



Effect of Immune Checkpoint Blockade on Tumor Progression in Experimental Autoimmune Encephalitis

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Experimental Autoimmune Encephalitis (EA) is a disease characterized by the presence of T cells that infiltrate the central nervous system (CNS), leading to inflammation and tissue damage. This condition can be induced in animal models by injecting myelin basic protein (MBP) or other CNS antigens into the brain. A common treatment for EA is the use of immunotherapy, which involves blocking specific immune checkpoints to prevent T cells from becoming too active. One such checkpoint is the programmed cell death protein 1 (PD-1), which is expressed on the surface of activated T cells. When PD-1 binds to its ligand, BTLA, it inhibits the T cell's ability to kill cancer cells. By blocking this interaction, immunotherapy can restore the T cell's anti-tumor activity. In this study, we used a mouse model of EA to investigate the effect of anti-PD-1 therapy on tumor progression. We found that mice treated with anti-PD-1 antibodies had significantly reduced tumor size compared to untreated mice. This suggests that targeting the PD-1 pathway may be a promising strategy for treating EA and related diseases.

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96 / 24 D
(10 /) EA 5 / 10 / 20 / A
2-4 . 32 C 5% C₂
450 ELI A

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0.4 B C (4. 10^6 /C) / 0.4 C
 C L (100/L) / A (1/0.5
 10⁶/C). I 0.4 B C (4.
 10⁶/C) / 0.4 C EA
 (4. 10⁶/C). D 10
 24 32 C 5% C₂A
 450 10 -70 C

C. *Chlorophyllum molybdites* (L.)

F, IL-1, IL-6, IFN, IL-2, IL-10, IL-1b, ELI A (B, I, C, CA), IL-15, IL-6, 30

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1

A. G. L. G. 1

B C 24 / 10 20 / EA
 (F2,10=8.22, $P = 0.0077$)
 EA 20 / 29% ($P = 0.015$).
 24 H -29 K
 EA 5 / 20 / EA
 (F3,24=2.09,
 $P = 0.128$, F3,24=2.3, $P = 0.108$, $\alpha = 0.1$).

about *Anaspididae* (Homoptera)

24. B C EA
(>0.1, < 2, 3).

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EA μ g/ml	7 1). Q J P O (n=6)		, / Q J P O (n=6)		IL-6, ng/ml (n=6)		,) 1 Q J P O (n=6)	
	Mean \pm SEM	P*	Mean \pm SEM	P*	Mean \pm SEM	P*	Mean \pm SEM	P*
HT-29-induced								
0			7.40 \pm 0.47		27.44 \pm 3.32			
5		NS		0.002		0.02	2.00 \pm 0.32	NS
10	0.57 \pm 0.07	NS	5.75 \pm 0.49	<0.001		0.013	1.82 \pm 0.23	NS
20			3.47 \pm 0.29	<0.001	13.39 \pm 2.31	<0.001		0.049
RKO-induced								
0	0.58 \pm 0.09		5.54 \pm 0.70					
5		NS	5.57 \pm 0.90	NS		0.004	2.99 \pm 0.40	NS
10		NS	4.12 \pm 0.44	0.018	23.40 \pm 3.32	0.03	3.41 \pm 0.43	NS
20	0.47 \pm 0.08	0.047	2.50 \pm 0.44	0.001		<0.001		NS

Note: PBMC were incubated for 24 hrs with HT-29 or RKO colon cancer cells in the absence (0) or the presence of EA at concentrations as indicated. The level of cytokines

Table 4: < E E ED HED7OC7EH EAD HE ED

Conclusion

The results of this study demonstrate that ellagic acid modulates the immune balance between human mononuclear and colon carcinoma cells. Ellagic acid inhibits the proliferation of human mononuclear cells and induces apoptosis in two colon carcinoma cell lines. Ellagic acid also modulates the expression of various cytokines and chemokines in both cell types. These findings suggest that ellagic acid may have therapeutic potential in cancer prevention and treatment.

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- D 7E7 7E D7CC7EDD 7D H EI BB B
- AE E D7OC7EDD 7D H BB 7B7 HD D7OC7EH E BI 7 HBBED7OC7ED C 1B7 HED I Liver Physiol 287: G7-17.
- H7D B1 7E B1 H7C I BB 7B7 HD D7OC7EH E BI 7 CEE HAA7 HEC 7D D E carcinogenesis and prevention strategies. Anticancer Res 29: 2727-2737.
- H1 HH D7OC7EDD BB 7B7 D H III D Pharmacol 9: 405-410.
- 7 7OB H HD H7 DHH KCH D HH 7 CBED H BB E C7 H 7 I 7< OHHE 7D H I
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