

Extracellular Vesicles as Mediators of Immune Cell Communication in Inflammatory Disease

Caelan Rios*

Department of Cellular and Molecular Immunology, Highland Coast University, Valparaíso, Chile

DESCRIPTION

Cells within the immune system communicate continuously to coordinate defense against infectious organisms, remove damaged tissue, and preserve physiological balance. Although cytokines, chemokines, and direct receptor interactions have traditionally received considerable attention, another communication system has gained increasing recognition during recent years. Extracellular vesicles have emerged as biologically active particles capable of transferring proteins, lipids, nucleic acids, enzymes, and signaling molecules between neighboring and distant cells. These membrane-enclosed structures are released by nearly every cell type and participate in numerous physiological and pathological processes. Their influence on immune regulation has become an important subject within cellular and clinical immunology because they contribute to inflammatory activity, antigen presentation, tissue repair, and disease progression across multiple clinical conditions.

Extracellular vesicles are heterogeneous in size, composition, and cellular origin. Their contents reflect the physiological condition of the cells from which they are released. Under normal conditions, these vesicles contribute to tissue maintenance by supporting communication between immune cells and structural cells. During infection, inflammation, or cellular stress, their molecular cargo changes substantially, allowing transmission of signals that influence immune behavior throughout the body. This ability to transport complex biological information distinguishes extracellular vesicles from soluble mediators that act through individual receptor-ligand interactions.

Immune cells release extracellular vesicles throughout their life cycle. Macrophages, dendritic cells, neutrophils, T lymphocytes, B lymphocytes, and natural killer cells all contribute to circulating vesicle populations. Each immune cell type produces vesicles containing distinct combinations of proteins, messenger Ribonucleic Acid (RNA), microRNA, lipids, and enzymes. After reaching recipient cells, these molecules influence gene expression, intracellular signaling pathways, and functional characteristics. Consequently, extracellular vesicles participate in

highly coordinated communication networks that extend beyond direct cellular contact.

Macrophage-derived extracellular vesicles illustrate the adaptability of immune communication. Macrophages respond rapidly to changes within the tissue environment, and their vesicles reflect these functional adjustments. During inflammatory conditions, macrophage-derived vesicles frequently contain mediators capable of attracting leukocytes, stimulating cytokine production, and modifying endothelial cell behavior. Under conditions associated with tissue recovery, vesicles released by macrophages may instead promote angiogenesis, extracellular matrix remodeling, and cellular survival. These contrasting biological activities demonstrate that extracellular vesicles mirror the physiological state of their parent cells rather than representing fixed signaling entities.

Dendritic cells also produce extracellular vesicles with considerable immunological significance. These vesicles may contain major histocompatibility molecules loaded with antigenic peptides together with costimulatory proteins involved in lymphocyte activation. Experimental observations indicate that dendritic cell-derived vesicles contribute to antigen presentation by interacting directly with T lymphocytes or by transferring antigen-presenting molecules to additional antigen-presenting cells. Such communication extends the influence of dendritic cells beyond their physical location and supports coordinated immune responses across lymphoid tissues.

T lymphocytes generate extracellular vesicles capable of influencing neighboring immune populations. These vesicles transport cytokines, signaling proteins, regulatory microRNAs, and membrane receptors that alter macrophage activity, dendritic cell maturation, and additional lymphocyte functions. Regulatory T-cell vesicles have attracted particular attention because they may suppress inflammatory responses through transfer of inhibitory molecules to recipient immune cells. This mechanism adds another dimension to immune regulation by permitting communication without continuous cell-to-cell contact.

Correspondence to: Caelan Rios, Department of Cellular and Molecular Immunology, Highland Coast University, Valparaíso, Chile, Email: caelan.rios@highlandcoastuni-cl.edu

Received: 09-Feb-2026, Manuscript No. JCCI-26-43921; **Editor assigned:** 11-Feb-2026, PreQC No. JCCI-26-43921 (PQ); **Reviewed:** 25-Feb-2026, QC No. JCCI-26-43921; **Revised:** 04-Mar-2026, Manuscript No. JCCI-26-43921 (R); **Published:** 11-Mar-2026, DOI: 10.35248/2155-9899.26.17.795

Citation: Rios C (2026). Extracellular Vesicles as Mediators of Immune Cell Communication in Inflammatory Disease. J Clin Cell Immunol. 17:795.

Copyright: © 2026 Rios C This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

B lymphocytes contribute to extracellular vesicle biology through the release of particles containing immunoglobulins, antigen-presenting molecules, and signaling proteins. These vesicles participate in communication with helper T lymphocytes and antigen-presenting cells while influencing antibody-mediated immune responses. Their composition changes according to activation status, illustrating the dynamic nature of vesicle-mediated signaling during immune responses. Continued investigation may clarify how B-cell vesicles contribute to autoimmune disease, chronic inflammation, and immune regulation.

