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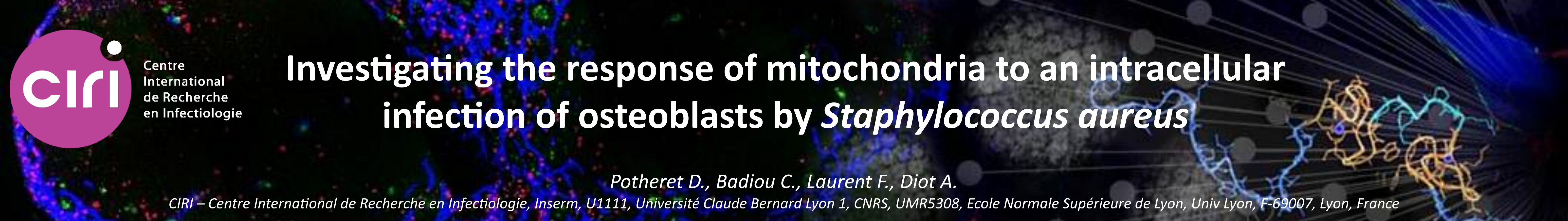
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Investigating the response of mitochondria to an intracellular infection of osteoblasts by *Staphylococcus aureus*

Potheret D., Badiou C., Laurent F., Diot A.

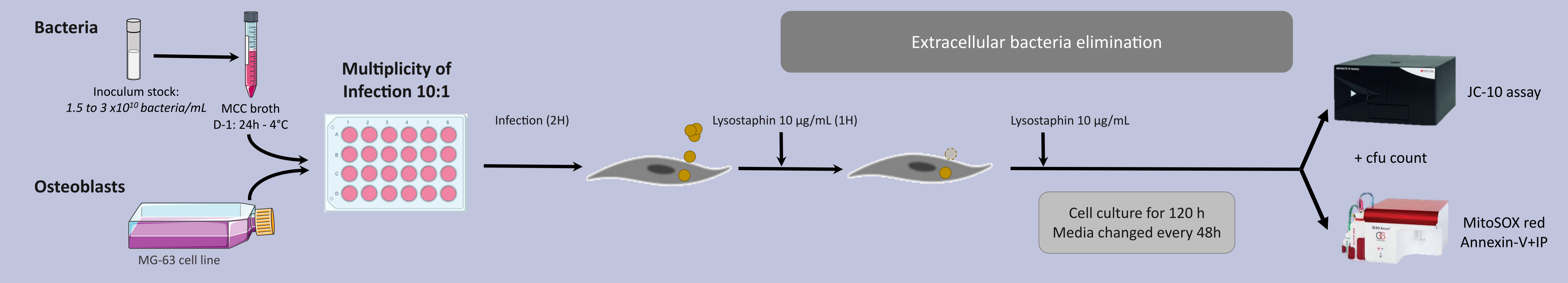
CIRI – Centre International de Recherche en Infectiologie, Inserm, U1111, Université Claude Bernard Lyon 1, CNRS, UMR5308, Ecole Normale Supérieure de Lyon, Univ Lyon, F-69007, Lyon, France

