Supplementary materials for ijms-2088333

Hispolon Methyl Ether, A Hispolon Analog, Suppresses the SRC/STAT3/Survivin Signaling Axis to Induce Cytotoxicity in Human Urinary Bladder Transitional Carcinoma Cell Lines

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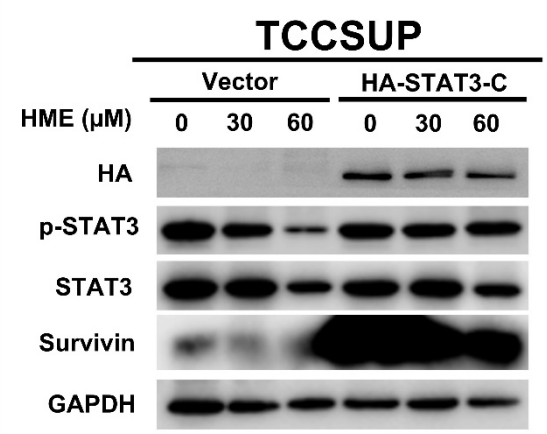

Figure S1. Ectopic expression of HA-STAT3-C sustains STAT3 activity following HME treatment. TCCSUP cell clones stably expressing HA-STAT3-C or the corresponding vector control were subjected to 24 h-treatment with HME (0, 30, 60 μM), followed by immunoblotting for the levels of HA, tyrosine 705-phosphorylated STAT3 (p-STAT3), total STAT3, Survivin, and GAPDH (loading control). The constant levels of p-STAT3 and Survivin in HME-treated STAT3-C stable clones illustrate the resistance of cells to HME-mediated STAT3 inhibition endowed by ectopic STAT3-C expression.