REVIEW ARTICLE

Microparticles: a component of various diseases

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KEY WORDS

cardiovascular disease, cellular biology, cellular interactions, microparticles, microvesicles

ABSTRACT

Microparticles (MPs) are phenotypically and functionally heterogeneous population of microvesicles. Although MP formation represents a physiological phenomenon. A multitude of pathologies, including inflammatory and autoimmune diseases, atherosclerosis, and malignancies, are associated with a considerable increase in circulating MPs. Elevated levels of platelet-, endothelial cell-, and monocyte-derived MPs have been documented in a number of clinical conditions in which vascular dysfunction and inflammation are important pathophysiological mechanisms (e.g., coronary artery disease or thrombotic microangiopathies). Knowledge of the functional properties of MPs will contribute to a better understanding of the pathological mechanisms of communication between cells and of the causes of various diseases.

Introduction In the 1940s, it was shown that clotting of platelet-poor plasma was prolonged after high-speed centrifugation.[1] This observation suggested that procoagulant subcellular particles could be present in plasma, which could be removed by sedimentation. In 1967, Wolf[2] demonstrated that activated platelets shed membrane fragments on electron microscopy, which he called platelet dust. Microparticles (MPs) are released from the cell surface following cell activation or apoptosis by a number of triggers including chemical stimuli, such as cytokines, thrombin, and endotoxin, or physical stimuli, such as shear stress or hypoxia.[3]

MPs represent a heterogeneous population of vesicles (TABLE). They are submicron (diameter of 100 to 1000 nm) and are released by budding and fission of the plasma membrane. Moreover, they express antigens specific for their parental cells. MPs can be detected in various biological fluids, peripheral blood,[4] urine,[5] ascitic fluids,[6] and synovial fluids.[7] Biological function of MPs depends on their origin. For example, microvesicles secreted by skeletal cells initiate bone mineralization,[8] whereas those secreted by normal endothelial cells have been implicated in angiogenesis.[9] Microvesicles packaged with microRNAs (miRNAs) or Minas have been shown to be released mainly from progenitors of differentiated cells and tumor cells.[10,11] This review summarizes the current literature relevant to MPs and information about their role in various diseases.

Composition of microparticles Various eukaryotic cell types release membrane-derived microvesicles under specific physiological or pathological conditions. Interestingly, this phenomenon seems conserved during evolution. Bacteria release microvesicles that are important components of biofilms.[12]

Vesiculation is a physiological mechanism that is used in cell growth, activation, and protection. For example, for mineral formation in cartilage, bone, and predentin, calcification is initiated by matrix vesicles released by chondrocytes, osteoblasts, and odontoblasts.[13] Vesicle shedding is also an important defense mechanism protecting against complement attack, by allowing the removal of the C5b-9 attack complex from the cell surface by a calcium (Ca++)-dependent elimination as shown for many cell types including

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TABLE Characteristics of secreted vesicles

Feature	Microparticles[3,28]	Exosomes[29,30]
size, nm	100–1000	50–100
sedimentation, g	20,000	100,000
origin	plasma membrane	multivesicular, internal compartments
annexin-V-binding capacity	high annexin-V-binding capacity	no/low annexin-V-binding capacity

platelets, polymorphonuclearleucocytes, erythrocytes, and oligodendrocytes.[14] Specific vesiculation is triggered or enhanced in pathological conditions such as inflammation, injury, vascular dysfunction, or cancer.[15]

The composition of microvesicles, however, depends largely on the cell type from which they originate, although the membrane composition of microvesicles remains distinct from that of the parental cell, often with significant remodeling that enables specialized functions. In this regard, not all plasma-membrane proteins are incorporated into shed vesicles.[16] Phosphatidylserine is relocated to the outer membrane leaflet, specifically at sites on the cell surface where microvesicle shedding occurs, while the topology of membrane proteins remains intact.[16-18] As recently shown in tumor cells, phosphatidylserine externalization occurs presumably in an effort to quell an immune response and promote tumor-cell survival.[19]

Cellular interactions MPs bear antigens of their cell of origin and can transfer these surface molecules to other cell types. In doing so, they may alter the biological activity of the recipient cells. Additionally, the binding of MP surface antigens to their specific counter-receptor may induce intracellular signalling pathways.[20]

For instance, in the context of delivery of the chemokine RANTES/CCL5 to inflamed endothelium, P-selectin and glycoprotein Ib (GPIb) on platelet MPs mediate transient interactions with endothelial cells, thus arranging a higher frequency of interactions with the endothelial surface, which culminates in considerable CCL5 deposition.[21] Furthermore, outside-in signaling mechanisms involving GPIIb/IIIa on MPs are additionally operative in CCL5 release and transfer. Similarly, incorporation of MPs is dependent on their expression profile of surface receptors and on the type and activation status of the target cells.[22]

Microparticle transfer There are several mechanisms by which MPs mediating intercellular signaling may be discerned. For example, MPs may act as circulating signaling modules affecting cellular properties and responses by activation of receptors on the target cell via presentation of membrane-associated, bioactive molecules. Also, MPs may mediate signaling by directly transferring part of their content or components including proteins, bioactive lipids, or RNA to the recipient cell, potentially resulting in cell activation, phenotypic modification, and reprogramming of cell function. This transfer may be sufficiently facilitated by transient interactions, or may require firm association, membrane assimilation, or definitive incorporation of MPs into the target cell.[23-25] MPs are the way by which cells communicate with each other or at a greater distance.