Background & Objective

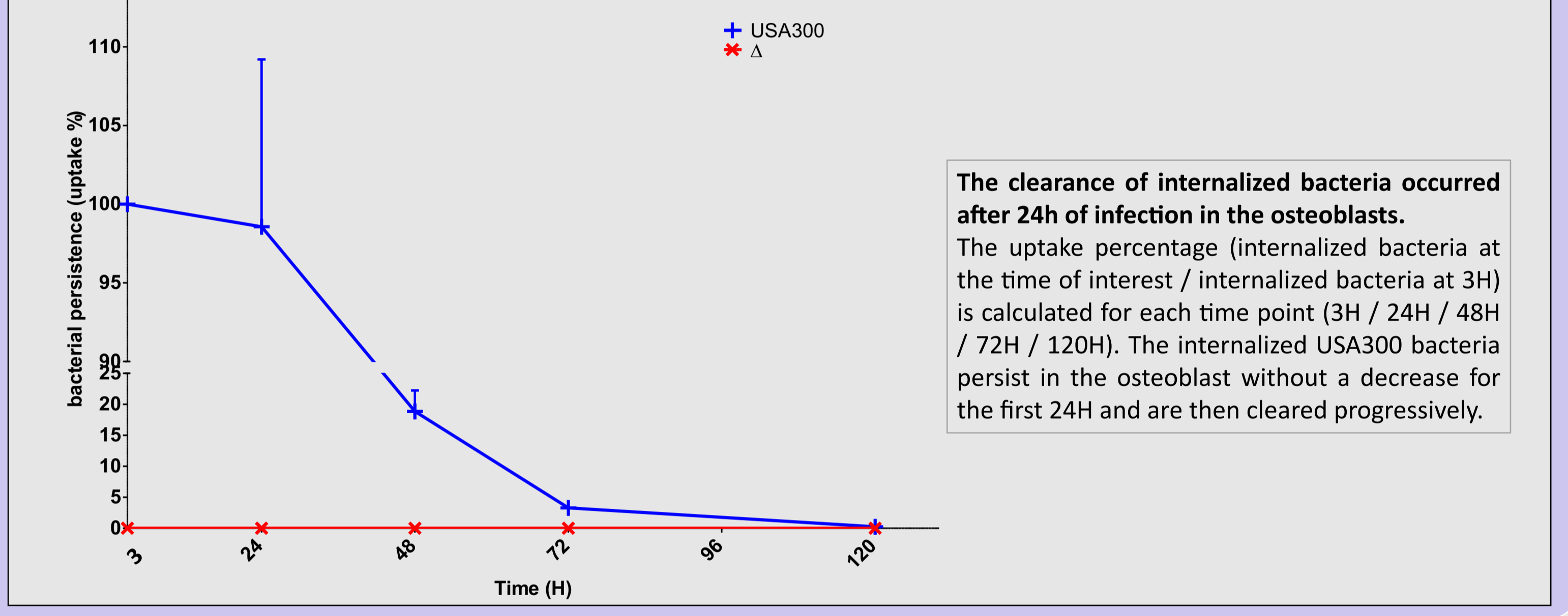
Staphylococcus aureus (SA) is an opportunistic pathogen responsible for difficult-to-treat infections, among which bone and joint infections (BJI). Three main factors that protect SA from the immune system and antibiotics are responsible for the large number (10-20%) of relapses observed during BJI treatments: biofilm formation, small colony variant phenotype and non-professional phagocyte cells invasion. SA inside osteoblasts have already been observed during a persistent case of BJI. Once internalized, SA is found in phago-endosomal vesicle where it can either persist or escape to induce cell death, notably due to mitochondria-dependent apoptosis. The response of mitochondria to an intracellular SA infection has been poorly studied so far. However, the few studies available suggest that SA can trigger oxidative damages together with mitochondria biogenesis and, in the case of a virulent strain, apoptosis.

Our objective is to observe the mitochondrial behavior during the internalization of SA USA300 SF8300 (USA300) into osteoblasts, and compare it to the one triggered by the contact between the internalization-deficient mutant USA300 SF8300 Δ fnbPs (Δ) and osteoblasts.

