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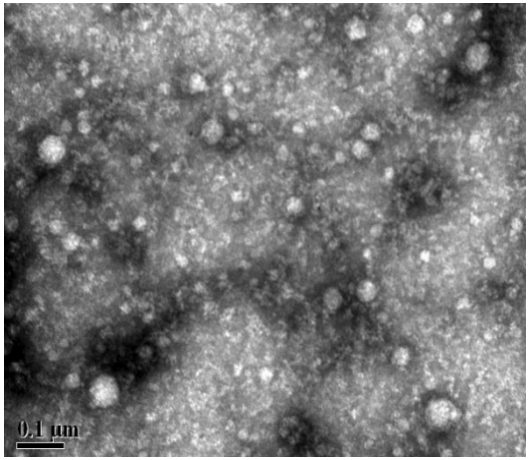
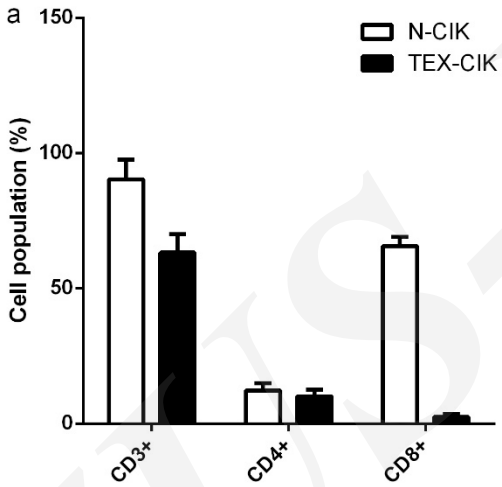
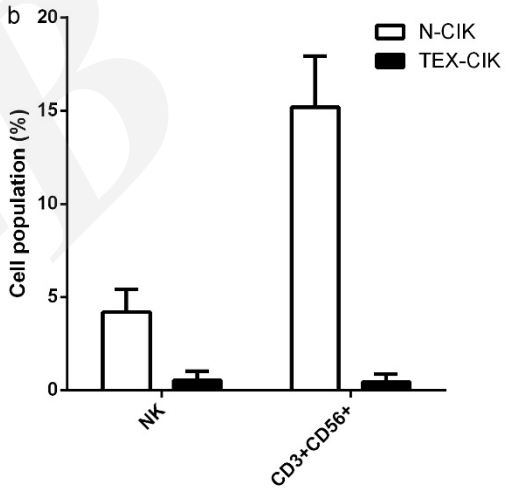
Cholangiocarcinoma derived exosomes inhibit the antitumor activity of cytokine-induced killer cells by down-regulating the secretion of TNF-α and perforin

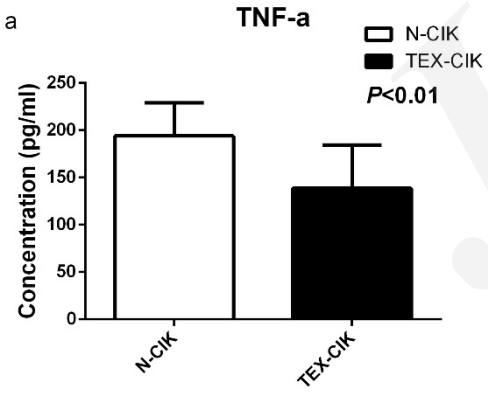
Key words: Cholangiocarcinoma, Tumor derived exosomes, Cytokine-induced killer cells, Immune escape

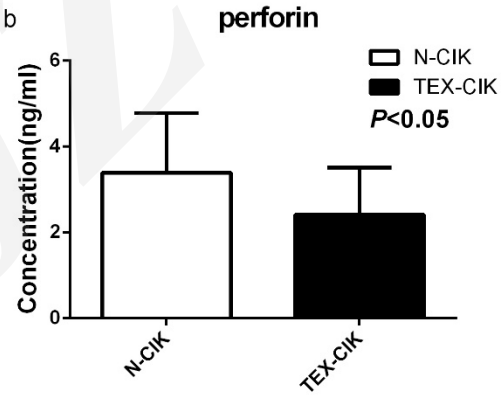
Research Summary

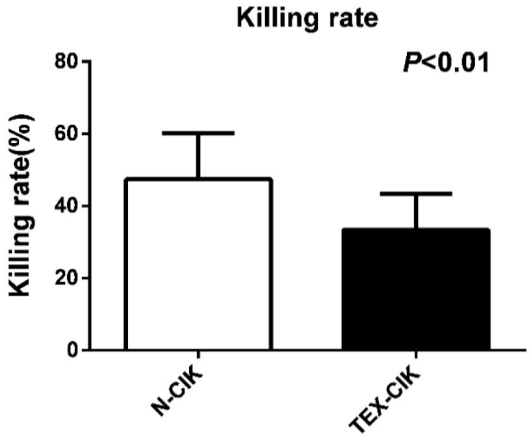












- The population of $CD3^+$, $CD8^+$, NK ($CD56^+$) and $CD3^+CD56^+$ cells decreased in the TEX-CIK group compared with the N-CIK group ($P<0.01$). However, the population change of $CD4^+$ cells was not statistically significant ($P>0.05$).
- The secretion of TNF-α ($P<0.01$) and perforin ($P<0.05$) was down-regulated by TEX in the CIK cells.
- The killing rate of the group TEX-CIK was significantly lower than the group N-CIK($P<0.01$).
- Conclusion: RBE cells derived exosomes (TEX) inhibit the antitumor activity of CIK cells by down- regulating the population of $CD3^+$, $CD8^+$, NK ($CD56^+$), $CD3^+CD56^+$ cells and the secretion of TNF-α and perforin. TEX may play an important role in cholangiocarcinoma immune escape