Année de publication : 2020

Floriane Pelon, Brigitte Bourachot, Yann Kieffer, Ilaria Magagna, Fanny Mermet-Meillon, Ana Costa, Anne-Marie Givel, Youmna Attieh, Jorge Barbazan, Laetitia Fuhrmann, Stéphanie Descroix, Danijela Vignjevic, Pascal Silberzan, Isabelle Bonnet, Claire Bonneau, Maria Carla Parrini, Anne Vincent-Salomon & Fatima Mechta-Grigoriou (2020 Jan 21)

Cancer-associated fibroblast heterogeneity in axillary lymph nodes drives metastases in breast cancer through complementary mechanisms

Nature Communication : 11 : 1-20 : DOI : 10.1038/s41467-019-14134-w

Résumé

Although fibroblast heterogeneity is recognized in primary tumors, both its characterization in and its impact on metastases remain unknown. Here, combining flow cytometry, immunohistochemistry and RNA-sequencing on breast cancer samples, we identify four Cancer-Associated Fibroblast (CAF) subpopulations in metastatic lymph nodes (LN). Two myofibroblastic subsets, CAF-S1 and CAF-S4, accumulate in LN and correlate with cancer cell invasion. By developing functional assays on primary cultures, we demonstrate that these subsets promote metastasis through distinct functions. While CAF-S1 stimulate cancer cell migration and initiate an epithelial-to-mesenchymal transition through CXCL12 and TGFβ pathways, highly contractile CAF-S4 induce cancer cell invasion in 3-dimensions via NOTCH signaling. Patients with high levels of CAFs, particularly CAF-S4, in LN at diagnosis are prone to develop late distant metastases. Our findings suggest that CAF subset accumulation in LN is a prognostic marker, suggesting that CAF subsets could be examined in axillary LN at diagnosis.

Année de publication : 2019

G Gentric, Y Kieffer, V Mieulet, O Goundiam, C Bonneau, F Nemati, I Hurbain, G Raposo, T Popova, MH Stern, V Lallemand-Breitenbach, S Müller, T Cañeque, R Rodriguez, A Vincent-Salomon, H de Thé, R Rossignol, F Mechta-Grigoriou (2019 Jan 5)

PML-Regulated Mitochondrial Metabolism Enhances Chemosensitivity in Human Ovarian Cancers

Cell Metabolism

Résumé

High-grade serous ovarian cancer (HGSOC) remains an unmet medical challenge. Here, we unravel an unanticipated metabolic heterogeneity in HGSOC. By combining proteomic, metabolomic, and bioergenetic analyses, we identify two molecular subgroups, low- and high-OXPHOS. While low-OXPHOS exhibit a glycolytic metabolism, high-OXPHOS HGSOCs rely on oxidative phosphorylation, supported by glutamine and fatty acid oxidation, and show chronic oxidative stress. We identify an important role for the PML-PGC-1α axis in the metabolic features of high-OXPHOS HGSOC. In high-OXPHOS tumors, chronic oxidative stress

promotes aggregation of PML-nuclear bodies, resulting in activation of the transcriptional co-activator PGC-1α. Active PGC-1α increases synthesis of electron transport chain complexes, thereby promoting mitochondrial respiration. Importantly, high-OXPHOS HGSOCs exhibit increased response to conventional chemotherapies, in which increased oxidative stress, PML, and potentially ferroptosis play key functions. Collectively, our data establish a stress-mediated PML-PGC-1α-dependent mechanism that promotes OXPHOS metabolism and chemosensitivity in ovarian cancer.

Année de publication : 2018

Givel AM, Kieffer Y, Scholer-Dahirel A, Sirven P, Cardon M, Pelon F, Magagna I, Gentric G, Costa A, Bonneau C, Mieulet V, Vincent-Salomon A, Mechta-Grigoriou F (2018 Mar 13)
miR200-regulated CXCL12β promotes fibroblast heterogeneity and immunosuppression in ovarian cancers.
Nature communications : DOI : 10.1038/s41467-018-03348-z.

