1. Phylogeny  
   CSF-1R, also known as FMS or c-Fms, is a receptor tyrosine kinase that belongs to the class III receptor tyrosine kinase family and is most closely related to the platelet‐derived growth factor receptor (PDGFR), KIT, and FLT3 kinases (chitu2015thepdgfrreceptor pages 17-19, scheijen2002tyrosinekinaseoncogenes pages 9-10). Orthologs of CSF-1R have been characterized in a broad range of vertebrates, including humans, mice, and chickens, which underscores its evolutionary conservation and fundamental role in hematopoietic and immune cell regulation (chitu2015thepdgfrreceptor pages 27-31, harne2020roleofcsf1csf1r pages 49-53). Its evolutionary origin can be traced to the proto‐oncogene c‑fms, originally identified from studies on feline sarcomas, and gene duplication events within the PDGFR family have maintained its highly conserved domain architecture that is critical for its function (scheijen2002tyrosinekinaseoncogenes pages 9-10, chitu2015thepdgfrreceptor pages 36-38). This conservation in domain organization, particularly in the extracellular ligand‐binding modules and the intracellular kinase core, reflects its pivotal role in the regulation of mononuclear phagocyte lineage cells (chitu2015thepdgfrreceptor pages 17-19).
2. Reaction Catalyzed  
   CSF-1R catalyzes the transfer of a phosphate group from ATP to specific tyrosine residues on substrate proteins, including autophosphorylation of the receptor itself (chitu2015thepdgfrreceptor pages 5-8, xiong2011acsf1receptor pages 1-2). The chemical reaction can be summarized as:  
     ATP + [protein]-L-tyrosine → ADP + [protein]-L-tyrosine-phosphate + H⁺  
   This ATP-dependent phosphorylation reaction activates the receptor and creates phosphotyrosine docking sites that are subsequently recognized by regulatory proteins containing SH2 or PTB domains (chitu2015thepdgfrreceptor pages 46-48). Upon ligand binding and subsequent receptor dimerization, a wave of autophosphorylation events is initiated that propagates the intracellular signal (xiong2011acsf1receptor pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of CSF-1R is strictly dependent on ATP as the phosphate donor, with the enzymatic reaction requiring ATP binding and hydrolysis for phosphorylation to occur (chitu2015thepdgfrreceptor pages 5-8). In addition to ATP, the kinase activity is typically supported by divalent metal cations—most often magnesium (Mg²⁺)—which coordinate with ATP in the active site to facilitate phosphotransfer (xiong2011acsf1receptor pages 1-2). These cofactors are indispensable for enabling the receptor’s enzymatic function and ensuring the proper spatial orientation of ATP with respect to its target substrate (chitu2015thepdgfrreceptor pages 5-8).
4. Substrate Specificity  
   CSF-1R displays substrate specificity by phosphorylating select tyrosine residues within its own intracellular domain as well as on associated adaptor proteins that mediate downstream signaling. Critical autophosphorylation sites include Tyr-559, Tyr-697, Tyr-721, and Tyr-807, which act as docking sites for a variety of signaling effectors such as Src family kinases (SFKs), the p85 regulatory subunit of phosphoinositide 3-kinase (PI3K), and adaptor proteins like Grb2 (chitu2015thepdgfrreceptor pages 46-48, chitu2015thepdgfrreceptor pages 51-53). The local amino acid context around these tyrosine residues favors binding of effector proteins that contain SH2 or PTB domains, thus ensuring that phosphorylation by CSF-1R maintains a high degree of substrate selectivity that is vital for the specificity of downstream responses (chitu2015thepdgfrreceptor pages 46-48).
5. Structure  
   CSF-1R is a type I transmembrane protein composed of several distinct domains that collectively facilitate its function as a receptor tyrosine kinase. The extracellular region is organized into five immunoglobulin (Ig)-like domains labeled D1 through D5; domains D1 to D3 are primarily responsible for high-affinity binding of its ligands CSF-1 and IL-34, whereas domains D4 and D5 are critical for receptor dimerization upon ligand engagement (chitu2015thepdgfrreceptor pages 5-8, chitu2015thepdgfrreceptor pages 36-38). The receptor is synthesized as an approximately 130 kDa immature protein, which undergoes extensive N-glycosylation to become a mature glycoprotein of around 165 kDa—a modification that is essential for proper folding, trafficking, and ligand recognition (chitu2015thepdgfrreceptor pages 109-112).

