BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
Chun-Shiang Chung	Assistant Professor of Surgery (Research)
eRA COMMONS USER NAME CSChung01	

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, and include postdoctoral training.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	YEAR(s)	FIELD OF STUDY
Chinese Culture University (Taipei, Taiwan)	B.S.	1980	Botany
National Chun-Hsing University (Taichung,	M.S.	1983	Botany
Colorado State University	M.S.	1991	Microbiology
University of Rhode Island	Ph.D.	1996	Biochemistry

A. POSITION AND HONORS

PAST EXPERIENCE:
Research Assistant, Institute of Microbiology, Medicine College of National Taiwan University, Taipei, Taiwan. 1983-85
Research Assistant, Institute of Botany, Academia Sinica, Taipei, Taiwan. 1986-87
Graduate Teaching and Research Assistant (Microbiology), Colorado State University, 1988-91
Graduate Teaching and Research Assistant (Microbiology, Chemistry), University of Rhode Island, 1992-96
Research Associate, Department of Surgery, Rhode Island Hospital/Brown University, 1996-2002
Instructor, Department of Surgery, Rhode Island Hospital/Brown University, 2002-2004
PRESENT: Assistant Professor (Res.), Department of Surgery, Rhode Island Hospital/Brown University 2004-present

MEMBERSHIPS IN SCHOLARLY SOCIETIES:
Shock Society, Society of Leukocyte Biology
Ad Hoc Reviewer: *Journals:* J. Surg. Res., Shock, Gastroenterology, Surgical Infections.

AWARDS:
URI Graduate Fellowship, University of Rhode Island, 1993-1994.
Travel Grant Award, the 21st Annual Conference on Shock, San Antonio, Texas, June, 1998.
Travel Grant Award, the 4th International Shock Congress, Philadelphia, PA, June 12-16, 1999.

B. SELECTED (from 55) PEER-REVIEWED PUBLICATIONS:

Mario, P., **Chung, C.S.**, Perl, U., Biffl, W.L., Cioffi, W.G., Ayala, A. 2007. Beneficial versus detrimental effects of neutrophils are determined by the nature of the insult. *J. Amer. Col. Surg.* (E-pub/in press)

Chung, C.S., Chen, Y.P., Grutkoski, P.S., Doughty, L.A., Ayala, A. 2007. SOCS-1 is a central mediator of steroid-increased thymocyte apoptosis and decreased survival following sepsis. *Apoptosis* (in press).

Winoski, N.C., **Chung. C.S.,** Cioffi, C., Ayala, A. 2007. The contribution of $CD4^+CD25^+$ T-regulatory cells to immune suppression in sepsis. *Shock.* (in press)

Chung, C.S., Watkins, L., Song, G.Y., Lomas, J., Cahoone, E.V., Cioffi, W.G., Ayala, A. 2006. Deficiency in $\gamma\delta$ T-lymphocytes compromises the ability of mice to survive polymicrobial sepsis. *Amer. J. Physiol.* 291:R1338-1343.

Doughty, L.A., Galen, B, Cooma-Ramberan, I., Carlton, S., **Chung, C.S.,** Ayala, A. 2006. Activation of common antiviral pathways can potentiate inflammatory responses to septic shock. *Shock* 26:187-194.

Lomas-Neira, J., **Chung, C.S.,** Perl, M., Gregory, S., Biffl, W., Ayala, A. 2006. Role of alveolar macrophage & migrating neutrophils in hemorrhage induced priming for ALI subsequent to septic challenge. *Amer. J. Physiol.* 290:L51-L58

Lomas-Neira, J.L., **Chung, C.S.,** Grutkoski, P.S., Carlton, S., Dunican, A., Simms, H.H., Cioffi, W.G., Ayala, A. 2005. Divergent roles of the murine neutrophil chemokines in hemorrhage induced priming for acute lung injury. *Cytokine* 31:169-179.

Song, G.Y., **Chung, C.S.,** Cioffi, W.G., Ayala, A. 2005. Insights into the contribution of STAT4 and STAT6 signaling to the morbidity seen in a low-mortality model of sepsis. *Intensive Care Med.* 31:1564-1569.

Lomas-Neira, J.L., **Chung, C.S.,** Grutkoski, P.S., Miller, E.J., Ayala, A. 2004. CXCR2 inhibition suppresses hemorrhage induced priming for acute lung injury in mice. *J. Leukoc. Biol.* 76:58-64.

