BIOGRAPHICAL SKETCH

NAME: Erickson, David

eRA COMMONS USER NAME: DAVID1981

POSITION TITLE: Associate Professor of Microbiology and Molecular Biology

EDUCATION/TRAINING

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
University of Lethbridge, Lethbridge AB Canada	BS	05/1999	Biochemistry
University of Calgary, Calgary AB Canada	Ph.D.	08/2003	Bacterial Pathogenesis
NIH/NIAID, Rocky Mountain Laboratories, Hamilton MT	Visiting fellow	08/2006	Bacterial Pathogenesis

A. Personal Statement

I have had extensive experience in the study of host-bacterial interactions, dissecting the molecular mechanisms that bacteria employ in overcoming the innate immune defenses of their hosts. During my Ph.D. training, I studied the virulence strategies of *Pseudomonas aeruginosa* in Cystic Fibrosis lung infections and in an insect infection model. I did postdoctoral training with Dr. Joseph Hinnebusch, Ph.D. at the Rocky Mountain Laboratories NIAID, NIH, in the area of *Yersinia* evolution and host adaptation. As an independent investigator, my lab has focused on how zoonotic pathogens resist innate immune defenses and adapt to inhabit specific environments. I am especially interested in bacterial pathogens that circulate in animals and in humans. This includes evolution of *Yersinia* biofilms, as well as niche adaptation of extraintestinal pathogenic strains of *Escherichia coli*. I am heavily focused on teaching and involvement of students in research. Undergraduate and graduate students participate in all aspects of our work, from developing hypotheses and planning experiments, to synthesizing data and presenting their work in scientific conferences and publications. These mentoring experiences have been made possible in part through previous support through the AREA R15 program. As a result, students have gone on to graduate degrees in public health, immunology, cell biology, and bacterial pathogenesis, while others have chosen M.D. programs. I have been able to pass on to my students my specific expertise in genetic manipulation of pathogens and conducting gene expression studies, functional genetic screens, and the use of alternative infection models in pathogenesis research.

B. Positions and Honors

Positions and Employment

1998-1999 Research associate, Agriculture Canada, Lethbridge, AB Canada
1999-2003 Graduate student, University of Calgary, Calgary, AB Canada
2003-2006 Postdoctoral fellow, Rocky Mountain Laboratories (NIH/NIAID)
2006-2012 Assistant Professor, Brigham Young University
2012-present Associate Professor, Brigham Young University

Service and Memberships

2009-2010 President, Intermountain Branch, American Society for Microbiology
2019-present Editorial board member, *Canadian Journal of Microbiology*
Ad-hoc reviewer Israeli Science Foundation
Ad-hoc reviewer *Microbes and Infection*
Ad-hoc reviewer *Microbial Pathogenesis*
Ad-hoc reviewer *Journal of Medical Entomology*

Ad-hoc reviewer *Virulence*
Ad-hoc reviewer *J Applied Microbiology*
Ad-hoc reviewer *Developmental and Comparative Immunology*
Ad-hoc reviewer *Frontiers in Cellular and Infection Microbiology*
Ad-hoc reviewer *Infection and Immunity*

Awards
2001-2003 Canadian Cystic Fibrosis Foundation graduate studentship (national award)

C. Contributions to Science

1. I have made significant contributions to understanding the evolution of *Yersinia pestis* from *Yersinia pseudotuberculosis*, specifically in the adaptation of *Y. pestis* to its lifestyle as a flea-borne pathogen. We showed that reduced biofilm formation and acute toxicity towards fleas are characteristics of *Y. pseudotuberculosis* that were key changes in the emergence of *Y. pestis*. I was the first author for each of these papers, and a portion this work was highlighted in *Nature Reviews Microbiology.*

