

International Conference on

CANCER THERAPY &

International Conference on

VACCINES & VACCINATION

July 23-24, 2018 | Osaka, Japan

LCZ 696 is superior to Valsartan in the prevention of cardiotoxicity induced by trastuzumab, pertuzumab and Trastuzumab-DM1 monoclonal antibodies

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Cardiotoxic effects related to anticancer drugs are among the leading causes of morbidity and mortality in cancer patients treated with Trastuzumab (T), Pertuzumab (P) and Trastuzumab-DM1 (TDM1) [1-3]. Sacubitril-valsartan (LCZ 696), a drug used for the treatment of heart failure in patients with a reduced ejection fraction, is a combination drug, made up of neprilysin inhibitor sacubitril and angiotensin II receptor blocker valsartan. In this study, we aim to assess whether LCZ 696, administered during T, P or TDM1 treatment, reduces in vitro anticancer drugs-related cardiotoxicity, more efficiently respect to Valsartan (V). We used our in Vitro model, the H9C2 rat cardiomyoblasts, treated with 200 nM of T, P or TDM1 for 3 days, and then treated in the absence or presence of 10 μ M of LCZ 696 or

V for additional 3 days.

Our results show that LCZ 696 is superior respect to V in the reduction of the cardiotoxic effects of T, P and TDM1, when administered to cultures of H9C2 cardiomyoblasts after antineoplastic treatments. Indeed, LCZ 696 was significantly more effective than V in reducing T related cardiotoxicity, increasing cell viability of 25 % more, respect to V ($p < 0.001$). LCZ 696 is more strong in the reduction of P related toxicity, increasing cell viability of 35% more respect to V, with $p < 0.001$. And finally, again more effective than V in reducing TDM1 toxicity, increasing cell viability of 10 % ($p < 0, 05$).

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