Abstract: Colorectal cancer, as one of the main health problems worldwide. Cancer stem cells (CSCs) are called tumor-initiating cells involved in tumor heterogeneity and dormancy. CSCs may cause drug resistance, metastasis and relapse of primary and metastatic cancers. Dysregulation of survive and their cross-talk with other cell pathways may alternative way for effective treatment. We aimed whether acteosid, affects stemness and inflamatuvary prosess in primary (HCT-116) and metastatic (Colo-741) colon CSCs. CSCs were obtained from both type of colon cancer cell lines using MINIMACS system using anti-CD133. CD133+ and CD133- cells from Colo-741 and HCT-116 were cultured without or with Acteosid for 48 h. Distribution of Caspase-3, Bcl, Bax, IL-1β, TNF-α, IL-6, IL8 and IL-10 analyzed using indirect immunohistoperoxidase technique. Acteosid increased the intensity of Bax/Bcl ratio on CD133+, CD133- Colo-741 cells, there wasn’t differences of Caspase-3 immunoreactivity. Oct-4 was also decreased in CD133+ and CD133- Colo-741 cells. Caspase 3 was slightly increased CD133+ cells after incubation with Acteoside. In conclusion, it has been observed that the viability and stemness of the HCT-116 was increased after Acteoside, these properties were not changed in CD133+ Colo-741 cells. Therefore inhibition of ROS may play a role during CSCs stemness and viability.

Keywords: colon cancer; cancer stemness; acteosid; inflammation; apoptosis