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Natural killer cell functionality is impaired by diet-induced obesity in a postmenopausal breast cancer mouse model

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Introduction: Obesity is a widespread disease and was identified as a major risk factor for malignant diseases, including postmenopausal breast cancer. Although the underlying mechanisms are poorly understood, it is known from in-vitro studies that essential functions of natural killer (NK) cells such as targeting tumor cells are disturbed in obese individuals. Thus, the aim of the present study is the investigation of NK cell functionality of obese mice in a postmenopausal breast cancer model.

Methods: To induce obesity, female mice (BALB/c) received either a standard chow (4% fat) or a high fat diet (34% fat) for up to 13 weeks. Thereafter, mice were ovariectomized and after 3 weeks of recovery, syngeneic 4T1-Luc2 mouse mammary tumor cells were injected orthotopically into the fat pad of the mammary gland. Twenty hours or 3 weeks after tumor cell challenging blood, tissues and tumors were collected and analyzed. Different techniques such as flow cytometry, Luminex and real-time PCR aimed to analyze numbers, activity and physiological properties of NK cells.

Results: Body weight, visceral fat amount, plasma leptin and IL-6 levels were significantly increased in diet-induced obese BALB/c mice. Tumor burden was increased in the obese animals as compared to their lean littermates. Interestingly, the functionality of NK cells was altered in the obese mice with breast cancer.

Conclusion: The present study on a postmenopausal breast cancer model helps to understand basic molecular mechanisms regulating NK cell functionality in obese individuals and the association of the elevated breast cancer risk in obesity.

Biography

Julia Spielmann has completed her PhD from Institute of Nutrition Science in Martin Luther University Halle-Wittenberg. She is a Postdoc at the Institute of Anatomy and Cell Biology Martin Luther University Halle-Wittenberg in Germany.

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