Résumé

High-grade serous ovarian cancers (HGSOC) have been subdivided into molecular subtypes. The mesenchymal HGSOC subgroup, defined by stromal-related gene signatures, is invariably associated with poor patient survival. We demonstrate that stroma exerts a key function in mesenchymal HGSOC. We highlight stromal heterogeneity in HGSOC by identifying four subsets of carcinoma-associated fibroblasts (CAF-S1-4). Mesenchymal HGSOC show high content in CAF-S1 fibroblasts, which exhibit immunosuppressive functions by increasing attraction, survival, and differentiation of $CD25^{+}FOXP3^{+}$T lymphocytes. The beta isoform of the CXCL12 chemokine (CXCL12β) specifically accumulates in the immunosuppressive CAF-S1 subset through a miR-141/200a dependent-mechanism. Moreover, CXCL12β expression in CAF-S1 cells plays a crucial role in CAF-S1 immunosuppressive activity and is a reliable prognosis factor in HGSOC, in contrast to CXCL12α. Thus, our data highlight the differential regulation of the CXCL12α and CXCL12β isoforms in HGSOC, and reveal a CXCL12β-associated stromal heterogeneity and immunosuppressive environment in mesenchymal HGSOC.

Costa A, Kieffer Y, Scholer-Dahirel A, Pelon F, Bourachot B, Cardon M, Sirven P, Magagna I, Fuhrmann L, Bernard C, Bonneau C, Kondratova M, Kuperstein I, Zinovyev A, Givel AM, Parrini MC, Soumelis V, Vincent-Salomon A, Mechta-Grigoriou F (2018 Mar 12)
Fibroblast Heterogeneity and Immunosuppressive Environment in Human Breast Cancer.
Cancer Cell : 463-479 : DOI : 10.1016/j.ccell.2018.01.011

Résumé

Carcinoma-associated fibroblasts (CAF) are key players in the tumor microenvironment. Here, we characterize four CAF subsets in breast cancer with distinct properties and levels of

activation. Two myofibroblastic subsets (CAF-S1, CAF-S4) accumulate differentially in triple-negative breast cancers (TNBC). CAF-S1 fibroblasts promote an immunosuppressive environment through a multi-step mechanism. By secreting CXCL12, CAF-S1 attracts $CD4^{+}CD25^{+}$T lymphocytes and retains them by OX40L, PD-L2, and JAM2. Moreover, CAF-S1 increases T lymphocyte survival and promotes their differentiation into $CD25^{High}FOXP3^{High}$, through B7H3, CD73, and DPP4. Finally, in contrast to CAF-S4, CAF-S1 enhances the regulatory T cell capacity to inhibit T effector proliferation. These data are consistent with FOXP3+ T lymphocyte accumulation in CAF-S1-enriched TNBC and show how a CAF subset contributes to immunosuppression.

Année de publication : 2017

Lucie Hebert, Dorine Bellanger, Chloé Guillas, Antoine Campagne, Florent Dingli, Damarys Loew, Alice Fievet, Virginie Jacquemin, Tatiana Popova, Didier Jean, Fatima Mechta-Grigoriou, Raphaël Margueron, Marc-Henri Stern (2017 Oct 27)

Modulating BAP1 expression affects ROS homeostasis, cell motility and mitochondrial function.

