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Primary Research Field

Cell Biology & Immunology

Education

1982 B.S. in Chemical Engineering, Shenyang Inst. Of Chem.
Engineering, Shenyang, P.R. China
1992-1994 ESL Program, Univ. MA, Lowell, MA

Professional Experience

2004-present Sr. Research Assistant, Rhode Island Hospital, Providence, RI
2001-2004 Research Assistant, Rhode Island Hospital, Providence, RI
1999-2001 Research Assistant, Providence College, Providence, RI

Abstracts (published)

1. Grutkoski, P.S., Chen, Y., Chung, C.S., Ayala, A. 2002. Suppressor of cytokine signaling (SOCS)-3 expression is upregulated in polymicrobial sepsis. *Shock* 17:8S.
2. Ding, Y.L., Chung, C.S., Bray, S., Chen, Y., Grutkoski, P.S., Carlton, S., Ayala, A. 2003. Polymicrobial sepsis induces divergent effects on dendritic cell function in mice. *Surg. Infect.* 4:95.
3. Grutkoski, P.S., Chen, Y., Chung, C.S., Ayala, A. 2003. Factors responsible for suppressors of cytokine signaling (SOCS)-3 upregulation in polymicrobial sepsis is site-specific. *Shock* 19:2A
4. Grutkoski, P.S., Chen, Y., Chung, C.S., Cioffi, W.G., Ayala, A. 2003. Putative mechanism of hemorrhage-induced leukocyte hyporesponsiveness: induction of suppressor of cytokine signaling (SOCS)-3. *J. Trauma* 55:206.

5. Grutkoski, P.S., Chen, Y., Chung, C.S., Ayala, A. 2003. Differential regulation of SOCS proteins during sepsis. *J. Leuko. Biol.* 74:47 suppl.
6. Chung, C.S., Grutkoski, P.S., Chen, Y., Ayala, A. 2004. Mechanisms of induction of the suppressor of cytokine signaling (SOCS) upregulation following trauma and sepsis. *Shock* 21:44A
7. Chung, C.S., Grutkoski, P., Chen, Y., Ayala, A. 2004. Inhibition of MyD88 signaling or NF- κ B activation by pyrrolidine dithiocarbamate (PDTC) reduced SOCS3 expression during sepsis. *FASEB J.*, 18:A433.
8. Newton, S., Ding, Y., Chung, C.S., Chen, Y., Lomas-Neira, J.L., Cioffi, W.G., Ayala, A. 2004. Sepsis induced changes in macrophage co-stimulatory molecule expression: CD86 as a regulator of anti-inflammatory IL-10 response. *Surg. Infect.* 5:104 (Abst).
9. Chung, C.S., Grutkoski, P., Chen, Y., Ayala, A. 2004. Blockade of TLR-NF κ B signaling downregulate SOCS-3 expression during sepsis. *Shock* 21:80 Suppl. 2.
10. Garber, M.E., Chung, C.S., Chen, Y., Ayala, A. 2004. Altered interleukin 16 expression during polymicrobial sepsis. *Shock* 21:17 Suppl. 2.
11. Chen, Y., Chung, C.S., Jones, L., Ayala, A. 2004. Deficiency of BID, a pro-apoptotic member of the Bcl2 family activated by death receptors, improves septic survival. . *Shock* 21:30 Suppl. 2.
12. Chung, C.S., Chen, Y., Doughty, L., Ayala, A. 2004. Differential induction of SOCS3 by JAK/STAT-dependent and independent pathways during sepsis. *J. Endotoxin Res.* 10:366
13. Garber, M.E., Chung, C.S., Chen, Y., Ayala, A. 2005. Expression of interleukin 16 is altered in murine model of hemorrhagic shock. *Shock.* 23:38 Suppl. 3.
14. Huang, X., Chung, C.S., Chen, Y., Ayala, A. 2005. Sepsis induces differential expression of ITIM receptors on CD4+ T cells. *Shock.* 23:61 Suppl. 3.
15. Lomas-Neira, J., Chung, C.S., Perl, M., Chen, Y., Ayala, A. 2005. MIP-2 and KC differentially contribute to the neutrophil activational/phosphoprotein status resultant from hemorrhage. *Shock.* 23:40 Suppl. 3.
16. Chen, Y., Chung, C.S., Wilson, D., Jones, L., Ayala, A. 2005. The role of BID protein in sepsis induced apoptosis. *Shock.* 23:47 Suppl. 3.
17. Rachel, T.M., Perl, M., Chung, C.S., Chen, Y.P., Ayala, A. 2005. Involvement of endoplasmic reticular (ER) stress and caspase-12 activation in leukocyte apoptosis in the early course of polymicrobial sepsis. *Shock.* 23:50 Suppl. 3.

