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Editorial

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Cell surface vimentin (CSV) redefines CTCs to monitor cancer progression in patients

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Abhisek Mitra
University of Texas MD Anderson Cancer Center, USA

Correspondence: Mitra, The University of Texas MD Anderson Cancer Center, 1515 Holcombe Blvd, Houston, USA, Tel 713-563-0440, Fax 713-534-5871, Email abhisekmi@gmail.com

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Editorial

Vimentin, a 57kDa intermediate filament protein, is normally expressed in mesenchymal cells. As a major cytoskeleton component of mesenchymal cells, it maintains cellular integrity, stabilizes cytoskeleton interactions and provides resistance under mechanical stress.[1] Biological characterization of Vimentin null cells suggested that it is important for mechanical stability and motility in vitro.[2] Furthermore Vimentin null mice had impaired wound healing without causing any other major physiological disorders.[3] Based on these studies, it can be inferred that Vimentin might have a supporting role in mechanical stability and integrity of cellular structure. In the recent years, understanding of the biological significance of Vimentin has been increasing steadily after its discovery as an epithelial mesenchymal transition (EMT) marker in aggressive cancer cells of epithelial origins- breast, prostate, colon, skin, lung and brain.[4] Inducible expression of Vimentin in large number of epithelial cancer correlates well with metastatic phenotypes in patients.[5,6] The possible explanation for Vimentin expression in epithelial cancer cells undergoing EMT process is to enhance migration and invasiveness.[7]

The dissemination of EMT positive cells is an important step in metastatic progression of cancer in patients. These EMT positive cells transform into circulating tumor cells (CTCs), which migrate from primary site to metastatic regions. Thus determining the number of CTCs plays an essential role in proper prognosis of tumor progression during therapeutic regimen. CTCs are heterogeneous population comprising of epithelial, EMT and mesenchymal types. Recent studies showed that a subpopulation of CTCs expresses Vimentin on the cell surface.[8,9] Interestingly, these cells also stained positively with established EMT markers such as Snail, Slug and Twist. The EMT bearing CTCs have acquired clinical significance in breast cancer patients.[6,10] In a comparative study between early and metastatic patients of breast cancer, EMT markers Vimentin and twist expressing CTCs were shown to increase from ~73-77% in the former to ~100% in the latter.[6] These findings suggest that an increase in EMT-positive CTC population in patients could indicate the progression of cancer towards drug-resistance or dormant phenotype or both besides the canonical concept of metastasis. Though, several technologies are available to capture CTCs from blood, enrichment of EMT positive CTCs is a prerequisite to design efficacious therapy for cancer patients.[11]

Cell surface Vimentin detecting antibody provides a powerful tool to isolate EMT positive population from a pool of heterogeneous CTCs. Currently, EpCAM and cytokeratins are considered the primary markers for the detection of circulating tumor cells.[12] These markers are preferentially detecting only epithelial CTCs not the EMT positive CTCs. It is a potential limitation in understanding the etiology of disease progression in advanced stage cancer patients which is required for designing suitable therapeutic strategies for both advanced and relapsed patients. The discovery of the novel CTC marker Vimentin bridges the gap in detecting between primary and metastatic stages in cancer progression.

Also Vimentin has been shown as a universal CTC marker for mesenchymal tumors.[8] Currently there is no suitable marker to detect CTCs from sarcoma patients in pre- and post-therapeutic periods. This antibody (CSV) can be used as a novel diagnostic tool in clinics to understand disease progression in sarcoma patients. Understanding the dynamics of EMT positive CTCs will revolutionize personalized cancer therapy. With the advancement of technological sophistication and superior sensitivity, CSV can be used as a noninvasive tool for monitoring tumors with heterogeneous genotypes in metastatic and relapsed patients. Furthermore, this antibody might be a powerful tool to the existing approaches to isolate cancer stem cells having EMT phenotypes which might provide better understanding of genomic, proteomic and epigenomic aberrations in metastasis, dormancy and relapse in cancer patients.[13]

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Conflicts of interest

Author declares there are no conflicts of interest.

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