The single transmembrane helix anchors the receptor into the plasma membrane, while the intracellular region is composed of a juxtamembrane domain, a split tyrosine kinase domain interrupted by a kinase insert, and a C-terminal tail. The juxtamembrane domain is involved in autoinhibition in the absence of ligand; binding of CSF-1 or IL-34 relieves this inhibition by promoting receptor dimerization and enabling catalytic activation (chitu2015thepdgfrreceptor pages 42-44). The kinase domain itself contains the activation loop, the C-helix, and a hydrophobic spine that are critical for catalytic activity. Crystal structure studies, with resolution down to approximately 2.7 Å, have elucidated the conformational changes that occur upon ligand binding and autophosphorylation, providing detailed insight into the mechanism of receptor activation (chitu2015thepdgfrreceptor pages 130-132). Moreover, the presence of multiple conserved tyrosine residues within the intracellular tail facilitates the recruitment of downstream signaling molecules and underscores the receptor’s complex role in signal transduction (chitu2015thepdgfrreceptor pages 51-53).

1. Regulation  
   The regulatory mechanisms governing CSF-1R activity are multifaceted and involve both positive and negative post-translational modifications. Ligand binding by CSF-1 or IL-34 triggers receptor dimerization and a cascade of autophosphorylation events, notably on tyrosine residues such as Tyr-559, Tyr-721, and Tyr-807, which are essential for relieving autoinhibition and initiating downstream signaling (chitu2015thepdgfrreceptor pages 46-48, chitu2015thepdgfrreceptor pages 51-53). These phosphorylation events serve as molecular switches, creating docking sites for critical adaptor proteins and enzymes—examples include the recruitment of Src family kinases via phosphorylated Tyr-559 and the binding of the p85 regulatory subunit of PI3K at Tyr-721 (chitu2015thepdgfrreceptor pages 46-48).

In addition, CSF-1R is subject to ubiquitination, a regulatory modification mediated primarily by the E3 ubiquitin ligases Cbl and Cbl-b; these enzymes tag the activated receptor for endocytosis and lysosomal degradation, thereby attenuating the signal and maintaining homeostatic control over receptor levels (huang2017cblandcblb pages 14-19, illig2011optimizationofa pages 1-2). The receptor’s extracellular region also undergoes N-glycosylation, which is essential for its maturation and proper cell surface expression (chitu2015thepdgfrreceptor pages 109-112). Negative regulation may further occur via dephosphorylation by specific protein tyrosine phosphatases, although the exact enzymes involved are not completely defined in the present context (mouchemore2012csf1signalingin pages 5-7). These combined regulatory strategies ensure that CSF-1R activation is tightly controlled, allowing for an appropriate cellular response to external cues and preventing aberrant signaling that could lead to disease (chitu2015thepdgfrreceptor pages 42-44).

1. Function  
   CSF-1R is a central mediator of monocyte and macrophage biology. It is expressed predominantly on cells of the mononuclear phagocyte system, including monocytes, tissue macrophages, osteoclasts, microglia, and dendritic cells (chitu2015thepdgfrreceptor pages 5-8, chitu2015thepdgfrreceptor pages 17-19). Activation of the receptor by its ligands, CSF-1 and IL-34, promotes proliferation, differentiation, and survival of these cells, thereby ensuring the homeostasis and rapid responsiveness of the immune system (chitu2015thepdgfrreceptor pages 8-11, chitu2015thepdgfrreceptor pages 22-25).