Oncotarget : 72513-72527 : DOI : 10.18632/oncotarget.19872

Résumé

The tumor suppressor BAP1 associates with ASXL1/2 to form the core Polycomb complex PR-DUB, which catalyzes the removal of mono-ubiquitin from several substrates including histone H2A. This complex also mediates the poly-deubiquitination of HCFC1, OGT and PCG1-α, preventing them from proteasomal degradation. Surprisingly, considering its role in a Polycomb complex, no transcriptional signature was consistently found among BAP1-inactivated tumor types. It was hypothesized that BAP1 tumor suppressor activity could reside, at least in part, in stabilizing proteins through its poly-deubiquitinase activity. Quantitative mass spectrometry and gene expression arrays were used to investigate the consequences of BAP1 expression modulation in the NCI-H226 mesothelioma cell line. Analysis of differentially expressed proteins revealed enrichment in cytoskeleton organization, mitochondrial activity and ROS management, while gene expression analysis revealed enrichment in the epithelial-to-mesenchymal transition pathway. Functional assessments in BAP1 inactivated, BAP1 wild-type and BAP1 catalytically dead-expressing NCI-H226 and QR mesothelioma cell lines confirmed alteration of these pathways and demonstrated that BAP1 deubiquitinase activity was mandatory to maintain these phenotypes. Interestingly, monitoring intracellular ROS levels partly restored the morphology and the mitochondrial activity. Finally, the study suggests new tumorigenic and cellular functions of BAP1 and shows for the first time the interest of studying the proteome as readout of BAP1 inactivation.

Gentric G, Mieulet V, Mechta-Grigoriou F (2017 Mar 20)

Heterogeneity in Cancer Metabolism: New Concepts in an Old Field.

Antioxidants & Redox Signaling : 26 : DOI : 10.1089/ars.2016.6750

Résumé

SIGNIFICANCE:
In the last years, metabolic reprogramming, fluctuations in bioenergetic fuels, and modulation of oxidative stress became newkey hallmarks of tumor development. In cancer, elevated glucose uptake and high glycolytic rate, as a source of adenosine triphosphate, constitute a growth advantage for tumors. This represents the universally known Warburg effect, which gave rise to one major clinical application for detecting cancercells using glucose analogs: the positron emission tomography scan imaging. Recent Advances: Glucose utilization and carbon sources in tumors are much more heterogeneous than initially thought. Indeed, newstudies emerged and revealed a dual capacity of tumor cells for glycolytic and oxidative phosphorylation (OXPHOS) metabolism. OXPHOS metabolism, which relies predominantly on mitochondrial respiration, exhibits fine-tuned regulation of respiratory chain complexes and enhanced antioxidant response or detoxification capacity.

CRITICAL ISSUES:
OXPHOS-dependent cancercells use alternative oxidizable substrates, such as glutamine and fatty acids. The diversity of carbon substrates fueling neoplastic cells is indicative of metabolic heterogeneity, even within tumors sharing the same clinical diagnosis. Metabolic switch supports cancercell stemness and their bioenergy-consuming functions, such as proliferation, survival, migration, and invasion. Moreover, reactive oxygen species-induced mitochondrial metabolismand nutrient availability are important for interaction with tumor microenvironment components. Carcinoma-associated fibroblasts and immune cells participate in the metabolic interplay with neoplastic cells. They collectively adapt in a dynamic manner to the metabolic needs of cancercells, thus participating in tumorigenesis and resistance to treatments.

Lefort, A Thuleau, Y Kieffer, P Sirven, I Bieche, E Marangoni, A Vincent-Salomon & F Mechta-Grigoriou (2017 Mar 2)

CXCR4 inhibitors could benefit to HER2 but not to triple-negative breast cancer patients

Oncogene : 1211–1222 : DOI : 10.1038

Résumé

The CXCR4 receptor and its ligand CXCL12 (also named stromal cell-derived factor 1, SDF1) have a critical role in chemotaxis and homing, key steps in cancer metastasis. Although myofibroblasts expressing CXCL12 are associated with the presence of axillary metastases in HER2 breast cancers (BC), the therapeutic interest of targeting CXCR4/CXCL12 axis in the different BC subtypes remains unclear. Here, we investigate this question by testing antitumor activity of CXCR4 inhibitors in patient-derived xenografts (PDX), which faithfully reproduce human tumor properties. We observed that two CXCR4 inhibitors, AMD3100 and TN14003, efficiently impair tumor growth and metastasis dissemination in both Herceptin-sensitive and Herceptin-resistant HER2 BC. Conversely, blocking CXCR4/CXCL12 pathway in triple-negative (TN) BC does not reduce tumor growth, and can even increase metastatic spread. Moreover, although CXCR4 inhibitors significantly reduce myofibroblast content in all

