BIOGRAPHICAL SKETCH

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NAME	POSITION TITLE
Peter Cawood Butler, MD	Professor of Medicine
eRA COMMONS USER NAME (credential, e.g., agency login) BUTLER	

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

INSTITUTION AND LOCATION	DEGREE *(if applicable)*	MM/YY	FIELD OF STUDY
University of Birmingham, UK	MD	08/75–07/80	Medicine
University of Birmingham, UK		08/80–07/81	Internship
University of Edinburgh/Birmingham, UK	MRCP	08/81–07/87	Resident
Mayo Medical College, Rochester, Minnesota, USA		08/87–07/90	Postdoctoral Researcher

A. Personal Statement

Peter Butler is a clinician investigator with a primary interest in mechanisms of islet dysfunction and beta cell loss in both type 1 and 2 diabetes. He also has a personal interest in mentoring and training students and post doctoral fellows in islet research.

B. Positions and Honors

Positions and Employment

1990-1991 Assistant Professor Medicine, East Carolina University, Greenville, NC
1991-1996 Associate Professor Medicine, Mayo Clinic, Rochester, MN
1991-1996 Associate Director, Clinical Research Center, Mayo Clinic, Rochester, MN
1996-1999 Chair, Diabetic Medicine, Professor of Medicine, University of Edinburgh, Edinburgh, Scotland, UK
1996-1999 Director, Wellcome Trust Clinical Research Center, University of Edinburgh, Edinburgh, Scotland, UK
1999-2004 Chief, Division of Endocrinology & Diabetes, Professor of Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA
2004-present Professor of Medicine, Chief, Division of Endocrinology, Diabetes & Hypertension, David Geffen School of Medicine, University of California, Los Angeles

Honors

1984 RD Lawrence British Diabetes Association Scholarship
1990 Henry Christian Award for top scoring abstract submitted to National American Federation for Clinical Research
1996 Elected to FRCP (Edinburgh)
2000 Best Clinical paper published in JCEM, Endocrine Society

C. Selected Peer-reviewed Publications (Selected from 121 peer-reviewed publications)

1. Janson J, Soeller WC, Roche PC, Nelson RT, Torchia AJ, Kruetter DK, **Butler PC**. Spontaneous diabetes mellitus in transgenic mice expressing human islet amyloid polypeptide. Proc. Natl. Acad. Sci. USA, 93: 7283-7288, 1996. PMCID: PMC38975
2. Janson J, Ashley RH, Harrison D, McIntyre S, **Butler PC**. The mechanism of islet amyloid polypeptide toxicity is membrane disruption by intermediate-sized toxic amyloid particles. Diabetes 48: 491-498, 1999.
3. Laedtke T, Kjems L, Pørksen N, Schmitz, O, Veldhuis JD, Kao PC, **Butler PC**. Overnight inhibition of insulin secretion restores pulsatility and the proinsulin/insulin ratio in Type 2 diabetes. Am. J. Physiol. Endocrinol. Metab. 279:E 520-528, 2000.
4. Butler AE, Janson J, Bonner-Weir S, Ritzel R, Rizza RA, **Butler PC**. β-cell deficit and increased apoptosis in humans with type-2 diabetes. Diabetes 52 (1):102-110, 2003.

