Correction: Degradation of HK2 by chaperone-mediated autophagy promotes metabolic catastrophe and cell death

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It recently came to the authors' attention that the tubulin loading control panels shown in Figs. 2 G and S2 F were incorrect as a result of errors introduced during figure preparation. The authors apologize for these mistakes. The conclusions of the experiments shown in these figures are not affected.

A corrected version of Fig. 2 is shown below. The LC3 panel originally published in the top part of panel G was correct and has not been changed. The images used to assemble the top half of panel G as well as one replicate data set were provided to the editors for assessment. No other error in figure preparation has been detected in Fig. 2 G.

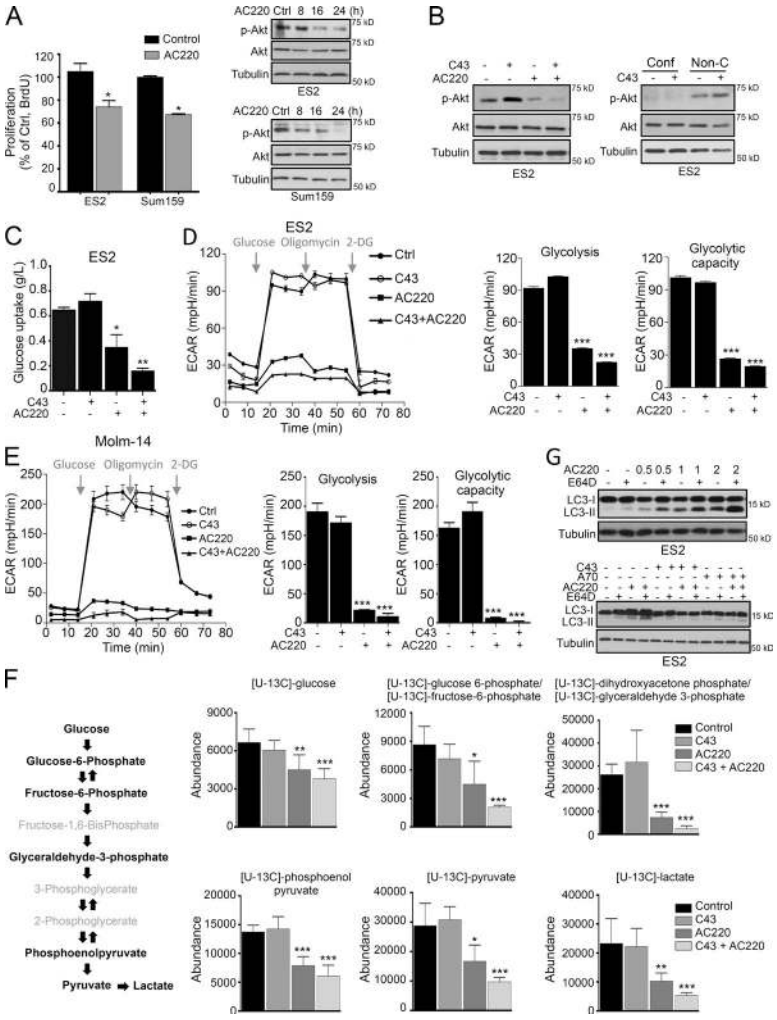

Figure 2. **Treatment with AC220 (Quizartinib) reduces glycolysis and induces macroautophagy.** (A) Proliferation capacity (%) of ES2 and Sum159 cells treated with AC220 for 16 h. Phospho- and total Akt levels of ES2 and Sum159 cells treated with AC220 up to 24 h. (B) WB of phospho- and total Akt levels of ES2 cells treated with AC220 and C43 (left), or C43 alone in confluent (Conf) or nonconfluent (Non-C) conditions (right) for 24 h. (C) Relative change in glucose levels in the culture medium of ES2 cells treated with AC220 and/or C43 (normalized to cell numbers) for 16 h. (D and E) The glycolytic activity and maximum glycolytic capacity of ES2 (D) or Molm-14 (E) cells, determined by ECAR, after AC220 and C43 treatment for 12 h (ES2) or 8 h (Molm-14). (F) Glucose flux analysis using [U^{13}C]glucose. A schematic depiction of intermediary metabolites of glycolysis is shown. ^{13}C enrichment of intracellular glucose-derived metabolites, marked in bold, is presented. (G) WB of LC3 protein levels in ES2 cells, treated with increasing concentrations of AC220 and/or 5 µM E64D for 16 h (top), or treated with C43 or A70 in combination with AC220 and/or 5 µM E64D for 24 h. Anti–α-tubulin was used as a loading control. Cells were treated with 0.1% DMSO (control: vehicle), 1 µM AC220, 10 µM C43, or 1 µM A70, unless otherwise stated. In all the experiments, treatment groups were compared with the control group, unless otherwise shown. Error bars indicate ±SD. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

A corrected version of Fig. S2 is shown below.