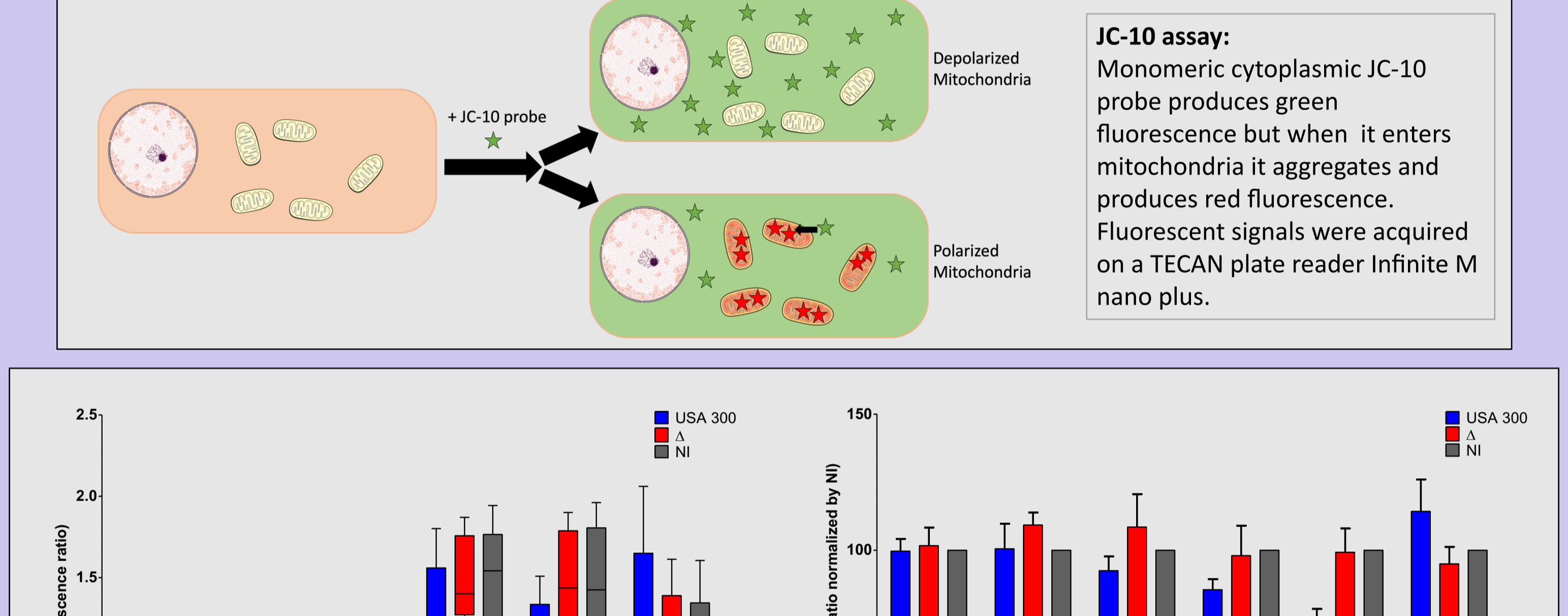
Methods



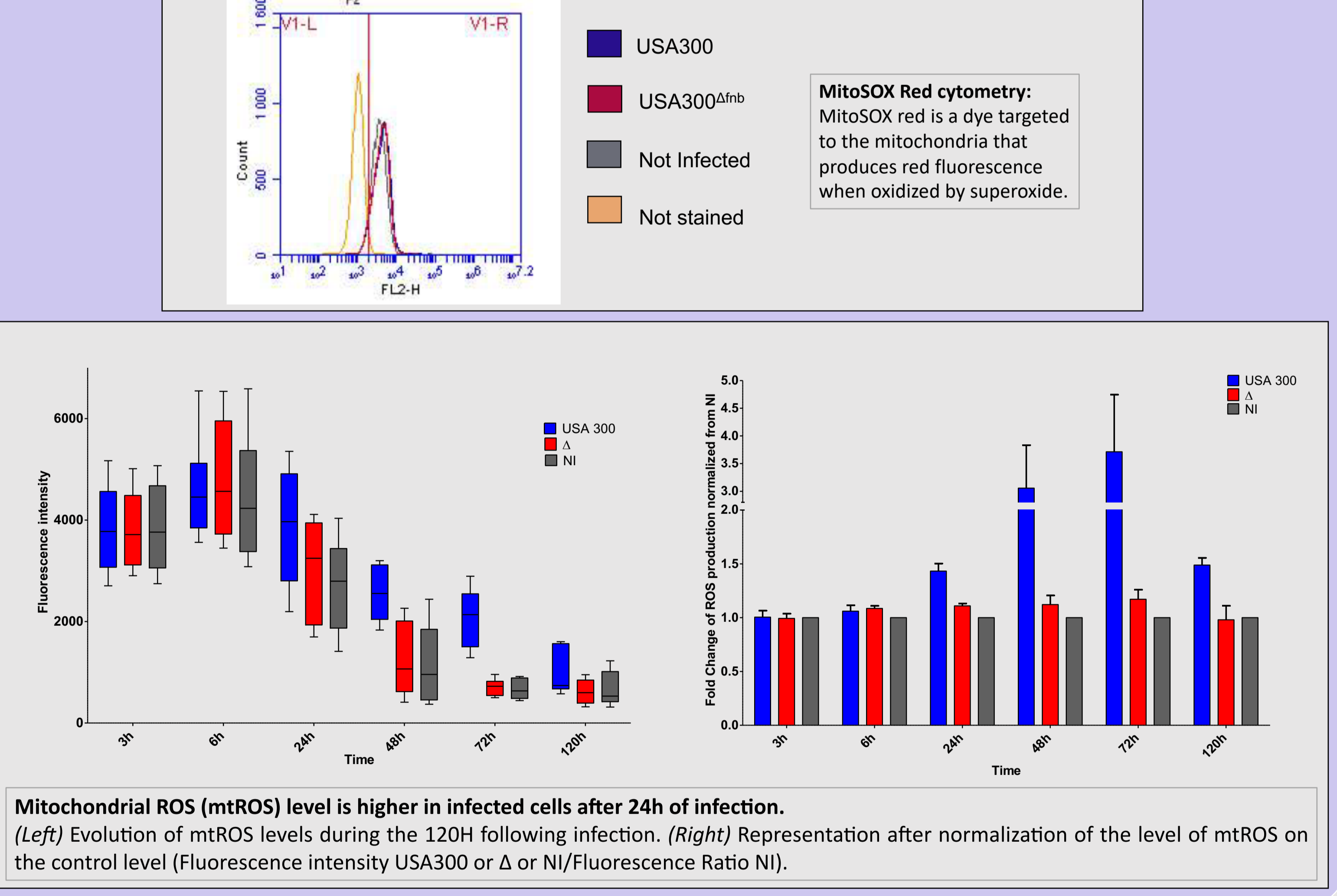
Results (1) The majority of internalized USA300 persists 24H inside MG-63 cells



