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Título	Crosstalk between cell death, autophagy and senescence in lung cancer cells under chemotherapy treatment
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Background: In cancer biology, chemotherapy-induced DNA damage not only leads to cell death but also triggers autophagy and senescence in tumor cells. These varied cellular responses are driven by intratumor heterogeneity, influencing whether cancer cells survive or die, and ultimately impacting patient prognosis. **Aims:** This study investigates the correlation between cell death, autophagy, and senescence induced by chemotherapeutics commonly used in the treatment of non-small cell lung cancer. **Methods:** A549 human lung adenocarcinoma cells were treated with 10μM and 40μM Cisplatin (Cis) and 6.6μM Etoposide (Etop) for 48 hours. Following treatment, cells were cultured in drug-free medium for 14 days to the analysis of Cumulative Population Doubling (CPD), autophagy, and senescence. **Results:** A significant decrease in cell viability was observed after 48 hours of treatment across all groups, with Cisplatin 40μM having the most pronounced effect. CPD analysis revealed differential impacts on cell proliferation, with Cisplatin treatments leading to lower CPD compared to the Control and Etoposide. Autophagy analysis showed a temporary increase in cells that survived the acute stress, suggesting a potential role of autophagy in chemotherapy resistance. Senescence levels also varied over time, with Cisplatin inducing higher levels of senescence between 5 to 10 days after treatments. Etoposide induced autophagy in the early days following treatment, alongside nuclear and cellular changes indicative of quiescence rather than senescence. **Conclusion:** Autophagy and senescence are closely linked to the mechanisms of resistance to cell death following chemotherapy. Understanding these mechanisms could contribute to the development of more effective cancer therapies.