BC subtypes, they decrease angiogenesis only in HER2 BC. Thus, our findings suggest that targeting CXCR4 could provide some therapeutic interest for HER2 BC patients, whereas it has no impact or could even be detrimental for TN BC patients.

Année de publication : 2016

S Lefort, A Thuleau, Y Kieffer, P Sirven, I Bieche, E Marangoni, A Vincent-Salomon, F Mechta-Grigoriou (2016 Sep 27)

CXCR4 inhibitors could benefit to HER2 but not to triple-negative breast cancer patients.

Oncogene : DOI : 10.1038/onc.2016.284

Résumé

The CXCR4 receptor and its ligand CXCL12 (also named stromal cell-derived factor 1, SDF1) have a critical role in chemotaxis and homing, key steps in cancer metastasis. Although myofibroblasts expressing CXCL12 are associated with the presence of axillary metastases in HER2 breast cancers (BC), the therapeutic interest of targeting CXCR4/CXCL12 axis in the different BC subtypes remains unclear. Here, we investigate this question by testing antitumor activity of CXCR4 inhibitors in patient-derived xenografts (PDX), which faithfully reproduce human tumor properties. We observed that two CXCR4 inhibitors, AMD3100 and TN14003, efficiently impair tumor growth and metastasis dissemination in both Herceptin-sensitive and Herceptin-resistant HER2 BC. Conversely, blocking CXCR4/CXCL12 pathway in triple-negative (TN) BC does not reduce tumor growth, and can even increase metastatic spread. Moreover, although CXCR4 inhibitors significantly reduce myofibroblast content in all BC subtypes, they decrease angiogenesis only in HER2 BC. Thus, our findings suggest that targeting CXCR4 could provide some therapeutic interest for HER2 BC patients, whereas it has no impact or could even be detrimental for TN BC patients.Oncogene advance online publication, 26 September 2016; doi:10.1038/onc.2016.284.

Geraldine Gentric, Virginie Mieulet, Fatima Mechta-Grigoriou (2016 May 28)

Heterogeneity in Cancer Metabolism: New Concepts in an old Field.

Antioxidants & redox signaling

Résumé

In the last years, metabolic reprogramming, fluctuations in bioenergetic fuels and modulation of oxidative stress became new key hallmarks of tumor development. In cancer, elevated glucose uptake and high glycolytic rate, as source of ATP, constitute a growth advantage for tumors. This represents the universally known « Warburg effect », which gave rise to one major clinical application for detecting cancer cells using glucose analogues: the PET-scan imaging.

Tina Gruosso, Virginie Mieulet, Melissa Cardon, Brigitte Bourachot, Yann Kieffer, Flavien Devun, Thierry Dubois, Marie Dutreix, Anne Vincent-Salomon, Kyle Malcolm Miller, Fatima Mechta-Grigoriou (2016 Mar 24)

Chronic oxidative stress promotes H2AX protein degradation and enhances chemosensitivity in breast cancer patients.

