Journal of Medical and Biological Science Research
Vol. 6 (2), pp. 11-13, May, 2020
ISSN: 2449-1810
Short Communication
https://doi.org/10.36630/jmbsr_20002
http://pearlresearchjournals.org/journals/jmbsr/index.html

Metastasis and immune-metabolic interference

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Accepted 2 April, 2020

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ABSTRACT

Cancer is a life-threatening disease attributable to unhindered growth and metastasis. Metastatic cells spread to distant locations in the body and form malignant tumors, the leading cause of death in cancer patients. A primary function of the immune system is to confer protection against cancer. Atopy (i.e., many allergies) has been suggested as cancer immunotherapy to suppress the metabolism of metastatic cells and provide a rate-limiting step in metastasis. This review explores hyper-allergenic skin creams designed to impede metastasis through immune-metabolic interference.

Keywords: Atopy; Immunoglobulin-E antibodies; Hyper-allergenic skin cream; Immune-metabolic interference; Metastasis; Recombinant allergen.

ABBREVIATIONS: IMI; immuno-metabolic interference.

INTRODUCTION

Cancer is a painful and tragic disease. Approximately 90% of cancer deaths are due to metastasis (Seyfried, 2013). During metastasis, metastatic cells break away from the primary tumor to form secondary tumors throughout the body. Predicting where metastatic cells locate is often unpredictable. Metastatic cells merge with trillions of healthy cells in the bloodstream (Dean, 2005) and flow through a circulatory network that involves thousands of miles of arteries, veins, and capillaries (Cleveland Clinic). In some forms of cancer, metastasis is less random. Patanaphan (1988) have reported that breast cancer metastasis sites are bones, lungs, brain, and the liver.

Cancer cells mutate quickly, allowing the formation of abnormal proteins that helps them to adapt and survive. Reiter (2018) has reported that primary and metastatic tumors within an individual all likely rely on the same genetic mutations to grow and spread.

Campbell et al. (2020) have reported that on average, cancer genomes contained 4–5 driver mutations when combining coding and non-coding genomic elements; however, in around 5% of cases, no drivers were identified, suggesting that cancer driver discovery is not yet complete.

Pavlova and Thompson (2016) have stated that a common feature of metastasis is the ability to acquire necessary nutrients from a frequently nutrient-poor environment and utilize these nutrients to both maintain viability and build new biomass.

Fiddler (2002) reports that the primary cause of death from cancer is due to metastasis that is resistant to conventional therapies. Several reasons account for the failure to treat metastases.First, neoplasms are biologically heterogeneous and contain subpopulations of cells with different angiogenic, invasive, and metastatic properties. Second, the process of metastasis selects for a small subpopulation of cells that pre-exist within a parental neoplasm. Third, the outcome of metastasis depends on multiple interactions (i.e., crosstalk) of metastatic cells with homeostatic mechanisms that the tumor cells usurp. Therapy targeted against metastatic tumor cells and the homeostatic factors that promote metastasis may be beneficial.

Fares et al. (2020) have stated, metastasis is the final frontier in cancer for which more efficacious therapies are needed.

This short communication discusses hyper-allergenic skin creams that induce the formation of immunoglobulin

E antibodies to impede metastasis through immune-metabolic interference (IMI).

DISCUSSION

Can the immune system inhibit metastasis? Corthay (2014) has stated that there are compelling pieces of evidence that a primary function of the immune system is to confer protection against cancer.

Cancer immunotherapy evolves as treatments attempt to discriminate between healthy and metastatic cells. Oncogenic metabolism might be the primary target for the prevention of metastasis.

Lee et al. (2018) have stated that understanding the molecular mechanisms between oncogenic metabolism and metastasis may reveal the role of the metabolism in tumor development and be crucial for the development of therapeutic strategies.

Activation immunotherapy is the stimulation of the immune system to improve the body's ability to fight cancer. Aalberse et al. (2001) have stated that immunoglobulin-E (IgE) antibodies that bind to homologous proteins are considered cross-reactive IgE antibodies.

Induced atopy is cancer immunotherapy intended to be a rate-limiting step in metastasis through immune-metabolic interference (IMI). IMI is a process wherein the body's energy and resources are directed towards atopy and away from metastasis. Rauw (2012) has stated, "The negative influence of activation of the immune system on growth is well established resulting from a redirection of resources toward an immune response and away from other functions".

The use of hyper-allergenic skin creams is non-specific active immunotherapy designed to induce atopic conditions (i.e., many allergies) and inhibit the progression of metastatic cells (Dochniak, 2020).

