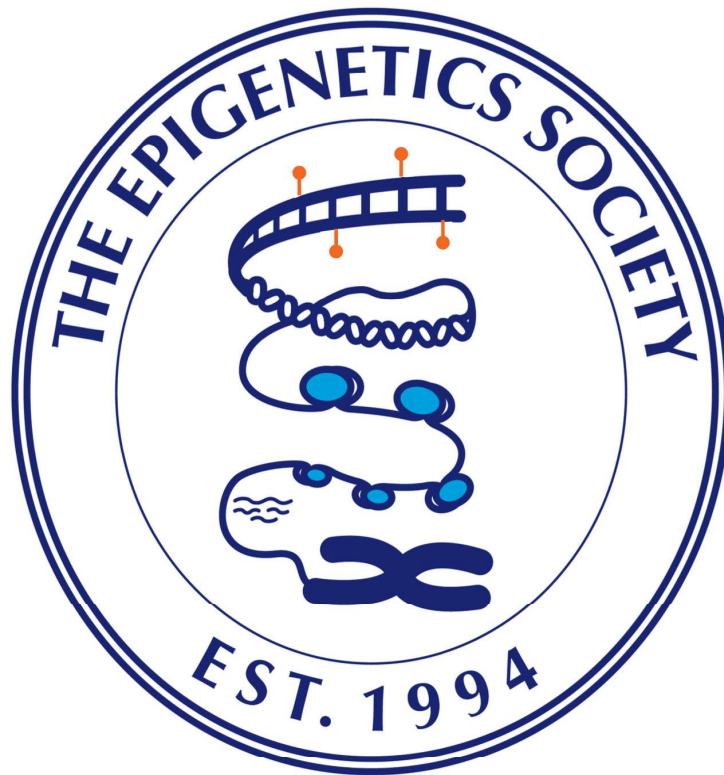
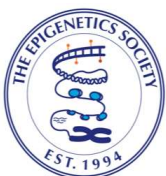




1st Epigenetics Society International Meeting

"Epigenetics of Disease and
Development"

Rome - Italy



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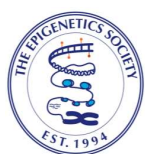
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Merkel cell carcinoma cells growth inhibited by the hypomethylating agent Guadecitabine

Author

Chiara Mazziotta – Department of Medical Sciences, University of Ferrara, Italy
Giulia Tonnini – Department of Medical Sciences, University of Ferrara, Italy
Giada Badiale – Department of Medical Sciences, University of Ferrara, Italy
Tommaso di Mambro – Department of Medical Sciences, University of Ferrara, Italy
Paolo Pinton – Department of Medical Sciences, University of Ferrara, Italy
Christian Felice Cervellera – Department of Medical Sciences, University of Ferrara, Italy
Mauro Tognon – Department of Medical Sciences, University of Ferrara, Italy
Antoine Touzé – ISP «Biologie des infections à polyomavirus» Team, UMR INRAE 1282, University of Tours, Tours, France
Fernanda Martini – Department of Medical Sciences, University of Ferrara, Italy;
Laboratory for Technologies of Advanced Therapies (LTTA), University of Ferrara, 44121, Ferrara, Italy
John Charles Rotondo – Department of Medical Sciences, University of Ferrara, Italy

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Abstract

Merkel cell carcinoma (MCC) is a rare, but aggressive skin cancer. Nearly 80% of MCCs are Merkel cell polyomavirus-positive (MCCP), while the remaining

cases are UV-induced and virus-negative (MCCN). Currently available MCC therapies are limited. Since impaired DNA methylation is common in cancer, epigenetic-based antitumor therapies are acquiring importance in clinical practice. Guadecitabine (GUAD) is a novel hypomethylating agent whose antitumor activity has been demonstrated in skin cancers. The antineoplastic activity of GUAD in MCC is unknown. This study aimed to evaluate the growth inhibition effect of GUAD in MCC cells. GUAD activity was evaluated by testing proliferation, viability, migration, and colony forming abilities in MCCN cells MCC13 and MCC26, and MSCP cells PeTa and WaGa and in the fibroblast control cell line HDFa. Apoptosis was investigated *via* western blot by evaluating the expression of apoptotic markers. After GUAD treatment, the methylome profile of each GUAD-treated MCC cell line was evaluated using the Infinium MethylationEPIC v2.0 Kit. Results indicate that GUAD can significantly reduce MCC cell proliferation, migration, and colony formation abilities, while increasing apoptosis/cell death in MCC cells compared to untreated cells/controls. A more pronounced sensitivity to GUAD was determined in MSCP cells. Increased levels of pro-apoptotic proteins, paralleled to decreased levels of anti-apoptotic proteins, were determined in GUAD-treated MCC cells. An extensive modulation of the methylome was detected in GUAD-treated MCC cells. GUAD demonstrated to be effective in reducing cell proliferation, migration, colony formation, inducing apoptosis and modulating the DNA methylome in MCC cells.

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