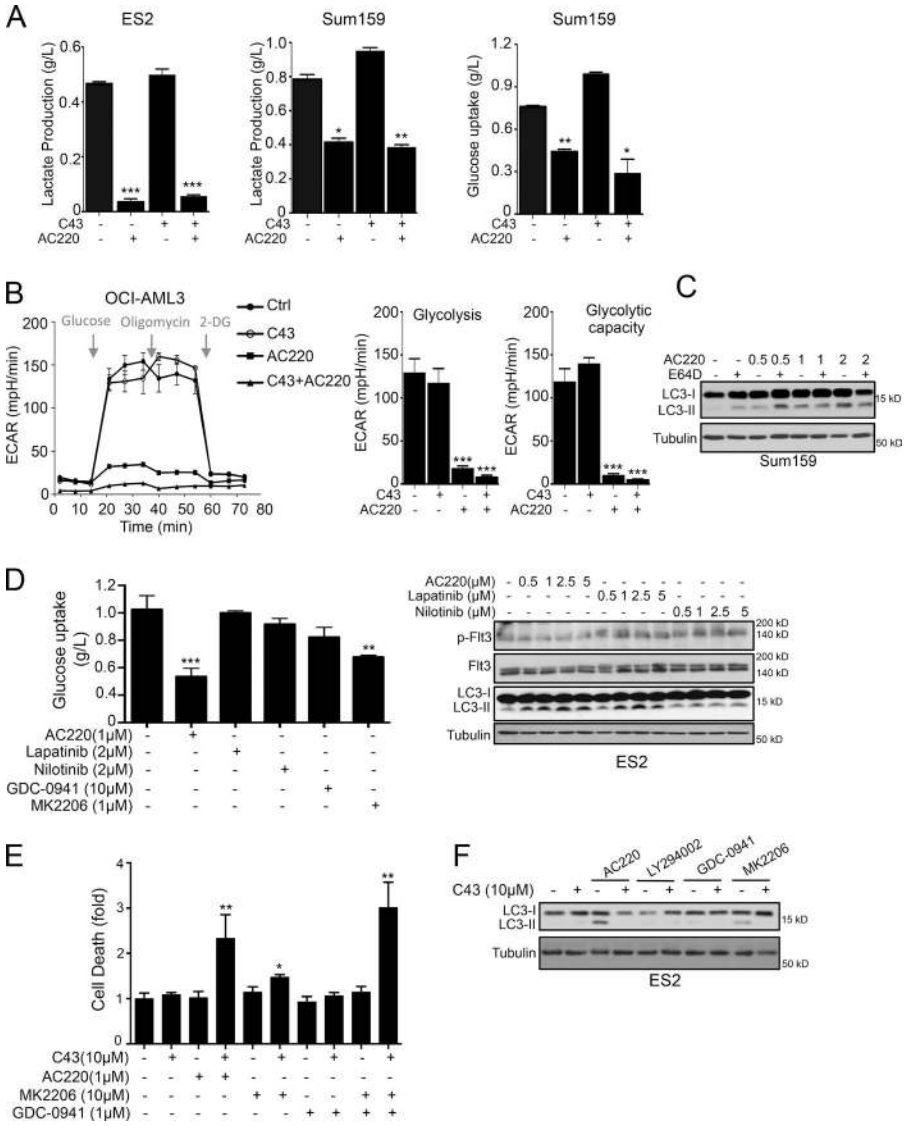

Figure S2. **The effect of combination treatment of AC220 and spautins on metabolism and cellular growth pathways.** (A) Relative changes of glucose or lactate levels in the cell medium of ES2 and Sum159 cells treated with AC220 and/or C43 (normalized to cell numbers) for 16 h. (B) The glycolytic activity and maximum glycolytic capacity of OCI-AML3 cells, determined by ECAR, after AC220 and C43 treatment for 8 h. (C) WB of LC3 protein levels in Sum159 cells, treated with increasing concentrations of AC220 in the presence or absence of 5 μM E64D for 16 h. (D) Relative changes of glucose levels in the culture medium of ES2 cells treated with the indicated inhibitors, and phospho- and total FLT3, LC3 protein levels in ES2 cells treated with increasing concentrations of AC220, Lapatinib, or Nilotinib for 16 h. (E) Cell death (fold) of ES2 cells treated with AC220, MK2206, or GDC-0941 for 24 h. (F) LC3 protein levels in ES2 cells treated with indicated inhibitors for 16 h. Anti–α-tubulin was used as a loading control. Cells were treated with 0.1% DMSO (control: vehicle) or 1 μM AC220 and 10 μM C43, unless otherwise stated. In all the experiments, treatment groups were compared with the control group, unless otherwise indicated. Error bars indicate ±SD. *, $P < 0.05$; **, $P < 0.01$; ***, $P < 0.001$.

Lastly, it came to the authors' attention that the tubulin panel shown in Figs. 1 E and 3 B looked similar to a tubulin panel shown in another publication from the lab. The data in the *JCB* paper were double checked and all the panels shown in Figs. 1 E and 3 B were assembled correctly. No modification of Figs. 1 E and 3 B is needed.

The HTML and PDF versions of this article have been corrected. The error remains only in the print version.