In the context of bone physiology, CSF-1R is essential for osteoclastogenesis, the process by which osteoclast precursors differentiate into mature bone-resorbing cells. This function is critical for normal bone remodeling and tooth development, a fact highlighted by the osteopetrotic phenotypes observed in mouse models lacking functional CSF-1R signaling (chitu2015thepdgfrreceptor pages 134-136, gow2013theroleof pages 49-53). Additionally, CSF-1R is involved in the reorganization of the actin cytoskeleton, leading to the formation of membrane ruffles and podosomes that underpin cell adhesion and migration; these processes are particularly relevant in tissue repair and in the invasive behavior of cancer cells (chitu2015thepdgfrreceptor pages 136-138, chitu2015thepdgfrreceptor pages 124-126).

Beyond immune cell regulation, CSF-1R signaling has important roles in reproductive biology and mammary gland development. The receptor is required for the normal development of milk ducts and acinar structures during pregnancy, as well as for overall fertility in both males and females (chitu2015thepdgfrreceptor pages 5-8, chitu2015thepdgfrreceptor pages 17-19). In the nervous system, CSF-1R expressed on microglia plays a crucial role in brain development and homeostasis, with its activity ensuring adequate clearance of apoptotic cells and maintenance of proper neural architecture (chitu2015thepdgfrreceptor pages 19-22, harne2020roleofcsf1csf1r pages 90-93). As such, CSF-1R functions as a master regulator, integrating extracellular signals to orchestrate a variety of cellular processes that collectively impact immune function, tissue remodeling, and development (chitu2015thepdgfrreceptor pages 22-25, gow2013theroleof pages 45-49).

1. Other Comments  
   The therapeutic targeting of CSF-1R has been an area of intense investigation due to its central role in macrophage and osteoclast biology, as well as its involvement in inflammatory and neoplastic diseases. Various small-molecule inhibitors, such as GW2580, PLX3397, and imatinib, along with neutralizing antibodies like AMG820 and IMC-CS4, have been developed to block the kinase activity of CSF-1R, thereby reducing macrophage infiltration, tumor-associated inflammation, and osteoclast-mediated bone resorption (chitu2015thepdgfrreceptor pages 124-126, illig2011optimizationofa pages 1-2, huang2017cblandcblb pages 14-19). Moreover, activating mutations in CSF-1R have been identified in several myeloid malignancies, whereas inactivating mutations have been linked to neurodegenerative disorders such as adult-onset leukoencephalopathy with axonal spheroids and pigmented glia (ALSP), highlighting the delicate balance required for normal receptor function (chitu2015thepdgfrreceptor pages 112-114, chitu2015thepdgfrreceptor pages 31-33).

In addition to oncological applications, CSF-1R inhibitors are being explored in the treatment of rheumatoid arthritis, where excessive macrophage activation contributes to chronic inflammation and joint destruction (gow2013theroleof pages 57-60, harne2020roleofcsf1csf1r pages 90-93). The receptor’s inhibition can also improve outcomes in models of central nervous system injury and neurodegeneration by modulating microglial activity (harne2020roleofcsf1csf1r pages 197-199). Collectively, these studies underscore CSF-1R as a validated therapeutic target whose modulation holds promise in a wide range of pathological conditions (chitu2015thepdgfrreceptor pages 124-126, gow2013theroleof pages 49-53).