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.

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.

Grutkoski, P.S., **Chung, C.S.,** Albina, J.E., Biffl, W., Ayala, A. Chapter. 39. Apoptosis in the Critically Ill. In: Textbook of Critical Care. 5th Edition, Ed: Fink, M.P., Abraham, E., Vincent, J-L., Kochanek. Elsevier Science, Philadelphia, PA, USA, (in press), 2004.

Grutkoski, P.S., Chen,Y., **Chung, C.S.**, Ayala, A. Sepsis induced SOCS3 expression is immunologically restricted to Phagocytes. *J. Leukoc. Biol.* 74:916-922, 2003.

Chung, C.S., Song, G.Y., Lomas, J., Simms, H.H., Chaudry, I.H., Ayala, A. Inhibition of Fas/Fas ligand signaling improves septic survival: differential effects on macrophage apoptotic and functional capacity. *J. Leuk. Biol.* 74: 344-351, 2003.

Lomas, J.L.,**Chung, C.S.,** Grutkoski, P.S., LeBlanc, B.W., Lavigne, L., Reichner, J., Gregory, S.H., Doughty, L.A., Cioffi, W.G., Ayala, A. 2002. Differential effects of MIP-2 and KC on hemorrhage induced neutrophil priming for lung inflammation: assessment by adoptive cell transfer in mice. *Shock* 19:358-365, 2003.

Ayala, A., **Chung, C.S.,** Lomas, J.L., Song, G.Y., Doughty, L.A., Gregory, S.H., Cioffi, W.G., LeBlanc, B.W., Reichner, J., Simms, H.H., Grutkoski, P.S. Shock induced neutrophil priming for acute lung injury in mice: divergent effects of TLR-4 and TLR-4/FasL deficiency. *Amer. J. Pathol.* 161:2283-2294, 2002.

Song, G.Y., **Chung, C.S.**, Jarrar, D., Cioffi, W.G., Ayala, A. Mechanism of immune dysfunction in sepsis: inducible NO mediated alterations in P38 MAPK activation. *J. of Trauma* 53:276-283, 2002.

Schwacha, M.G., **Chung, C.S.**, Ayala, A., Bland, K.I., Chaudry, I.H. Cyclooxygenase 2-mediated suppression of macrophage interleukin-12 production after thermal injury. *Amer. J. Physiol.* 282: C263-C270, 2002.

Yang, S.L., **Chung, C.S.**, Ayala, A., Chaudry, I.H., Wang, P. Differential alterations in cardiovascular responses during the progression of polymicrobial sepsis in the mouse. *Shock* 17: 55-60, 2002.

Chung, C.S., Wang, W., Chaudry, I.H., Ayala, A. Increased apoptosis in lamina propria B cells during polymicrobial sepsis is FasL but not endotoxin mediated. *Am. J. Physiol.* 280:G812-G818, 2001.

Chung, C.S., S.L. Yang, Song, G.Y., Lomas, J., Wang, P., Simms, H.H., Chaudry. I.H., Ayala, A. Inhibition of Fas signaling prevents hepatic injury and improves organ blood flow during sepsis. *Surgery* 130:339-45, 2001.

Song, G.Y., **Chung, C.S.**, Chaudry, I.H., Ayala, A. A novel mechanism by which p38 MAPK antagonism improves survival following polymicrobial sepsis: attenuation of developing immune suppression but not early pro-inflammation. *Amer. J. Physiol.* 281:C662-C669, 2001.

Song, G.Y., **Chung, C.S.,** Jarrar, D., Chaudry, I.H., Ayala, A. Evolution of immune suppressive macrophage phenotype as a product of p38 MAPK activation of polymicrobial sepsis. *Shock* 15:42-48, 2001.

Ayala, A., Song, G.Y., **Chung, C.S.**, Redmond, K.M., Chaudry, I.H. Immune hyporesponsiveness in polymicrobial sepsis: the role of necrotic (injured) tissue and endotoxin. *Crit. Care. Med*. 28:2949-2955, 2000.

Chung, C.S., Song, G.Y., Moldawer, L.L., Chaudry, I.H., Ayala, A. Neither Fas ligand nor TLR-4 mediated endotoxin is responsible for inducible peritoneal phagocyte apoptosis during sepsis/peritonitis. *J. Surg. Res.* 91:147-153, 2000.