Neutrophils, traditionally viewed as short-lived antimicrobial cells, also release extracellular vesicles with diverse biological functions. These vesicles influence endothelial permeability, leukocyte recruitment, coagulation pathways, and macrophage activation. Depending on the surrounding environment, neutrophil-derived vesicles may amplify inflammatory responses or contribute to inflammatory resolution. Such observations reinforce the concept that innate immune cells participate in complex regulatory networks extending beyond immediate antimicrobial defense.

Communication between immune cells and vascular endothelial cells is essential for controlling leukocyte migration during inflammation. Extracellular vesicles released by activated endothelial cells modify adhesion molecule expression, vascular permeability, and cytokine production. Conversely, immune cell-derived vesicles alter endothelial responses through transfer of inflammatory mediators and regulatory nucleic acids. This bidirectional communication influences the movement of circulating leukocytes into inflamed tissues while affecting local vascular function.

MicroRNAs transported within extracellular vesicles have become an area of considerable scientific interest. These small non-coding RNA molecules regulate gene expression after entering recipient cells. Individual extracellular vesicles often carry multiple microRNAs capable of influencing diverse signaling pathways simultaneously. Changes in microRNA composition have been reported during autoimmune disease, chronic infection, cardiovascular disorders, and malignant conditions. Their stability within membrane-enclosed vesicles enhances their capacity to function as long-distance signaling molecules throughout the circulation.

Inflammatory diseases affecting different organs demonstrate unique patterns of extracellular vesicle activity. In rheumatoid arthritis, vesicles derived from immune cells and synovial fibroblasts influence cartilage degradation, cytokine production, and leukocyte recruitment within affected joints. In inflammatory bowel disease, vesicles released by intestinal epithelial cells, resident immune populations, and infiltrating leukocytes contribute to mucosal inflammation and epithelial repair. Pulmonary inflammatory disorders likewise involve vesicle-mediated communication between alveolar macrophages, epithelial cells, and vascular endothelium. These observations indicate that vesicle biology adapts to tissue-specific physiological conditions while participating in common immunological mechanisms.

The intestinal microbiota also influences extracellular vesicle communication. Commensal bacteria release membrane vesicles containing microbial components capable of interacting with epithelial cells and resident immune populations. These bacterial vesicles contribute to immune education under physiological conditions while influencing inflammatory responses during intestinal disease. Alterations in microbial composition modify the nature of vesicle-mediated communication, potentially affecting both local and systemic immune activity. Investigation of host-microbial vesicle interactions continues to expand understanding of immune regulation beyond traditional concepts of cellular communication.

Clinical interest in extracellular vesicles extends beyond their biological functions because they may serve as accessible biomarkers. Vesicles circulate in blood, urine, saliva, cerebrospinal fluid, bronchoalveolar lavage fluid, and additional biological specimens. Their molecular contents often reflect pathological changes occurring within tissues before overt clinical symptoms become apparent. Measurement of vesicle-associated proteins, lipids, and nucleic acids may therefore support disease monitoring, assessment of inflammatory activity, and evaluation of treatment response. Standardization of laboratory methods remains necessary before widespread clinical implementation can occur.

Technological progress has substantially improved the study of extracellular vesicles. High-resolution flow cytometry, nanoparticle tracking analysis, electron microscopy, proteomic profiling, and next-generation sequencing collectively permit increasingly detailed characterization of vesicle populations. Single-vesicle analytical methods now allow investigators to distinguish heterogeneous subpopulations that previously appeared identical using conventional laboratory techniques. Integration of these technologies with computational analysis has expanded understanding of vesicle diversity and biological function across numerous diseases.

Despite these advances, several scientific challenges remain. Isolation procedures vary among laboratories, creating differences in vesicle purity and yield. Distinguishing extracellular vesicle subtypes remains technically demanding because overlapping physical characteristics complicate classification. In addition, biological variability among patients requires careful interpretation of experimental findings. International efforts directed toward methodological consistency will strengthen comparisons among studies and improve reproducibility within the field.

CONCLUSION

Age, metabolic health, environmental exposure, and existing medical conditions all influence extracellular vesicle production and composition. Chronic inflammatory disorders, obesity, diabetes mellitus, cardiovascular disease, and aging have each been associated with alterations in circulating vesicle profiles. These observations suggest that extracellular vesicle biology reflects the combined influence of systemic physiology and local tissue conditions.