1. References
2. Chitu, V., Caescu, C. I., Stanley, E. R., Lennartsson, J., Rönnstrand, L., & Heldin, C.-H. (2015). The PDGFR Receptor Family. In Receptor Tyrosine Kinases: Family and Subfamilies (pp. 373-538). doi:10.1007/978-3-319-11888-8\_10
3. Gow, D. J. (2013). The role of macrophage colony stimulating factor-1 (CSF-1) in postnatal growth. [Peer-reviewed article].
4. Harne, R. (2020). Role of csf1/csf1r signalling in avian macrophage biology. doi:10.7488/era/43
5. Huang, L. (2017). Cbl and cbl-b dictate csf-1r endocytic traffic and signaling in macrophages. [Peer-reviewed article].
6. Illig, C. R., Manthey, C. L., Wall, M. J., Meegalla, S. K., Chen, J., Wilson, K. J., et al. (2011). Optimization of a potent class of arylamide colony-stimulating factor-1 receptor inhibitors leading to anti-inflammatory clinical candidate JNJ-28312141. Journal of Medicinal Chemistry, 54, 7860-7883. doi:10.1021/jm200900q
7. Mouchemore, K. A., & Pixley, F. J. (2012). CSF-1 signaling in macrophages: pleiotrophy through phosphotyrosine-based signaling pathways. Critical Reviews in Clinical Laboratory Sciences, 49, 49-61. doi:10.3109/10408363.2012.666845
8. Scheijen, B., & Griffin, J. (2002). Tyrosine kinase oncogenes in normal hematopoiesis and hematological disease. Oncogene, 21, 3314-3333. doi:10.1038/sj.onc.1205317
9. Verstraete, K., & Savvides, S. N. (2012). Extracellular assembly and activation principles of oncogenic class iii receptor tyrosine kinases. Nature Reviews Cancer, 12, 753-766. doi:10.1038/nrc3371
10. Xiong, Y., Song, D., Cai, Y., Yu, W., Yeung, Y., & Stanley, E. R. (2011). A csf-1 receptor phosphotyrosine 559 signaling pathway regulates receptor ubiquitination and tyrosine phosphorylation\*. The Journal of Biological Chemistry, 286, 952-960. doi:10.1074/jbc.m110.166702
11. Zhang, C., Ibrahim, P. N., Zhang, J., Burton, E. A., Habets, G., Zhang, Y., et al. (2013). Design and pharmacology of a highly specific dual FMS and KIT kinase inhibitor. Proceedings of the National Academy of Sciences, 110, 5689-5694. doi:10.1073/pnas.1219457110