a. **Erickson D.L**., Jarrett C.O., Callison J.A., Fischer E.R., Hinnebusch B.J.. 2008. Loss of a biofilm-inhibiting glycosyl hydrolase during the emergence of *Yersinia pestis*. J. Bacteriol. **190**:8163-70. PMID: 18931111
b. **Erickson D.L.**, Waterfield N.R., Vadyvaloo V., Long, D., Fischer E.R.., Ffrench-Constant R.H., Hinnebusch B.J. 2007. Acute oral toxicity of *Yersinia pseudotuberculosis* towards *Xenopsylla cheopis*: implications for the evolution of flea-borne transmission of *Yersinia pestis*. Cell. Microbiol. **9**:2658-66 PMID: 17587333
c. **Erickson D.L.**, Jarrett C.O., Wren B.W., Hinnebusch B.J. 2006. Serotype differences and lack of biofilm formation characterize *Y. pseudotuberculosis* infection of the *Xenopsylla cheopis* flea vector of *Yersinia pestis*. J. Bacteriol. **188**:1113-1119 PMID: 16428415

2. My early publications focused on the regulation of virulence factor production during *P. aeruginosa* infections. My major research accomplishments include publishing the first demonstration of *in vivo* production of quorum-sensing signals during lung infections in Cystic Fibrosis patients. In this paper we demonstrated that these systems actively control virulence factor production and may especially influence chronic vs. acute infections. This paper has been highly cited. We were able to show a role for the stringent response in regulating quorum sensing, and subsequent follow-up work (in which I contributed as a collaborator) has shown that this system is also indispensable for virulence in multiple infection models.

a. **Erickson D.L.**, Endersby R., Kirkham A., Stuber K., Vollman D.D., Mitchell I., Rabin H.R., and Storey D.G. 2002. *Pseudomonas aeruginosa* quorum-sensing systems may control virulence factor expression in the lungs of patients with cystic fibrosis. Infect. Immun.**70**:1783-1790. PMID: 11895939
b. **Erickson D.L.**, Lines L.J., Pesci E.C., Venturi V., and Storey D.G. 2004. The *Pseudomonas aeruginosa relA* contributes to virulence in *Drosophila*. Infect. Immun. **72**:5638-5645. PMID: 1538546
c. Vogt S.L., Green C., Stevens K.M., Day B., **Erickson D.L**., Woods D.E. and Storey D.G. 2011. The stringent response is essential for *Pseudomonas aeruginosa* virulence in the rat lung agar bead and *Drosophila melanogaster* feeding models of infection. Infect. Immun. **79**:4094-104 PMID: 21788391

3. My lab has made pioneering efforts to describe the relevant innate immune defenses of fleas, *Yersinia* genes required for counteracting innate immune defenses of insects and forming biofilms. Findings have advanced our understanding of the differences between *Y. pestis* and *Y. pseudotuberculosis* that explain their very different lifestyles and environmental niches, and regulatory pathways that influence bacterial survival. In each of these reports I oversaw the work and was the corresponding author.

a. Zhou W., Johnson K.L., Mortensen R.D, **Erickson D.L.** 2012. Gene expression analysis of *Xenopsylla cheopis* fleas suggests a role for reactive oxygen species in response to *Yersinia pestis* infection. J. Med. Entomol 49(2):363-70 PMID: 22493856
b. **Erickson D.L.**, Russell C.W., Johnson K.L., Hileman T., Stewart R.M. 2011. PhoP and OxyR transcriptional regulators contribute to *Yersinia pestis* virulence and survival within *Galleria mellonella*. Microbial Pathogenesis **51**:389-95 PMID: 21964409
c. Schachterle J.K., Stewart R.M., Schachterle M.B., Calder J.T., Kang H., Prince J.T., **Erickson D.L.** 2018. *Yersinia pseudotuberculosis* BarA-UvrY two-component regulatory system represses biofilms via