18. Chung, C.S., Chen, Y.P., Doughty, L., Ayala, A. 2005. JAK/STAT and TLR pathways differentially regulate SOCS3 protein expression during sepsis. *Shock*. 23:8 Suppl. 3.
19. Chen, Y., Chung, C.S., Ayala, A. 2006. The role of Myd88-independent, TRIF pathway in polymicrobial sepsis. *Shock*. 25:74 suppl
20. Huang, X., Chung, C.S., Chen, Y., Ayala, A. 2006. Sepsis induces elevated expression of inhibitory receptor PD-1 and its ligand PD-L1 on immune cells. *Shock*. 25:46 suppl
21. Lomas-Neira, J., Chung, C.S., Perl, M., Chen, Y., Ayala, A. 2006. Neutralization of KC and MIP-2: divergent effects on activation pathways in mouse pro-myelocytic cell-line (MPRO)-consistent with mouse blood PMN. *Shock*. 25:29 suppl
22. Chung, C.S., Chen, Y., Ayala, A. 2006. Hemorrhagic shock induces differential expression of SOCS-1 and SOCS-3 proteins. *Shock*. 25:61 suppl.
23. Huang, X., Chung, C.S., Chen, Y., Ayala, A. 2007. Antigen presenting cells contribute to impaired Th1 response through upregulating PD-1/B7-H1 expression. *Shock* 27:62 suppl 1.
24. Chung, C.S., Horner, B., Chen, Y., Ayala, A. 2007. Role of NKT cells in the immune dysfunction and injury in sepsis. *Shock* 27:52 suppl 1.
25. Chung, C.S., Chen, Y., Perl, M., Ayala, A. 2007. Silencing of SOCS-3 reduces lung inflammation, neutrophil influx and injury after hemorrhagic shock (hem) and sepsis. *J. Leukocyte Biol.* 82:(in press) suppl.
26. Huang, X., Chung, C.S., Chen, Y., Ayala, A. Antigen presenting cells contribute to impaired Th1 response through upregulating PD-1/B7-H1 expression. *Shock* 2007; 27:62 suppl 1.
27. Venet, F., Lomas-Neira, J., Chen, Y., Chung, C.S., Ayala, A. 2008. Dendritic cells as anti-inflammatory regulators of extra-pulmonary acute lung injury. *FASEB J.* 22:848.9.
28. Huang, X., Wang, Y.L., Venet, F., Swan, R., Chen, Y., Chung, C.S., Lepape, A., Monneret, G., Ayala, A. PD-1 deficiency protects mice from the lethality of sepsis by balancing efficient pathogen clearance and suppression of pro-inflammatory cytokine production. *FASEB J.* 2008; 22:675.23.
29. Venet, F., Lomas-Neira, J., Chen, Y., Chung, C.S., Ayala, A. 2008. Mechanisms of extra-pulmonary acute lung injury: the dendritic cell as a regulator of the macrophage's inflammatory response. *Shock* 29:47 (Suppl. 1).
30. Venet, F., Lomas-Neira, J., Huang, X., Chen, Y., Chung, C.S., Ayala, A. 2008. Mechanisms of extra-pulmonary acute lung injury: the dendritic cell as a regulator of macrophage's recruitment and inflammatory response. *J. Leukocyte Biol.* 84:59 suppl.

31. Huang, X., Venet, F., Lepape, A., Yuan, Z., Chen, Y., Monneret, G., Chung, C.S., Ayala, A. 2009. A pathological role for PD-1 in sepsis induced lethality: dysregulating the balance between efficient monocyte/macrophage mediated pathogen clearance, cell death and the innate inflammatory response. *Shock* 31: 16 suppl.

Publications

1. Grutkoski, P.S., Chen, Y., Chung, C.S., Ayala, A. 2003. Sepsis-induced SOCS-3 expression is immunologically restricted to phagocytes. *J. Leukoc. Biol.* 74:916-922.
2. Grutkoski, P.S., Chen, Y., Chung, C.S., Cioffi, W.G., Ayala, A. 2004. Putative mechanism of hemorrhage-induced leukocyte hyporesponsiveness: induction of suppressor of cytokine signaling (SOCS)-3. *J. Trauma* 56:742-748.
3. Ding, Y., Chung, C.S., Bray, S., Chen, Y., Grutkoski, P.S., Carlton, S., Albina, J.E., Ayala, A. 2004. Polymicrobial sepsis induces divergent effects on splenic and peritoneal dendritic cell function in mice. *Shock*. 22:137-144.
4. Newton, S., Ding, Y., Chung, C.S., Chen, Y., Lomas-Neira, J.L., Ayala, A. 2004. Sepsis induced changes in macrophage co-stimulatory molecule expression: CD86 as a regulator of anti-inflammatory IL-10 response. *Surg. Infect.* 5:375-383.
5. Chung, C.S., Chen, Y.P., Grutkoski, P.S., Doughty, L.A., Ayala, A. 2007. The loss of SOCS-1 expression contributes to increased lymphoid apoptosis and decreased ability to survive sepsis. *Apoptosis* 12:1143-1153.
6. Huang X., Venet, F., Lepape, A., Yuan, Z., Wang, Y.L., Chen, Y., Swan, R., Kherouf, H., Monneret, G., Chung, C.S., Ayala, A. 2008. PD-1 on macrophages/monocytes: a critical role in suppressed innate immunity during sepsis. *P.N.A.S. USA* 106:6303-6308.
7. Venet, F., Chung, C.S., Huang, X., Lomas-Neira, J., Chen, Y., Ayala, A. 2008. Indirect lung injury: a role for regulatory T lymphocytes in the development of pulmonary inflammation. *J. Immunology* (in revision)
8. Venet, F., Huang, X., Chung, C.S., Monneret, G., Chen, Y., Ayala, A. 2009. Plasmacytoid dendritic cells control lung inflammation and monocyte recruitment in indirect acute lung injury in mice. *J. Immunology* (submitted)

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