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ATIP3, a novel prognostic marker and therapeutic target for metastatic breast tumors

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Breast cancer metastasis, a leading cause of death by malignancy in women worldwide, does not fully benefit from available therapies. Efforts are being made to identify molecular markers that may predict metastatic outcome and represent new therapeutic targets for personalized treatments against metastatic breast tumors. Studies from our group have shown that ATIP3, the major product of MTUS1 candidate tumor suppressor gene, is down-regulated in invasive breast tumors of the triple-negative subtype. Low levels of ATIP3 correlate with reduced overall survival among patients with metastatic breast cancer. Ectopic expression of ATIP3 into breast cancer cell lines limits tumor growth and metastatic colonization in experimental mouse models. Furthermore we show that ATIP3 is a novel microtubule-associated protein that regulates microtubule dynamics and consequently mitosis, cell polarity, directionality and migration. Our results identify ATIP3 as a new promising therapeutic target against metastatic breast tumors of poor prognosis.

Biography

Upon completion of her Ph.D. thesis at the Institut Pasteur (Paris) in 1989, Clara Nahmias joined the Institut Cochin at the University Paris Descartes where she directed research on growth-inhibitory angiotensin II AT2 receptor signalling pathways. This led to the identification of novel intracellular cascades associated to G protein coupled receptor activation, including functional activation of tyrosine phosphatase SHP-1 and trans-inactivation of receptor tyrosine kinases, and the cloning of a novel family of proteins designated ATIP (AT2 receptor-interacting proteins). Her major focus of research since 2004 concerns the multiple effects of ATIPs in brain functions and tumor suppression. Recent studies have identified ATIP3 as a novel biomarker for invasive, triple negative and metastatic breast tumors. Concurrently, her team investigates the role of angiotensin II and its membrane receptors as novel regulators of breast cancer metastasis. These studies are expected to lead to the development of targeted treatment against invasive breast cancer progression and metastatic dissemination.

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P53 codon 72 polymorphism and lung cancer risk: Evidence from 27,958 subjects

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The role of p53 codon 72 polymorphism in the development of lung cancer remains obscure due to inconsistent findings of individual case-control studies published to date. A meta-analysis was conducted to better estimate the association between the p53 codon 72 variant and lung cancer risk. All relevant publications from the PubMed, Embase, Web of Science, and Wanfang databases were retrieved. Based on the inclusion criteria, 39 publications involving 44 independent case-control studies were finally included into this meta-analysis. Data were extracted and the pooled odds ratio (OR) with the corresponding 95% confidence interval (95% CI) was calculated. The overall pooled ORs showed no significant relationship of the p53 codon 72 polymorphism with increased or decreased risk of lung cancer in all gene contrast models ($OR_{Pro\ vs.\ Arg}$ =1.04, 95%CI =0.96-1.13, POR<0.001; $OR_{Pro/Pro\ vs.\ Arg/Arg}$ =1.07, 95%CI=0.91-1.25, POR<0.001; $OR_{Arg/Pro\ vs.\ Arg/Arg}$ =1.04, 95%CI =0.94-1.15, POR<0.001; $OR_{Pro/Pro + Arg/Pro\ vs.\ Arg/Arg}$ =1.04, 95%CI =0.94-1.16, POR<0.001; $OR_{Pro/Pro\ vs.\ Arg/Arg + Arg}$/Pro =1.07, 95%CI =0.93-1.23, POR<0.001). According to the ethnicity, no significant association was observed in subgroup analyses of the Asians, Caucasians, Africans and the mixed population. Similar finding was found in subgroup analyses of hospital-based and population-based studies. Concerning the histological types of lung cancer, the p53 codon 72 variant exerts risk effect on the lung carcinogenesis in patients with adenocarcinoma ($OR_{Arg/Pro\ vs.\ Arg/Arg}$ =1.10, 95%CI =1.00-1.22, POR=0.048). Additionally, subgroup analysis by the smoking status demonstrated that the p53 codon 72 variant seemed to play a protective role in lung carcinogenesis among the non-smokers but not the smokers in the contrast model of Arg/Pro vs. Arg/Arg ($OR_{Arg/Pro\ vs.\ Arg/Arg}$ =0.71, 95%CI =0.50-1.00, POR=0.049). The present meta-analysis suggests the p53 codon 72 polymorphism may weakly modify the risk for lung cancer among the adenocarcinoma patients and non-smokers. Nevertheless, this association needs further confirmation in future studies with high quality.

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