HWAN BAIK, SUNG SOO KIM, JOOHUN HA, INSUG KANG and WONCHAE CHOE

Mol Med Rep 14: 4873-4880, 2016; DOI: 10.3892/mmr.2016.5802

Following the publication of the above article and a Corrigendum published in July 2018, the authors have noted an additional error, associated with the presentation of Fig. 1C. Fig 1C showed that β-N-methylamino-L-alanine induces neuronal apoptotic cell death; however, an error was made in the compilation of this figure and an incorrect band image was selected for α-actinin, the loading control panel for Fig. 1C. A corrected version of Fig. 1 is shown opposite, incorporating the correct α-actinin protein bands in Fig. 1C. This change affects neither the interpretation of the data nor conclusions of this work.

We regret that this further error went unnoticed at the time, and thank the Editor for allowing us the opportunity to publish this additional Corrigendum.

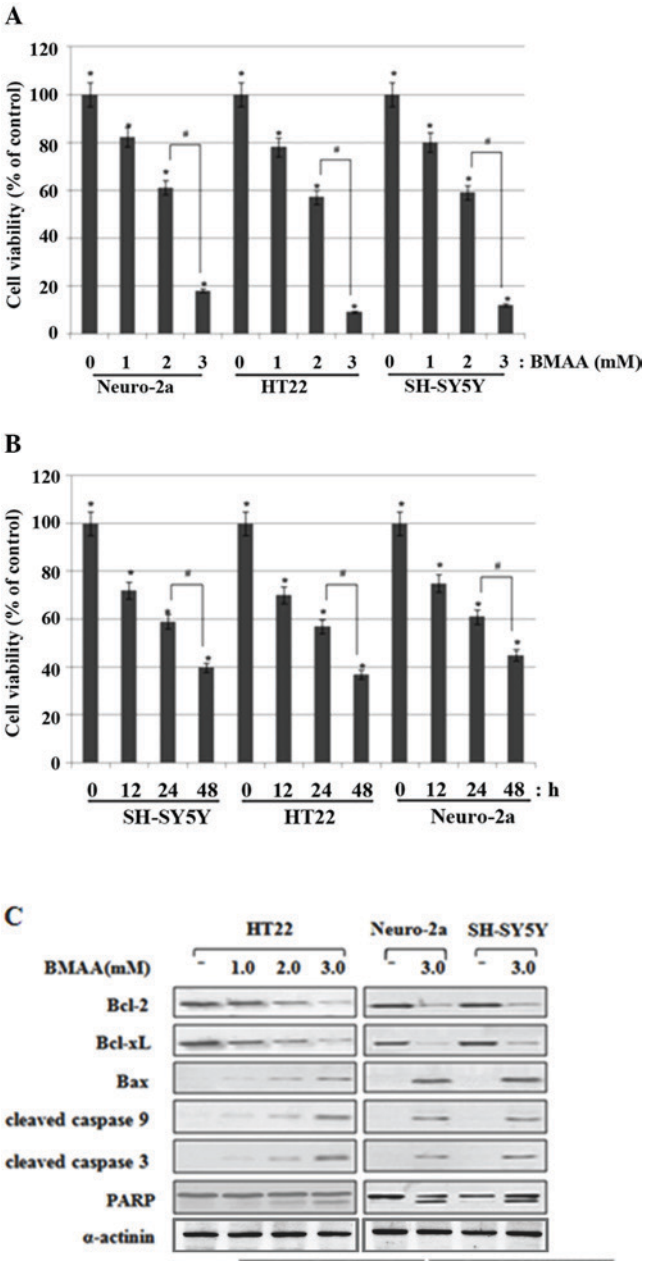

Figure 1. Effects of BMAA on cell viability and apoptosis of neuronal cells. (A) Neuronal cells were treated with 1.0, 2.0, or 3.0 mM BMAA and were analyzed at 24 h after the beginning of exposure to BMAA. The percentage cell viabilities are representative of at least three different experiments and are expressed as the mean ± standard deviation (*P<0.05 vs. untreated cells; $^{\#}$P<0.05 vs. 2.0 mM-treated cells). (B) The cell viabilities were measured at 12, 24 and 48 h after treatment with 2.0 mM BMAA. The data are expressed as the mean ± standard deviation obtained from at least three independent experiments (*P<0.05 vs. untreated cells; $^{\#}$P<0.05 vs. 24 h-treated cells). (C) The cells were treated with increasing doses of BMAA (1.0, 2.0 and 3.0 mM) for 24 h. The protein expression levels of Bcl-2 family members, and the cleavage of caspase and PARP, were assessed by western blot analysis. α-actinin was used as a loading control. The data are representative of at least three different experiments. BMAA, β-N-methylamino-L-alanine; Bcl, B-cell lymphoma; Bax, Bcl-2-associated X protein; PARP, poly-ADP ribose polymerase.