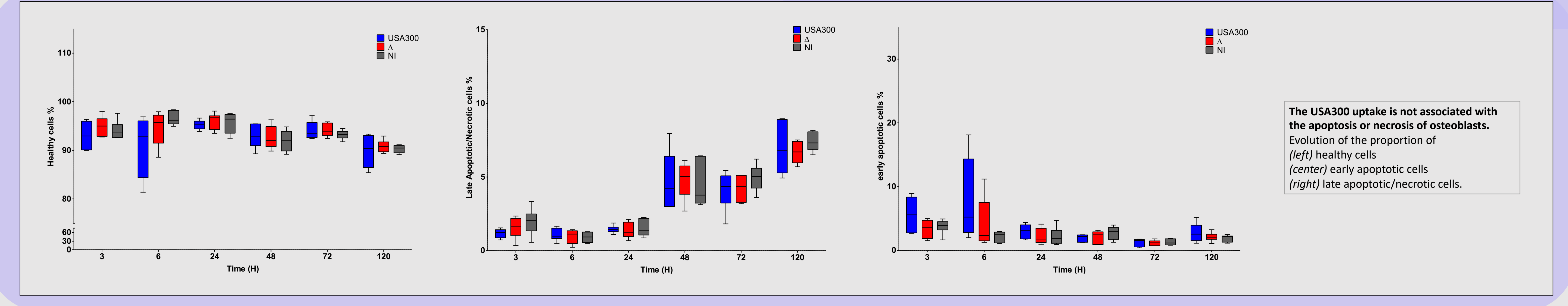
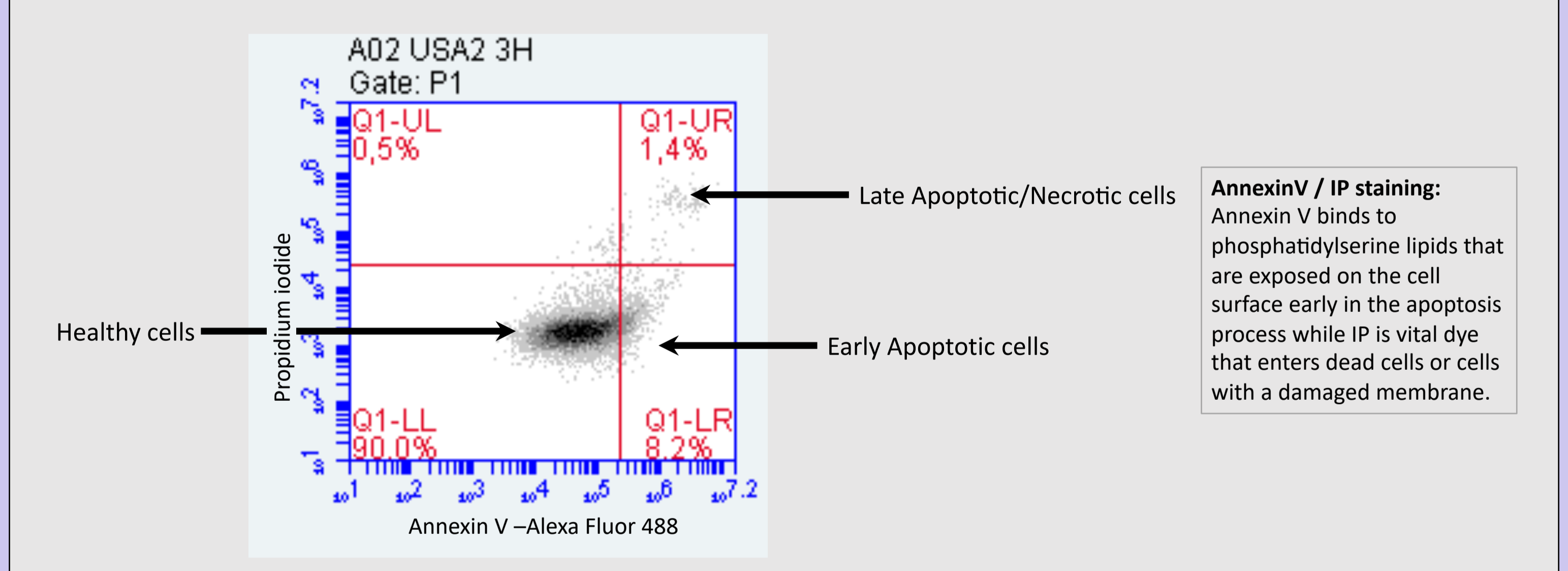
Results (2) USA300 internalization induces a decrease of MMP in MG-63 cells



Results (3) USA300 induces ROS production after 24H of infection



Results (4) Infection by USA300 does not trigger MG-63 apoptosis



Conclusions

These results indicate that when bacteria find a shelter inside a host cell, this might trigger mtROS production/signalisation to clear the pathogen. However, these data are a first step towards the understanding of the mitochondrial response to an intracellular infection. We are currently studying the anti-oxidative response of the cell as well as the mitochondrial network morphology. Finally, we will assess how the modulation of mtROS production might affect the intracellular bacterial persistence in order to appreciate if this could be a new therapeutic strategy to develop.