A hyper-allergenic skin cream designed to inhibit metastatic breast cancer may include the allergens: Timothy grass pollen (rPhl p 7); Hevea Brasiliensis (rHev B 1); Honeybee venom (Api m 1); Cockroach (Bla g 2); and Alternaria mold (rAlt a 6) [16].

Wang et al. (2017) have reported that the overexpression of S-100 protein has a role in breast cancer . A recombinant allergen from Timothy Grass Pollen (rPhl p 7) (Thermo Fischer Scientific, 2020) may induce the humoral immune system to produce antibodies that inhibit the expression of human S-100 proteins. The rPhl p 7 allergen (~ 9 kDa) and the human S-100 proteins (9-13 kDa) are Ca2+-binding proteins with helix-loop-helix conformation.

Fritz et al. (1999) have shown that Rho GTPases are over-expressed in breast tumors. The Rho family of GTPases is a group of small (~21 kDa) signaling G proteins, a subfamily of the Ras superfamily. A recombinant rubber elongation factor allergen (rHev b 1, ~ 14kDa) (Thermo Fischer Scientific, 2020) from Hevea Brasiliensis may induce the humoral immune system to produce IgE antibodies that inhibit the expression of human GTPases (Dochniak, 2016).

Chen et al. (2016) have shown that the expression of phospholipase plays an essential role in the infiltration, metastasis, and mucous epithelium atypicality of breast infiltrating ductal carcinoma. Phospholipase A2 belongs to a family of enzymes that catalyze the cleavage of fatty acids from the sn-2 position of phospholipids. A recombinant phospholipase A2 allergen (rApi m 1, ~ 16kDa) (Thermo Fischer Scientific, 2020) from Honeybee venom may induce the humoral immune system to produce IgE antibodies that inhibit the expression of human phospholipase A2 isoforms (~14 kDa).

Liaudet-Coopman et al. (2006) have shown that aspartic protease is an independent marker of poor prognosis in breast cancer and correlated with the incidence of clinical metastasis. The lysosomal aspartic protease cathepsin D is over-expressed in breast cancer, hyper-secreted by epithelial cells. Cathepsin-D is an aspartic endo-protease that degrades proteins and activates precursors of bioactive proteins in pre-lysosomal compartments. A recombinant aspartic-protease allergen (rBla g 2, ~ 36kDa) (Thermo Fischer Scientific, 2020) from cockroach may induce the humoral immune system to produce IgE antibodies that inhibit the expression of human aspartic protease cathepsin-D (~ 48 kDa).

Tu et al. (2010) have shown that increased expression of enolase-1 may affect 4-Hydroxytamoxifen resistance in breast cancer therapy. Enolase-1 (~ 48 kDa) is a glycolytic enzyme expressed in most tissues. A recombinant enolase allergen (rAlt a 6, ~ 45kDa) (Thermo Fischer Scientific, 2020) from Alternaria mold may induce the humoral immune system to produce IgE antibodies that inhibit the over-expression of human alpha-enolase.

Patients with stage-IV breast cancer can have a weakened immune system because treatments, including chemotherapy, surgery, and some forms of radiation, leave them more vulnerable to infection. Inactivated vaccines support the adaptive and maladaptive aspect of our immune system by stimulating the production of antibodies; and by reducing complications from infectious disease. Inactivated vaccines do not cause infection and weaken the immune system because they consist of non-infectious particles from bacteria, viruses, and pathogens. The Centers for Disease Control and Prevention and the Advisory Committee on Immunization Practices recommend certain vaccines for routine use in adults, including those with cancer. Shah et al. (2018) have reported that certain vaccine-preventable diseases have higher incidence rates among cancer patients and are associated with

worse clinical outcomes.

CONCLUSION

Exploring the maladaptive aspect of our immune system, to starve-out cancerous cells is a formidable task. Induced humoral immunity is a disruptive therapy designed to manipulate biochemical processes and disrupt metastatic cells during metastasis. Patients diagnosed with stage-IV cancer have an opportunity to self-medicate with hyper-allergenic skin creams, to produce IgE antibodies, and to effect metastasis through immune-metabolic interference.

AUTHOR DISCLOSURE

This review discusses cancer immunotherapy using a hyper-allergenic skin cream.

REFERENCES

Aalberse RC, Akkerdaas J, Van RR (2001). Cross-reactivity of IgE antibodies to allergens. Allergy; 56(6): 478–490.