EMBO molecular medicine : 527-49 : DOI : 10.15252/emmm.201505891

Résumé

Anti-cancer drugs often increase reactive oxygen species (ROS) and cause DNA damage. Here, we highlight a new cross talk between chronic oxidative stress and the histone variant H2AX, a key player in DNA repair. We observe that persistent accumulation of ROS, due to a deficient JunD-/Nrf2-antioxidant response, reduces H2AX protein levels. This effect is mediated by an enhanced interaction of H2AX with the E3 ubiquitin ligase RNF168, which is associated with H2AX poly-ubiquitination and promotes its degradation by the proteasome. ROS-mediated H2AX decrease plays a crucial role in chemosensitivity. Indeed, cycles of chemotherapy that sustainably increase ROS reduce H2AX protein levels in Triple-Negative breast cancer (TNBC) patients. H2AX decrease by such treatment is associated with an impaired NRF2-antioxidant response and is indicative of the therapeutic efficiency and survival of TNBC patients. Thus, our data describe a novel ROS-mediated regulation of H2AX turnover, which provides new insights into genetic instability and treatment efficacy in TNBC patients.

Christophe Couderc, Alizée Boin, Laetitia Fuhrmann, Anne Vincent-Salomon, Vinay Mandati, Yann Kieffer, Fatima Mechta-Grigoriou, Laurence Del Maestro, Philippe Chavrier, David Vallerand, Isabelle Brito, Thierry Dubois, Leanne De Koning, Daniel Bouvard, Daniel Louvard, Alexis Gautreau, Dominique Lallemand (2016 Jan 26)

AMOTL1 integrates Hippo signaling to promote breast cancer progression by inducing tumor cell proliferation and migration

Neoplasia (New York, N.Y.) : 10-24 : DOI : 10.1016/j.neo.2015.11.010

Résumé

The Hippo signaling network is a key regulator of cell fate. In the recent years, it was shown that its implication in cancer goes well beyond the sole role of YAP transcriptional activity and its regulation by the canonical MST/LATS kinase cascade. Here we show that the motin family member AMOTL1 is an important effector of Hippo signaling in breast cancer. AMOTL1 connects Hippo signaling to tumor cell aggressiveness. We show that both canonical and noncanonical Hippo signaling modulates AMOTL1 levels. The tumor suppressor Merlin triggers AMOTL1 proteasomal degradation mediated by the NEDD family of ubiquitin ligases through direct interaction. In parallel, YAP stimulates AMOTL1 expression. The loss of Merlin expression and the induction of Yap activity that are frequently observed in breast cancers thus result in elevated AMOTL1 levels. AMOTL1 expression is sufficient to trigger tumor cell migration and stimulates proliferation by activating c-Src. In a large cohort of human breast

tumors, we show that AMOTL1 protein levels are upregulated during cancer progression and that, importantly, the expression of AMOTL1 in lymph node metastasis appears predictive of the risk of relapse. Hence we uncover an important mechanism by which Hippo signaling promotes breast cancer progression by modulating the expression of AMOTL1.