Recent studies have even suggested that central nervous system-derived vesicles may enter the bloodstream and interact with endothelial cells in the peripheral circulation, representing a novel communication channel between the nervous and cardiovascular systems.[26] Based on their ability to transfer part of their components and content to target cells, MPs quantitatively and qualitatively complement traditional methods of intercellular communication, such as direct secretion of signaling molecules, physical interaction of membrane proteins, and involvement of gap junctions.[27-30]

Microvesicles and cancer Microvesicles in cancer patients were first documented in 1978, when they were identified in cultures of spleen nodules and lymph nodes obtained from a male patient with Hodgkin disease.[31] Microvesicles shed from tumor cells facilitate the transfer of soluble proteins,[32] nucleic acids,[33] functional transmembrane proteins,[34] chemokine receptors,[35] tissue factor,[34] and receptor tyrosine kinases such as epidermal growth factor receptor and human epidermal growth factor receptor 2.[36,37]

Angiogenesis is vital for tumor survival and tumor growth and occurs by proliferation of endothelial cells to form a mesh of blood vessels that infiltrate into the tumor, facilitating the supply of nutrients and oxygen for tumor growth as well as removal of waste products.[38]

Lipids from microvesicles can affect endothelial-cell migration and angiogenesis. For example, sphingomyelin, a major component of microvesicles shed from the fibrosarcoma cell line HT1080, together with vascular endothelial growth factor (VEGF), was shown to confer migratory and angiogenesis-inducing properties on endothelial cells.[39] Thus, microvesicles secreted by tumor cells induce endothelial cells to release microvesicles which contain VEGF and sphingomyelin to promote angiogenesis.[40] It is interesting that in lung cancer models, hypoxia induces an increased release of microvesicles.[41]

Cancer cells interact with the stroma and actively modify the microenvironment to favor their own progression.[42] Accordingly, a recent study by Castellana et al.[43] highlighted a mechanism of reciprocal communication between cancer cells and microvesicles. In this study, microvesicles released by PC3 cells, an invasive prostate cancer cell line, triggered extracellular signal-regulated kinase phosphorylation, matrix metallopeptidase 9 upregulation, increased motility, and resistance to apoptosis in fibroblasts in the surrounding

microenvironment. In turn, the activated fibroblasts shed microvesicles to facilitate the migration and invasion of the prostate cancer line. Microvesicles released by lung cancer cells also activate and chemoattract stromal fibroblasts as well as endothelial cells to facilitate tumor cell growth.[41]

Direct fusion of microvesicles produced by human melanoma or colorectal carcinoma cells with monocytes inhibited the differentiation of monocytes to antigen-presenting cells both in vitro and in vivo.[44]

Hypothetically, cancer cells can fuse with microvesicles derived from noncancer cells to camouflage behind the lipids and membrane-specific proteins of nontransformed cells.[40] A study by Tesselaar et al.[45] identified a low number of circulating microvesicles from cancer patients that stained for both mucin 1, a cancer-cell marker, and GPIIIa, a protein that is exclusively present on platelets.

Given the importance of matrix degradation in tumor metastases, it is logical to hypothesize that there is a direct correlation between the number of invasive microvesicles and tumor progression. Indeed, protease-loaded membrane vesicles with invasive properties have been observed in malignant ovarian ascites that are derived from women with ovarian cancer stage I to IV.[46] This study also showed that late-stage ascites contained substantially more vesicles than those in early-stage disease, although the invasive ability of the vesicles was approximately the same, irrespective of the disease stage.

Microparticles and cardiovascular disease Endothelium-derived nitric oxide (NO) is the major mediator of acetylcholine-induced vasorelaxation of rat aorta in vitro. Exposure of the rat aorta to endothelial MPs (EMPs) obtained from cultured endothelial cells resulted in impaired acetylcholineinduced relaxation and reduced NO production.[47] The same effect was seen using circulating MPs obtained from patients after myocardial infarction (MI).[48] This response was abolished by the removal of the endothelium or by inhibition of NO synthetase. The effect was not seen with nonischemia-induced MPs or the MP supernatant. Of note, this effect was seen with MI--induced MPs at 3 times lower concentrations than nonischemic MPs, suggesting qualitatively different biological activity.[48]

Procoagulant MPs, particularly EMPs, are elevated in patients with acute coronary syndromes compared with patients with stable anginal symptoms or control subjects.[49] This reflects the degree of acute vascular injury and inflammation at the time of measurement.

