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HMGB1 promotes ductular reaction and tumorigenesis in autophagy-deficient livers

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Expression of concern

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Corrigendum

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The Editors posted an Expression of Concern for this article following notification that an investigative committee at the University of Liverpool had data integrity concerns regarding the mass spectrometry data contributed by Daniel J. Antoine and shown in Figure 1G of this paper. The authors have provided a corrected version of this article and a description of changes below.

In our published work, we reported that HMGB1 is actively released from autophagy-deficient hepatocytes via a pathway from NRF2 to inflammasomes to promote ductular reaction, hepatic progenitor cell expansion, and tumorigenesis. We based our conclusions on multiple lines of evidence. Release of HMGB1 from autophagy-deficient hepatocytes was documented by immunoblotting, immunostaining, and ELISA analysis in different age groups of autophagy-deficient mice. The release of HMGB1 through an active mechanism is supported by kinetics analysis that shows tissue injury can be separated from the release process and by pharmacological and genetic analyses showing that the molecular elements of NRF2 and CASPASE 1 are required. The impact of HMGB1 on ductular reaction and tumor progression was also documented by both in vivo and in vitro evidence using knockout mice, cell fractionation, and transcriptional analysis.

Figure 1G showed the results of an analysis of HMGB1 isoforms by mass spectrometry that was undertaken in a separate laboratory by Daniel J. Antoine. In February 2019, we learned that these data were likely compromised. We contacted the journal, and the Editorial Board gave us permission to correct the study. In the corrected version, all conclusions based on Figure 1G have been removed, and the journal has published an online version of the original article with the unreliable statements crossed out and the modified text highlighted in red (Supplemental File, Redaction). Figure 1G only suggested the formation of the released HMGB1, but carried no significance as to the releasing mechanisms and the functional significance of HMGB1 release in autophagy-deficient conditions. We thus believe that the major conclusions of the study on the releasing mechanism and functional significance of HMGB1 in autophagy-deficient conditions are independent of Figure 1G and are accurate and that the corrected paper is reliable.