BIOGRAPHICAL SKETCH

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NAME: Renee Ormsby

eRA COMMONS USER NAME (credential, e.g., agency login):

POSITION TITLE: Postdoctoral Research Fellow

EDUCATION/TRAINING *(Begin with baccalaureate or other initial professional education, such as nursing, include postdoctoral training and residency training if applicable. Add/delete rows as necessary.)*

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	Completion Date MM/YYYY	FIELD OF STUDY
Flinders University, Bedford Park, SA, Australia	B.S.	04/2009	Chemistry
University of Adelaide, Adelaide, SA, Australia	Ph.D.	02/2019	Orthopaedic Research

A. Personal Statement

I have extensive experience in applying bone histology and quantitative histomorphometry to understanding pathologic processes in bone. Osteoclasts, as well as osteocytes, are key cells that actively resorb the bone and can contribute to the loss of bone that occurs in osteoporosis. Understanding and identifying the mechanisms behind bone resorptive pathways is important in order to find key therapeutic targets that prevent bone loss, and potentially stimulate bone formation.

B. Positions and Honors

Positions and Employment

2008-2009 Technical Officer. Department of Orthopaedics and Trauma, Royal Adelaide Hospital, Adelaide, South Australia.
2009-2015 Research Assistant. Biomedical Orthopaedic Research Group, Centre for Orthopaedic and Trauma Research, University of Adelaide.
2015-2019 Laboratory Manager/Research Assistant. Biomedical Orthopaedic Research Group, Centre for Orthopaedic and Trauma Research, University of Adelaide.
2020- Post-doctoral Fellow. Department of Orthopedic Research, Brigham and Womens Hospital, Boston, Massachussetts.

Professional Memberships

2010- Member, Australian New Zealand Bone Mineral Society
2014- Member, Australian New Zealand Orthopaedic Research Society
2015- Member, Australian Society for Medical Research
2016- Member, Orthopaedic Research Society

C. Contributions to Science

1. Osteocytes in peri-prosthetic osteolysis

We identified a key role for osteocytes in the response to prosthetic derived wear particles, which stimulate foreign body responses, in pathologic bone loss. We showed that osteocytes are capable of stimulating both osteoclastic resorptive pathways as well as inducing perilacunar remodelling in response to wear particles by upregulating key bone degrading enzymes, classically associated with osteoclasts. This includes the production of carbonic anhydrase 2 which acidifies the bone environment. This work revealed a previously unknown role for osteocytes in peri-prosthetic osteolysis and provides new insight into the mechanisms that cause bone loss and prostheses failure.

1. **Ormsby RT**, Solomon LB, Stamenkov R, Findlay DM, Atkins GJ. Evidence for Gender-Specific Bone Loss Mechanisms in Periprosthetic Osteolysis. J Clin Med. 2019 Dec 24;9(1). doi: 10.3390/jcm9010053. PubMed PMID: 31878362; PubMed Central PMCID: PMC7019811.
2. **Ormsby RT**, Solomon LB, Yang D, Crotti TN, Haynes DR, Findlay DM, Atkins GJ. Osteocytes respond to particles of clinically-relevant conventional and cross-linked polyethylene and metal alloys by up-regulation of resorptive and inflammatory pathways. Acta Biomater. 2019 Mar 15;87:296-306. doi: 10.1016/j.actbio.2019.01.047. Epub 2019 Jan 25. PubMed PMID: 30690207.
3. **Ormsby RT**, Cantley M, Kogawa M, Solomon LB, Haynes DR, Findlay DM, Atkins GJ. Evidence that osteocyte perilacunar remodelling contributes to polyethylene wear particle induced osteolysis. Acta Biomater. 2016 Mar;33:242-51. doi: 10.1016/j.actbio.2016.01.016. Epub 2016 Jan 18. PubMed PMID: 26796208.
4. Kogawa M, Wijenayaka AR, **Ormsby RT**, Thomas GP, Anderson PH, Bonewald LF, Findlay DM, Atkins GJ. Sclerostin regulates release of bone mineral by osteocytes by induction of carbonic anhydrase 2. J Bone Miner Res. 2013 Dec;28(12):2436-48. doi: 10.1002/jbmr.2003. PubMed PMID: 23737439.

2. Autocrine Regulation of Bone

We have identified key relationships between the expression of vitamin D responsive genes in human bone and key regulators of bone resorption and formation. We have shown that CYP27b1, the enzyme that converts Vitamin D into its active form (1,25-dihydroxyvitamin D), is coordinately expressed with markers of bone mineralization, DMP1 and OPN, and with markers of osteoclast activity including TRAP and NFATC1. We demonstrated that CYP27b1 plays an intrinsic role in osteoclast differentiation, as specific knockdown of CYP27B1 *in vitro* resulted in decreased osteoclast differentiation as well as decreased NFATC1 expression. This work described an important role for vitamin D in regulating bone resorption, as well as promoting the coupling of bone resorption and formation.

Kogawa M, Findlay DM, Anderson PH, **Ormsby R**, Vincent C, Morris HA, Atkins GJ. Osteoclastic metabolism of 25(OH)-vitamin D3: a potential mechanism for optimization of bone resorption. Endocrinology. 2010 Oct;151(10):4613-25. doi: 10.1210/en.2010-0334. Epub 2010 Aug 25. PubMed PMID: 20739402.

Ormsby RT, Findlay DM, Kogawa M, Anderson PH, Morris HA, Atkins GJ. Analysis of vitamin D metabolism gene expression in human bone: evidence for autocrine control of bone remodelling. J Steroid Biochem Mol Biol. 2014 Oct;144 Pt A:110-3. doi: 10.1016/j.jsbmb.2013.09.016. Epub 2013 Oct 10. Review. PubMed PMID: 24120913.

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