EMPs and platelet MPs were measured in 25 patients with deep vein thrombosis or pulmonary embolism, compared with healthy controls.[50] EMP levels were markedly elevated in patients with venous thromboembolism (VTE) compared with controls. Platelet MPs were not elevated despite higher platelet expression of the activation marker, P-selectin. Increased leukocyte expression of the activation marker, CD11b, and EMP-monocyte conjugates in VTE patients was also observed. The observed elevation of EMPs reflects the state of endothelial activation in VTE. EMPs may also contribute to thrombus development by localizing the inflammatory effects of leukocytes at sites of endothelial injury and providing a source of TF and a catalytic phospholipid surface themselves. Persistent D-dimer elevation following a period of anticoagulation for VTE is predictive of recurrence and may reflect ongoing hypercoagulability. Similar studies of MPs in this setting may also be useful to provide further evidence about the ongoing state of endothelial activation in these patients.[51]

Conclusion Over the last decade, it has been revealed that MPs from the cell are suitable forms of communication cells. MPs may be detected in blood and other fluids. Their phospholipid membrane originates from the native cell membrane, and there are many recepoters on their surface. Possibly, there is a complex system governing the mechanisms of MP formation.

It is currently unknown how secretion vesicles are organized, and whether a particular area of the plasma membrane that encapsulates the particle is randomly selected. It is also unknown what determines the particle size or what determines the surface which will form MPs from the plasma membrane. Is it relevant that some MPs can enter the cell and merge with its membrane via endocytosis? MPs may change the form of intracellular signal by affecting the various cascades, namely, the winding surface receptors, and thus block or amplify the signal. The size of MPs allows penetration through the plasma membrane and enables to enter cell structures. The potential proangiogenic effect of MPs may have a great therapeutic importance in the future because of the possibility of postischemic induction of neovascularization.

One of the causes of various disease states may be defective formation and the release of vesicles. In addition, an interesting finding is that MPs contain lipids, soluble proteins, nucleic acids, functional transmembrane proteins, chemokine receptors, and TF. From this perspective, the question arises whether a cell may send MPs to the target cell and produce a complex structure with a scheduled task, for example, to influence metabolism, growth, division, or resistance.

Other mysteries include the relationship between cancer and vesicles. How MPs from tumor cells affect immunity? Are they source of their spread? And also, how do they affect the surrounding healthy tissue? What is the role of MPs in the differentiation, proliferation, and tumor aggressiveness? Reliable studies should determine the number of MPs in benign and malignant tumors. By determining the quantitative and qualitative differences in MPs between the two types of tumors, the role of MPs in tumor development

could be clarified. It would be interesting to identify a link between their quantity and proteolytic properties (associated with the presence of matrix metalloproteases) and different levels of invasiveness of malignant and benign tumors. This could be a new possibility for determining tumor malignancy.

One possible defense against disease is the production of MPs by healthy cells to inform their neighborhood about a pathological state, inflammation, apoptosis, immune responses, and alteration of intracellular and extracellular environment. That is how healthy cells activate their defense mechanisms.

The future offers us far-reaching possibilities as concerns the use of artificial MPs. Currently, nanoscience is trying to make nanoparticles able to improve detection and assistance in therapy. MPs contain medicine which is sended to the host cell where it affects cell biology. If we discover and understand the cellular mechanisms that occur in this area, we can program the cell to be able to avoid unnecessary substances and communicate with the surrounding cells. MPs will be able to evaluate the physiological and pathophysiological processes of the cell. These conclusions are for now more a desire than reality, but perhaps they offer a useful vision of where to direct our research efforts.

Note I apologize to authors whose work I have not cited or only cited indirectly owing to space constraints.

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ARTYKUŁ POGLĄDOWY

Mikrocząstki – składowa wielu chorób

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SŁOWA KLUCZOWE

biologia komórkowa, choroby układu sercowo-naczyniowego, mikrocząstki, oddziaływania międzykomórkowe

STRESZCZENIE

Mikrocząstki są zjawiskiem niejednorodnym fenotypowo i czynnościowo. Tworzenie mikrocząstek ma charakter fizjologiczny, ale wiele stanów patologicznych, takich jak choroby zapalne i autoimmunologiczne, miażdżyca oraz nowotwory złośliwe, łączy się ze znacznym zwiększeniem liczby krążących mikrocząstek. W stanach chorobowych, w których dochodzi do zaburzonej czynności śródbłonka oraz do zapalenia jako ważnych mechanizmów fizjopatologicznych (np. w chorobie naczyń wieńcowych lub w mikroangiopatiach zakrzepowych), wykazano zwiększenie stężenia mikrocząstek pochodzących z płytek krwi, komórek śródbłonka naczyń i monocytów. Poznanie właściwości funkcjonalnych mikrocząstek pozwoli na lepsze zrozumienie mechanizmów patofizjologicznej komunikacji pomiędzy komórkami i lepsze poznanie mechanizmów rozwoju chorób.

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