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Summary

ASSESSMENT OF CHANGES IN THE IMMUNE CELL INFILTRATE SUBPOPULATION IN SYNGENEIC MC38 COLORECTAL CANCER IN MICE AFTER INTAKE OF A-GLUTAMYL-TRYPTOPHAN

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Gut Microbiota | Mechanisms & Personalised Medicine | Digestive Oncology | Oesophagus

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α-glutamyl-tryptophan did not slow MC38 colorectal tumor growth in mice but prevented the immunosuppressive shift in tumor immune infiltrate seen in controls, maintaining CD3+ T cells and reducing M2-like macrophages.

KEY POINTS

- α-glutamyl-tryptophan at 0.02 mg/kg showed no significant difference in tumor growth kinetics versus saline control in a syngeneic MC38 colorectal cancer mouse model with 25 C57BL/6 mice.
- At day 21 post-transplantation, the experimental group maintained higher CD3+ T cells and prevented the twofold decline in DN NK1.1+ cells observed in controls.
- The experimental group showed reduced M2-like macrophages with high MHC expression, an immunosuppressive population that increased in controls.
- Control mice exhibited a pronounced decline in CD3+, MHC II low CD206+ M2-like, and DN NK1.1+ cells, indicating transition toward an immunosuppressive tumor microenvironment that was not observed with α-glutamyl-tryptophan.
- This is the first evaluation of immune infiltrate changes with α-glutamyl-tryptophan in a transplantable syngeneic tumor model in immunocompetent animals, supporting further investigation as an immunopreventive agent in precancerous conditions like chronic atrophic gastritis.

– The speaker, employed by Cytomed which financially supported the research, framed the findings as suggesting α-glutamyl-tryptophan may modulate the tumor microenvironment toward a less immunosuppressive state.

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