5. Ritzel RA, **Butler, PC**. Replication increases beta cell vulnerability to human islet amyloid polypeptide induced apoptosis. Diabetes. 52:1701-1708, 2003.
6. Butler AE, Jang J, Gurlo T, Carty MD, Soeller WS, **Butler PC**. Diabetes due to a progessive defect in beta cell mass in rats transgenic for human islet amyloid polypeptide (HIP rat). A new model for type-2 diabetes. Diabetes 53:1509-1516, 2004.
7. Meier JJ, Kayed R, Lin CY, Gurlo T, Haataja L, Jayasinghe S, Langen R, Glabe CG, **Butler PC**. Inhibition of human IAPP fibril formation does not prevent beta-cell death: Evidence for distinct actions of oligomers and fibrils of human IAPP. Am J Physiol Endocrinol Metab 291: E1317-24, 2006.
8. Ritzel RA, Meier JJ, Lin CY, Veldhuis JD, **Butler PC**. Human islet amyloid polypeptide oligomers disrupts cell coupling, induces apoptosis, and impair insulin secretion in isolated human islets. Diabetes 56(1):65-71, 2007.
9. Lin CY, Gurlo T, Kayed R, Butler AE, Haataja L, Glabe CG, **Butler PC**. Toxic Human Islet Amyloid Polypeptide (h-IAPP) Oligomers are Intracellular, and Vaccination to Induce Anti-Toxic Oligomer Antibodies Does not Prevent h-IAPP Induced ß-Cell Apoptosis in h-IAPP Transgenic Mice. Diabetes 56(5):1324-32, 2007.
10. Huang CJ, Lin CY, Haataja L, Gurlo T, Butler AE, Rizza RA, **Butler PC**. High expression rates of human islet amyloid polypeptide induce endoplasmic reticulum stress-mediated beta cell apoptosis, a characteristic of humans with Type 2 but not Type 1 Diabetes. Diabetes, 56(8):2016-27, 2007.
11. Huang CJ, Haataja L, Galasso R, Gurlo T, Butler AE, Wu X, Soeller W, **Butler PC**. Induction of endoplasmic reticulum stress induced beta cell apoptosis and accumulation of polyubiquinated proteins by human islet amyloid polypeptide. Am J Physiol Endocrinol Metab. 293(6):E1656-62, 2007.
12. Matveyenko AV, Gurlo T, Daval M, Butler AE, **Butler PC**. Successful versus failed Adaptation to High Fat Diet induced Insulin Resistance; the role of IAPP induced Beta Cell Endoplasmic Reticulum Stress. Diabetes. 2009 Apr;58(4):906-16. Epub 2009 Jan 16. PMCID: PMC2661593
13. Matveyenko AV, Dry S, Cox HI, Moshtaghian A, Gurlo T, Galasso R, Butler AE, **Butler PC**. Beneficial Endocrine but adverse Exocrine effects of Sitagliptin in the HIP rat model of Type 2 Diabetes, interactions with Metformin. Diabetes. 2009 Jul;58(7):1604-15. PMCID: PMC2699878
14. Huang CJ, Gurlo T, Haataja L, Costes S, Daval M, Ryazantsev S, Wu X, Butler AE, **Butler PC**. Calcium-activated calpain-2 is a mediator of beta cell dysfunction and apoptosis in type 2 diabetes. Journal of Biological Chemistry. 2010 Jan 1;285(1):339-48. Epub 2009 Oct 27. PMCID: PMC2804181
15. Gurlo T, Ryazantsev S, Huang CJ, Yeh MW, Reber HA, Hines OJ, O'Brien TD, Glabe CG**, Butler PC**. Evidence for Proteotoxicity in {beta} Cells in Type 2 Diabetes, Toxic Islet Amyloid Polypeptide Oligomers form Intracellularly in the Secretory Pathway. Am J Pathol. 2010 Feb;176(2):861-9. Epub 2009 Dec 30. PMCID: PMC2808091

D. Research Support

Ongoing Research Support

R01DK059579 Butler (PI) NIH/NIDDK
Role of IAPP in Islet Dysfunction in Diabetes 6/1/01 – 3/31/14
The major goals of this project are to establish the mechanisms of Islet Amyloid Polypeptide (IAPP) induced beta cell apoptosis and dysfunction using transgenic rodent models.
Role: PI

R01DK077967 Butler (PI) NIH/NIDDK 1/15/08 – 1/31/13
Capacity and Mechanisms of Beta Cell Regeneration in Humans
The studies in this grant would address the question, to what extent is beta cell regeneration feasible in humans?
Role: PI

R01DK061539 Butler (PI) NIH/NIDDK 7/1/02 – 3/31/13
Role of Pulsatile Insulin Secretion
The major goals of this project are to establish the role of pulsatile insulin secretion.
Role: PI