Campbell PJ, Getz G, Korbel JO (2020). Pan-cancer analysis of whole genomes. Nature 578:82–93.

Chen HZ, Qu YH, Diao CY, Wang XH, Gao M, Song LJ, Gao XZ, Han J, Wang F, Li SL, Han XW (2016). Expression of phospholipase A2 in breast cancer tissues and its significance. Int J Clin Exp Pathol; 9(11):11820-11825.

Cleveland Clinic. How does blood flow through the body? https://my.clevelandclinic.org/health/articles/17059-how-does-blood-flow-through-your-body (Accessed March 29, 2020)

Corthay A (2014). Does the immune system naturally protect against cancer? Frontiers in Immunol; 5:197.

Dean L (2005). Blood Groups and Red Cell Antigens [Internet]. National Center for Biotechnology Information, Bethesda (MD), Chapter 1, Blood and the cells it contains.

Dochniak MJ (2016) Rubber elongation factor and natural allergy-oncology. BAOJ Cancer Res Ther; 2(2): 027.

Dochniak MJ (2020). Maladaptive immunity and metastasizing cancer. Cancer Med J;3: 31-34.

Dochniak MJ (2020). Pediatric Metastasis and cIgE Antibodies. Open J Pediatr Neonatal Care; 5(1): 001-003.

Fares J, Fares MY, Khachfe HH, Salhab HA, Fares Y (2020). Signal Transduction and Targeted Therapy. 5(28):1-17.

Fidler IJ (2002). Critical determinants of metastasis. Semin. Cancer Biol;12(2):89-96.

Fritz G, Just I, Kaina B (1999). Rho GTPases are over-expressed in human tumors. Int J Cancer; 81: 682-687.

Lee SY, Ju MK, Jeon HM, Lee YJ, Kim CH, Park HG, Han SI, Kang HS (2018). Oncogenic Metabolism Acts as a Prerequisite Step for Induction of Cancer Metastasis and Cancer Stem Cell Phenotype. Hindawi; p.28.

Liaudet-Coopman E, Beaujouin M, Derocq D, Garcia M, Glondu-Lassis M, Laurent-Matha V, Prébois C, Rochefort H, Vignon F (2006). Cathepsin D: newly discovered functions of a long-standing aspartic protease in cancer and apoptosis. Cancer letters, 237(2):167–179.

Patanaphan V, Salazar OM, Risco R (1988). Breast cancer: metastatic patterns and their prognosis. South Med J; 81(9):1109-1112.

Pavlova NN, Thompson CB (2016). The Emerging Hallmarks of Cancer Metabolism. Cell metabol; 23(1): 27-47.

Rauw WM (2012). Immune response from a resource allocation perspective. Frontiers in genetics, 3:267.

Reiter JG, Makohon-Moore AP, Gerold JM, Heyde A, Attiyeh MA, Kohutek ZA, Tokheim CJ, Brown A, DeBlasio RM, Niyazov J, Zucker A, Karchin R, Kinzler KW, Iacobuzio-Donahue CA, Vogelstein B, Nowak MA (2018). Minimal functional driver gene heterogeneity among untreated metastases. Science; 361(6406):1033-1037.

Seyfried TN, Huysentruyt LC (2013). On the origin of cancer metastasis. Critical reviews in oncogenesis, 18(1-2): 43–73.

Shah MK, Kamboj M (2018). Immunizing Cancer Patients: Which Patients? Which Vaccines? When to Give? Cancer Network. https://www.cancernetwork.com/oncology-journal/immunizing-cancer-patients-which-patients-which-vaccines-when-give (Accessed March 1, 2020)

Thermo Fischer Scientific (2020). Allergens, Phadia Immunology Reference Laboratory. https://www.thermofisher.com/content/dam/tfs/SDG/IDD/PiRL/538631.01%20Allergen%20Component%20Cross-Reactivity%20Map%20042512.pdf (Accessed March 1, 2020)

Tu SH, Chang CC, Chen CS, Tam KW, Wang YJ, Lee CH, Lin HW, Cheng TC, Huang CS, Chu JS, Shih NY, Chen LC, Leu SJ, Ho YS, Wu CH (2010). Increased expression of enolase alpha in human breast cancer confers tamoxifen resistance in human breast cancer cells. Breast cancer research and treatment, 121(3):539-553.

Wang T, Huo X, Chong Z, Khan H, Liu R, Wang T (2018). A review of S100 protein family in lung cancer. Clin Chim Acta; 2476:54–59.