Chung, C.S., Chaudry, I.H., Ayala, A. 2000. The apoptotic response of the lymphoid immune system to trauma, shock and sepsis. In: Yearbook of Intensive Care and Emergency Medicine-2000. Editors: Vincent, J.L. Springer-Verlag, Heidelberg, Germany pg 27-40. (Review)

Ayala, A., Xu, Y.X., **Chung, C.S.**, Chaudry, I.H. Does Fas ligand or endotoxin contribute to thymic apoptosis during polymicrobial sepsis? *Shock*, 11:211-217, 1999.

Samy, T.S.A., Schwacha, M.G., **Chung, C.S.**, Cioffi, W.G., Bland, K.I., Chaundry, I.H. Proteasome participates in the alteration of signal transduction in T and B lymphocytes following trauma-hemorrhage. *Biochem. Biophys.Acta* 1453(1):92-104, 1999.

Ayala, A., **Chung, C.S.**, Xu, Y.X., Evans, T.A., Redmond, K.M., Chaudry, I.H. Increased inducible apoptosis in CD4+ T-lymphocytes during polymicrobial sepsis is mediated by Fas ligand (FasL) and not endotoxin. *Immunology* 97: 45-55, 1999.

Chung, C.S., Xu, Y.X., Wang, W., Chaudry, I.H., Ayala, A. Is fas ligand or endotoxin responsible for mucosal lymphocyte apoptosis in septis? *Archives of Surgery* 133(11):1213-20, 1998.

Chung, C.S., Xu, Y.X., Chaudry, I.H., Ayala, A. Sepsis induces increased apoptosis in lamina propria mononuclear cells which is associated with altered cytokine gene expression. *J. Surg. Res.* 77:63-70, 1998.

Chung, C.S., Pearson, L.D., Ayers, V.K., Collins, J.K. Monoclonal antibodies that distinguish between encephalitogenic bovine herpesvirus type 1.3 and respiratory bovine herpesvirus type 1.1. *Clin. Diagn. Lab. Immnum.* 1:83-88, 1994.

Marcom, K.A., Pearson, L.D., **Chung, C.S.**, Poulson, J.M., DeMartini, J.C. Epitope analysis of capsid and matrix proteins of North American ovine lentivirus field isolates. *J. Clin. Microbiol.*29:1472-1479, 1991

C. RESEARCH PROJECTS (ONGOING OR COMPLETED DURING THE LAST 3 YEARS):

Immune Hyporesponsiveness in Shock: Role of SOCS-1 and SOCS-3 Proteins.
Principle Investigator: Chun-Shiang Chung
Agency: Shock Society/Novo Nordisk
Type: Fellowship in Hemostasis and Shock
Aims: The objectives of the study are to determine if changes of SOCS-1 and/or SOCS-3 expression pathologically suppresses the immune response in macrophages/lymphocytes following shock.

Programmed Cell Death: Role in Septic Immune Dysfunction (Ongoing)
Co-Investigator: Chun-Shiang Chung
Agency: NIH/GMS
Type: R01-GM53209-07; Period 6/30/04-5/31/08
Aims: The aim of this study is determine role of programmed cell death/apoptosis in the induction of lymphocyte/neutrophil/phagocyte immune dysfunction and to further determine mediators and cell-cell interactions which effect apoptosis in polymicrobial sepsis. P.I.: Alfred Ayala

Differential Effects of Sepsis on Macrophage Function. (Ongoing)
Collaborator: Chun-Shiang Chung
Agency: NIH/GMS
Type: R01-GM46354-07; Period: 10/01/03-9/30/08
Aims: The objectives of this project are to determine how sepsis, as opposed to chronic low dose endotoxin infusion, alters various macrophage, lymphocyte and hepatocyte functional capacities in mice.
P.I.: Alfred Ayala

Regulatory Mechanisms of Acute Lung Injury: Phagocyte Apoptosis. (Ongoing)
Co-Investigator: Chun-Shiang Chung
Agency: NIH/NIHLBI
Type: R01-GM63898-01; Period: 9/01/03-6/31/07
Aims: The global objective of this was to determine the contribution of the neutrophil apoptotic response to the development of acute lung injury induced by a salient two-hit model of hemorrhage (priming) followed by subsequent infectious polymicrobial septic challenge (triggering) in the mouse.
P.I.: Alfred Ayala