


CORRECTION

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# Correction to: Pyroptosis: a new paradigm of cell death for fighting against cancer

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**Correction to: J Exp Clin Cancer Res 40, 153 (2021)**  
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Following publication of the original article [1], the authors identified minor errors in image-typesetting in Fig. 1; specifically the GzmA and GzmB labels had been transposed.

The corrected figure is given here. The correction does not have any effect on the results or conclusions of the paper. The original article has been corrected.

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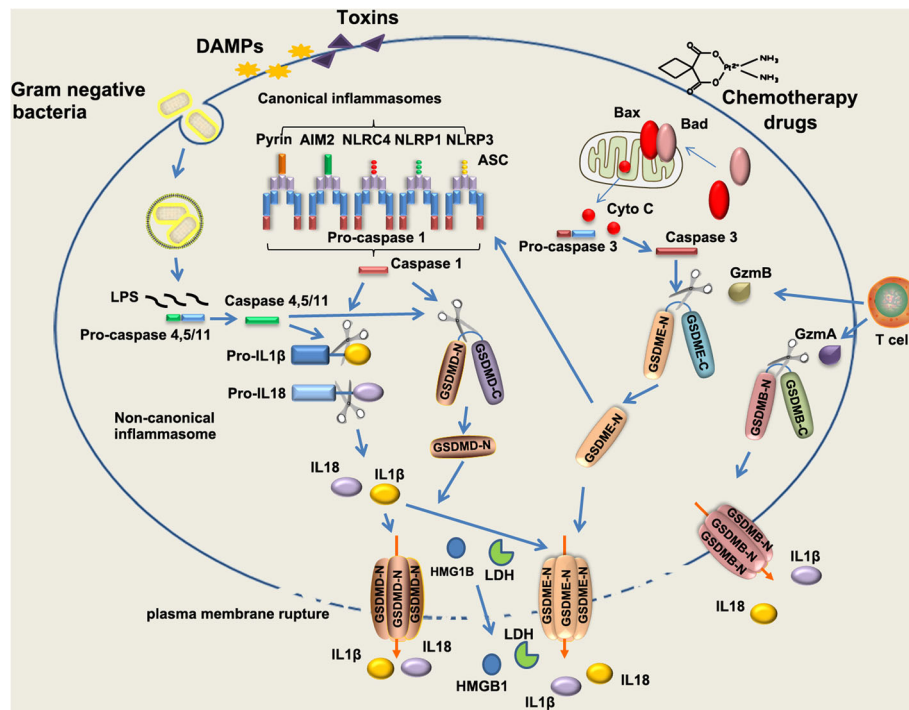
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**Fig. 1** The canonical inflammasome and non-canonical inflammasome pathway in pyroptosis. The canonical inflammasome is assembled in response to exogenous pathogens and endogenous damage by intracellular sensor proteins, including NLRP1b, NLRC4, NLRP3, AIM2 and Pyrin. The canonical inflammasomes recruit pro-caspase 1 through inflammasome adaptor protein ASC, leading self-cleavage and activation of caspase 1. Active caspase 1 cleaves pro-inflammatory cytokines pro-IL-1 $\beta$ , pro-IL-18, leading to maturation of IL-1 $\beta$ , IL-18. Active caspase 1 cleaves GSDMD protein at the middle linker, liberating the cytotoxic N-terminus to form pore on plasma membrane, which allows the release of mature IL-1 $\beta$ , IL-18. In non-canonical pathway, LPS directly binds to murine pro-caspase 11 or its human homologs pro-caspase 4 and 5, leading activation of caspase 11/4/5. In non-canonical inflammasome pathway, cleavage of GSDMD is executed by active caspase 11 or caspase 4 and 5 upon direct binding of cytosolic LPS. Chemotherapy drugs could induce pyroptosis in epithelial cells through activating mitochondrial death machinery and caspase 3. In this case, GSDME is cleaved by active caspase 3. GSDME-N in turn activates NLRP3 inflammasome, leading to activation of caspase 1/GSDMD cascade, which promotes maturation of IL-1 $\beta$ , IL-18. Gasdermins could be cleaved by Lymphocyte-derived granzymes proteases, unleashing the pore-formation ability to trigger pyroptosis of cancer cells