Daniel J Klionsky, Kotb Abdelmohsen, Akihisa Abe, Md Joynal Abedin, Hagai Abeliovich, Abraham Acevedo Arozena, Hiroaki Adachi, Christopher M Adams, Peter D Adams, Khosrow Adeli, Peter J Adhihetty, Sharon G Adler, Galila Agam, Rajesh Agarwal, Manish K Aghi, Maria Agnello, Patrizia Agostinis, Patricia V Aguilar, Julio Aguirre-Ghiso, Edoardo M Airoldi, Slimane Ait-Si-Ali, Takahiko Akematsu, Emmanuel T Akporiaye, Mohamed Al-Rubeai, Guillermo M Albaiceta, Chris Albanese, Diego Albani, Matthew L Albert, Jesus Aldudo, Hana Algül, Mehrdad Alirezaei, Iraide Alloza, Alexandru Almasan, Maylin Almonte-Beceril, Emad S Alnemri, Covadonga Alonso, Nihal Altan-Bonnet, Dario C Altieri, Silvia Alvarez, Lydia Alvarez-Erviti, Sandro Alves, Giuseppina Amadoro, Atsuo Amano, Consuelo Amantini, Santiago Ambrosio, Ivano Amelio, Amal O Amer, Mohamed Amessou, Angelika Amon, Zhenyi An, Frank A Anania, Stig U Andersen, Usha P Andley, Catherine K Andreadi, Nathalie Andrieu-Abadie, Alberto Anel, David K Ann, Shailendra Anoopkumar-Dukie, Manuela Antonioli, Hiroshi Aoki, Nadezda Apostolova, Saveria Aquila, Katia Aquilano, Koichi Araki, Eli Arama, Agustin Aranda, Jun Araya, Alexandre Arcaro, Esperanza Arias, Hirokazu Arimoto, Aileen R Ariosa, Jane L Armstrong, Thierry Arnould, Ivica Arsov, Katsuhiko Asanuma, Valerie Askanas, Eric Asselin, Ryuichiro Atarashi, Sally S Atherton, Julie D Atkin, Laura D Attardi, Patrick Auberger, Georg Auburger, Laure Aurelian, Riccardo Autelli, Laura Avagliano, Maria Laura Avantaggiati, Limor Avrahami, Suresh Awale, Neelam Azad, Tiziana Bachetti, Jonathan M Backer, Dong-Hun Bae, Jae-Sung Bae, Ok-Nam Bae, Soo Han Bae, Eric H Baehrecke, Seung-Hoon Baek, Stephen Baghdiguian, Agnieszka Bagniewska-Zadworna, Hua Bai, Jie Bai, Xue-Yuan Bai, Yannick Bailly, Kithiganahalli Narayanaswamy Balaji, Walter Balduini, Andrea Ballabio, Rena Balzan, Rajkumar Banerjee, Gábor Bánhegyi, Haijun Bao, Benoit Barbeau, Maria D Barrachina, Esther Barreiro, Bonnie Bartel, Alberto Bartolomé, Diane C Bassham, Maria Teresa Bassi, Robert C Bast, Alakananda Basu, Maria Teresa Batista, Henri Batoko, Maurizio Battino, Kyle Bauckman, Bradley L Baumgarner, K Ulrich Bayer, Rupert Beale, Jean-François Beaulieu, George R Beck, Christoph Becker, J David Beckham, Pierre-André Bédard, Patrick J Bednarski, Thomas J Begley, Christian Behl, Christian Behrends, Georg Mn Behrens, Kevin E Behrns, Eloy Bejarano, Amine Belaid, Francesca Belleudi, Giovanni Bénard, Guy Berchem, Daniele Bergamaschi, Matteo Bergami, Ben Berkhout, Laura Berliocchi, Amélie Bernard, Monique Bernard, Francesca Bernassola, Anne Bertolotti, Amanda S Bess, Sébastien Besteiro, Saverio Bettuzzi, Savita Bhalla, Shalmoli Bhattacharyya, Sujit K Bhutia, Caroline Biagosch, Michele Wolfe Bianchi, Martine Biard-Piechaczyk, Viktor Billes, Claudia Bincoletto, Baris Bingol, Sara W Bird, Marc Bitoun, Ivana Bjedov, Craig Blackstone, Lionel Blanc, Guillermo A Blanco, Heidi Kiil Blomhoff, Emilio Boada-Romero, Stefan Böckler, Marianne Boes, Kathleen Boesze-Battaglia, Lawrence H Boise,

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Guidelines for the use and interpretation of assays for monitoring autophagy (3rd edition).

Autophagy : 1-222 : DOI : 10.1080/15548627.2015.1100356

Résumé

Luciana Batista, Brigitte Bourachot, Bogdan Mateescu, Fabien Reyal, Fatima Mechta-Grigoriou (2016 Jan 5)

Regulation of miR-200c/141 expression by intergenic DNA-looping and transcriptional read-through.