CsrB. Front Cell Infect Microbiol 8(13):323 PMID: 30280093

d. Calder J.T, Christman N.D., Hawkins J.M., **Erickson D.L.** A trimeric autotransporter enhances biofilm cohesiveness in *Yersinia pseudotuberculosis* but not in *Yersinia pestis.* bioRxiv **[Preprint]** 2020.03.31.019323; doi: https://doi.org/10.1101/2020.03.31.019323

4. In collaboration with Drs. Eric Wilson and Jovanka Voyich, we have investigated bacterial factors promote colonization of mucosal tissues. We have examined the effects of cell envelope structure on bacterial colonization and resistance to host antimicrobial chemokines CCL25 and CCL28. These chemokines are prevalent in mammary glands and in the intestines of mammals and may influence survival of pathogens like *Y. pseudotuberculosis* and mastitis-associated *Escherichia coli*. In my role as a co-investigator I helped design experiments and interpret data, as well as directing and training student researchers.

a. Pallister KB, Mason S, Nygaard TK, Liu B, Griffith S, Jones J, Linderman S, Hughes M, **Erickson D**, Voyich JM, Davis MF, Wilson E. 2015. Bovine CCL28 Mediates Chemotaxis via CCR10 and Demonstrates Direct Antimicrobial Activity against Mastitis Causing Bacteria. PLoS One 10(9):e0138084. PMID: 26359669

b. **Erickson DL**, Lew CS, Kartchner B, Porter NT, McDaniel SW, Jones NM, Mason S, Wu E, Wilson E. 2016. Lipopolysaccharide Biosynthesis Genes of *Yersinia pseudotuberculosis* Promote Resistance to Antimicrobial Chemokines. PLoS One 11(6):e0157092. PubMed PMID: 27275606

c. Hoffman JM, Sullivan S, Wu E, Wilson E, **Erickson DL**. 2017. Differential impact of lipopolysaccharide defects caused by loss of RfaH in *Yersinia pseudotuberculosis* and *Yersinia pestis*. Sci Rep. 7(1):10915. PubMed PMID: 28883503

5. More recently, we have made an effort to characterize the distinguishing features of mastitis-associated *E. coli*. For the first time, we demonstrated a role for an individual gene common in these strains (the ferric dicitrate recaptor) as necessary for fitness in lactating mammary glands. This work also identified a Group 3 capsule present in a virulent mastitis strain that we have now found is regulated by zinc and by bile salts. These findings have implications for bacterial colonization of the digestive tract as well as other extraintestinal tissues. In each of these reports I oversaw the work and was the corresponding author.

a. Olson MA, Siebach TW, Griffitts JS, Wilson E, **Erickson DL.** 2018. Genome-wide identification of fitness factors in mastitis-associated *Escherichia coli*. Appl Environ Microbiol 84(2):e02190-17 PubMed PMID: 29101196

b. Olson M.A., Grimsrud A., Richards A.C., Mulvey M.A., Wilson E., Erickson D.L. A link between zinc uptake, bile salts, and a capsule required for virulence of a mastitis-associated extraintestinal pathogenic *Escherichia coli* strain. bioRxiv **[Preprint]** 2020.06.25

Complete List of Published Work in MyBibliography:
http://www.ncbi.nlm.nih.gov/sites/myncbi/1PUy03pFlrfki/bibliography/48935137/public/?sort=date&direction=ascending.

D. Research Support

Current Support

2020-2022 Role: Principal Investigator
Marcus E. Jensen Poultry Disease Research Endowment
The goal of this project is to identify fitness factors of avian pathogenic *E. coli* and develop new strategies to limit their growth in agricultural settings.

Relevant past funding

2012-2016 Role: Co-Principal Investigator with Dr. Eric Wilson
NIH# 1R15AI1958-01 Title: Identification of Bacterial Resistance Mechanisms to Antimicrobial Chemokines
The goal of his study was to understand genes of *Yersinia pseudotuberculosis* in mediating bacterial evasion of host antimicrobial peptides including chemokines CCL28 and CCL25.