References

1. (chitu2015thepdgfrreceptor pages 109-112): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
2. (chitu2015thepdgfrreceptor pages 112-114): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
3. (chitu2015thepdgfrreceptor pages 124-126): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
4. (chitu2015thepdgfrreceptor pages 130-132): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
5. (chitu2015thepdgfrreceptor pages 134-136): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
6. (chitu2015thepdgfrreceptor pages 136-138): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
7. (chitu2015thepdgfrreceptor pages 17-19): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
8. (chitu2015thepdgfrreceptor pages 19-22): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
9. (chitu2015thepdgfrreceptor pages 22-25): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
10. (chitu2015thepdgfrreceptor pages 36-38): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
11. (chitu2015thepdgfrreceptor pages 42-44): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
12. (chitu2015thepdgfrreceptor pages 5-8): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
13. (gow2013theroleof pages 45-49): DJ Gow. The role of macrophage colony stimulating factor-1 (csf-1) in postnatal growth. Unknown journal, 2013.
14. (gow2013theroleof pages 57-60): DJ Gow. The role of macrophage colony stimulating factor-1 (csf-1) in postnatal growth. Unknown journal, 2013.
15. (huang2017cblandcblb pages 14-19): L Huang. Cbl and cbl-b dictate csf-1r endocytic traffic and signaling in macrophages. Unknown journal, 2017.
16. (illig2011optimizationofa pages 1-2): Carl R. Illig, Carl L. Manthey, Mark J. Wall, Sanath K. Meegalla, Jinsheng Chen, Kenneth J. Wilson, Shelley K. Ballentine, Renee L. DesJarlais, Carsten Schubert, Carl S. Crysler, Yanmin Chen, Christopher J. Molloy, Margery A. Chaikin, Robert R. Donatelli, Edward Yurkow, Zhao Zhou, Mark R. Player, and Bruce E. Tomczuk. Optimization of a potent class of arylamide colony-stimulating factor-1 receptor inhibitors leading to anti-inflammatory clinical candidate 4-cyano-n-[2-(1-cyclohexen-1-yl)-4-[1-[(dimethylamino)acetyl]-4-piperidinyl]phenyl]-1h-imidazole-2-carboxamide (jnj-28312141). Journal of Medicinal Chemistry, 54:7860-7883, Oct 2011. URL: https://doi.org/10.1021/jm200900q, doi:10.1021/jm200900q. This article has 46 citations and is from a highest quality peer-reviewed journal.
17. (mouchemore2012csf1signalingin pages 5-7): Kellie A. Mouchemore and Fiona J. Pixley. Csf-1 signaling in macrophages: pleiotrophy through phosphotyrosine-based signaling pathways. Critical Reviews in Clinical Laboratory Sciences, 49:49-61, Apr 2012. URL: https://doi.org/10.3109/10408363.2012.666845, doi:10.3109/10408363.2012.666845. This article has 75 citations and is from a peer-reviewed journal.
18. (scheijen2002tyrosinekinaseoncogenes pages 9-10): B. Scheijen and J. Griffin. Tyrosine kinase oncogenes in normal hematopoiesis and hematological disease. Oncogene, 21:3314-3333, May 2002. URL: https://doi.org/10.1038/sj.onc.1205317, doi:10.1038/sj.onc.1205317. This article has 268 citations and is from a domain leading peer-reviewed journal.
19. (xiong2011acsf1receptor pages 1-2): Y. Xiong, Da Song, Yunfei Cai, Wenfeng Yu, Y. Yeung, and E. Richard Stanley. A csf-1 receptor phosphotyrosine 559 signaling pathway regulates receptor ubiquitination and tyrosine phosphorylation\*. The Journal of Biological Chemistry, 286:952-960, Nov 2011. URL: https://doi.org/10.1074/jbc.m110.166702, doi:10.1074/jbc.m110.166702. This article has 59 citations.
20. (chitu2015thepdgfrreceptor pages 27-31): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
21. (chitu2015thepdgfrreceptor pages 31-33): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
22. (chitu2015thepdgfrreceptor pages 46-48): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
23. (chitu2015thepdgfrreceptor pages 51-53): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
24. (chitu2015thepdgfrreceptor pages 8-11): Violeta Chitu, Cristina I. Caescu, E. Richard Stanley, Johan Lennartsson, Lars Rönnstrand, and Carl-Henrik Heldin. The pdgfr receptor family. Receptor Tyrosine Kinases: Family and Subfamilies, pages 373-538, Jan 2015. URL: https://doi.org/10.1007/978-3-319-11888-8\_10, doi:10.1007/978-3-319-11888-8\_10. This article has 7 citations.
25. (gow2013theroleof pages 49-53): DJ Gow. The role of macrophage colony stimulating factor-1 (csf-1) in postnatal growth. Unknown journal, 2013.
26. (harne2020roleofcsf1csf1r pages 197-199): Rakhi Harne. Role of csf1/csf1r signalling in avian macrophage biology. Unknown journal, Jan 2020. URL: https://doi.org/10.7488/era/43, doi:10.7488/era/43. This article has 0 citations.
27. (harne2020roleofcsf1csf1r pages 49-53): Rakhi Harne. Role of csf1/csf1r signalling in avian macrophage biology. Unknown journal, Jan 2020. URL: https://doi.org/10.7488/era/43, doi:10.7488/era/43. This article has 0 citations.
28. (harne2020roleofcsf1csf1r pages 90-93): Rakhi Harne. Role of csf1/csf1r signalling in avian macrophage biology. Unknown journal, Jan 2020. URL: https://doi.org/10.7488/era/43, doi:10.7488/era/43. This article has 0 citations.