Nature communications : 8959 : DOI : 10.1038/ncomms9959

Résumé

The miR-200 family members have been implicated in stress responses and ovarian tumorigenesis. Here, we find that miR-200c/141 transcription is intimately linked to the transcription of the proximal upstream gene PTPN6 (SHP1) in all physiological conditions tested. PTPN6 and miR-200c/141 are transcriptionally co-regulated by two complementary mechanisms. First, a bypass of the regular PTPN6 polyadenylation signal allows the transcription of the downstream miR-200c/141. Second, the promoters of the PTPN6 and miR-200c/141 transcription units physically interact through a 3-dimensional DNA loop and exhibit similar epigenetic regulation. Our findings highlight that transcription of intergenic miRNAs is a novel outcome of transcriptional read-through and reveal a yet unexplored type

of DNA loop associating two closely located promoters. These mechanisms have significant relevance in ovarian cancers and stress response, pathophysiological conditions in which miR-200c/141 exert key functions.

Année de publication : 2015

Tina Gruosso, Camille Garnier, Sophie Abelanet, Yann Kieffer, Vincent Lemesre, Dorine Bellanger, Ivan Bieche, Elisabetta Marangoni, Xavier Sastre-Garau, Virginie Mieulet, Fatima Mechta-Grigoriou (2015 Oct 13)

MAP3K8/TPL-2/COT is a potential predictive marker for MEK inhibitor treatment in high-grade serous ovarian carcinomas.

Nature communications : 8583 : DOI : 10.1038/ncomms9583

Résumé

Ovarian cancer is a silent disease with a poor prognosis that urgently requires new therapeutic strategies. In low-grade ovarian tumours, mutations in the MAP3K BRAF gene constitutively activate the downstream kinase MEK. Here we demonstrate that an additional MAP3K, MAP3K8 (TPL-2/COT), accumulates in high-grade serous ovarian carcinomas (HGSCs) and is a potential prognostic marker for these tumours. By combining analyses on HGSC patient cohorts, ovarian cancer cells and patient-derived xenografts, we demonstrate that MAP3K8 controls cancer cell proliferation and migration by regulating key players in G1/S transition and adhesion dynamics. In addition, we show that the MEK pathway is the main pathway involved in mediating MAP3K8 function, and that MAP3K8 exhibits a reliable predictive value for the effectiveness of MEK inhibitor treatment. Our data highlight key roles for MAP3K8 in HGSC and indicate that MEK inhibitors could be a useful treatment strategy, in combination with conventional chemotherapy, for this disease.

Année de publication : 2014

Benjamin G Cuiffo, Antoine Campagne, George W Bell, Antonio Lembo, Francesca Orso, Evan C Lien, Manoj K Bhasin, Monica Raimo, Summer E Hanson, Andriy Marusyk, Dorraya El-Ashry, Peiman Hematti, Kornelia Polyak, Fatima Mechta-Grigoriou, Odette Mariani, Stefano Volinia, Anne Vincent-Salomon, Daniela Taverna, Antoine E Karnoub (2014 Dec 18)

MSC-regulated microRNAs converge on the transcription factor FOXP2 and promote breast cancer metastasis.

Cell stem cell : 762-74 : DOI : 10.1016/j.stem.2014.10.001

Résumé

Mesenchymal stem/stromal cells (MSCs) are progenitor cells shown to participate in breast tumor stroma formation and to promote metastasis. Despite expanding knowledge of their contributions to breast malignancy, the underlying molecular responses of breast cancer

cells (BCCs) to MSC influences remain incompletely understood. Here, we show that MSCs cause aberrant expression of microRNAs, which, led by microRNA-199a, provide BCCs with enhanced cancer stem cell (CSC) properties. We demonstrate that such MSC-deregulated microRNAs constitute a network that converges on and represses the expression of FOXP2, a forkhead transcription factor tightly associated with speech and language development. FOXP2 knockdown in BCCs was sufficient in promoting CSC propagation, tumor initiation, and metastasis. Importantly, elevated microRNA-199a and depressed FOXP2 expression levels are prominent features of malignant clinical breast cancer and are associated significantly with poor survival. Our results identify molecular determinants of cancer progression of potential utility in the prognosis and therapy of breast cancer.