Resource Summary Report

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Beta Cell Biology Consortium

RRID:SCR_005136
Type: Tool

Proper Citation

Beta Cell Biology Consortium (RRID:SCR_005136)

Resource Information

URL: http://www.betacell.org/

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Description: THIS RESOURCE IS NO LONGER IN SERVICE, documented May 10, 2017. A pilot effort that has developed a centralized, web-based biospecimen locator that presents biospecimens collected and stored at participating Arizona hospitals and biospecimen banks, which are available for acquisition and use by researchers. Researchers may use this site to browse, search and request biospecimens to use in qualified studies. The development of the ABL was guided by the Arizona Biospecimen Consortium (ABC), a consortium of hospitals and medical centers in the Phoenix area, and is now being piloted by this Consortium under the direction of ABRC. You may browse by type (cells, fluid, molecular, tissue) or disease. Common data elements decided by the ABC Standards Committee, based on data elements on the National Cancer Institute''s (NCI''s) Common Biorepository Model (CBM), are displayed. These describe the minimum set of data elements that the NCI determined were most important for a researcher to see about a biospecimen. The ABL currently does not display information on whether or not clinical data is available to accompany the biospecimens. However, a requester has the ability to solicit clinical data in the request. Once a request is approved, the biospecimen provider will contact the requester to discuss the request (and the requester''s questions) before finalizing the invoice and shipment. The ABL is available to the public to browse. In order to request biospecimens from the ABL, the researcher will be required to submit the requested required information. Upon submission of the information, shipment of the requested biospecimen(s) will be dependent on the scientific and institutional review approval. Account required. Registration is open to everyone., documented on August 1, 2015. Consortium that aims to facilitate interdisciplinary collaborations to advance the understanding of pancreatic islet development and function, with the goal of developing innovative therapies to correct the loss of beta cell mass in diabetes, including cell reprogramming, regeneration and replacement. They are responsible for collaboratively generating the necessary reagents, mouse strains, antibodies,

assays, protocols, technologies and validation assays that are beyond the scope of any single research effort. The scientific goals for the BCBC are to: * Use cues from pancreatic development to directly differentiate pancreatic beta cells and islets from stem / progenitor cells for use in cell-replacement therapies for diabetes, * Determine how to stimulate beta cell regeneration in the adult pancreas as a basis for improving beta cell mass in diabetic patients, * Determine how to reprogram progenitor / adult cells into pancreatic beta-cells both in-vitro and in-vivo as a mean for developing cell-replacement therapies for diabetes, and * Investigate the progression of human type-1 diabetes using patient-derived cells and tissues transplanted in humanized mouse models. Many of the BCBC investigator-initiated projects involve reagent-generating activities that will benefit the larger scientific community. The combination of programs and activities should accelerate the pace of major new discoveries and progress within the field of beta cell biology.

Abbreviations: BCBC

Resource Type: biomaterial supply resource, material resource, cell repository

Keywords: RIN, Resource Information Network, pancreatic islet, mouse, beta cell, pancreas, pancreatic development, embryonic stem cell, cell line, genomics, antibody, adenovirus, functional genomics, mouse embryonic stem cell line, mouse strain, protocol, embryonic stem cell line, data sharing, data set, gene expression, gene, pancreatic islet development, pancreatic islet function, basic science, basic research, cell reprogramming, cell regeneration, cell replacement

Related Condition: Type 1 diabetes, Diabetes

Funding: NIDDK DK-01-014;
NIDDK DK-01-17;
NIDDK DK-01-18;
NIDDK DK-09-011

Availability: THIS RESOURCE IS NO LONGER IN SERVICE

Resource Name: Beta Cell Biology Consortium

Resource ID: SCR_005136

Alternate IDs: nlx_144143

Record Creation Time: 20220129T080228+0000

Record Last Update: 20250508T064936+0000

Ratings and Alerts

No rating or validation information has been found for Beta Cell Biology Consortium .

No alerts have been found for Beta Cell Biology Consortium .

Data and Source Information

Source: SciCrunch Registry

Usage and Citation Metrics

We found 59 mentions in open access literature.

Listed below are recent publications. The full list is available at dkNET.

van Arensbergen J, et al. (2017) A distal intergenic region controls pancreatic endocrine differentiation by acting as a transcriptional enhancer and as a polycomb response element. PloS one, 12(2), e0171508.

Churchill AJ, et al. (2017) Genetic evidence that Nkx2.2 acts primarily downstream of Neurog3 in pancreatic endocrine lineage development. eLife, 6.

Elghazi L, et al. (2017) Role of nutrients and mTOR signaling in the regulation of pancreatic progenitors development. Molecular metabolism, 6(6), 560.

Dai C, et al. (2017) Age-dependent human ? cell proliferation induced by glucagon-like peptide 1 and calcineurin signaling. The Journal of clinical investigation, 127(10), 3835.

Mussar K, et al. (2017) A CCR2+ myeloid cell niche required for pancreatic ? cell growth. JCI insight, 2(15).

Chakravarthy H, et al. (2017) Converting Adult Pancreatic Islet ? Cells into ? Cells by Targeting Both Dnmt1 and Arx. Cell metabolism, 25(3), 622.

Swisa A, et al. (2017) PAX6 maintains ? cell identity by repressing genes of alternative islet cell types. The Journal of clinical investigation, 127(1), 230.

Schmidt SF, et al. (2016) Integrative Genomics Outlines a Biphasic Glucose Response and a ChREBP-ROR? Axis Regulating Proliferation in ? Cells. Cell reports, 16(9), 2359.

Bandrowski A, et al. (2016) The Resource Identification Initiative: A Cultural Shift in Publishing. The Journal of comparative neurology, 524(1), 8.

Bandrowski A, et al. (2016) The Resource Identification Initiative: a cultural shift in publishing. Brain and behavior, 6(1), e00417.

Bandrowski A, et al. (2016) The Resource Identification Initiative: A Cultural Shift in Publishing. Neuroinformatics, 14(2), 169.

Kim YH, et al. (2015) Cell cycle-dependent differentiation dynamics balances growth and endocrine differentiation in the pancreas. PLoS biology, 13(3), e1002111.

Bandrowski A, et al. (2015) The Resource Identification Initiative: A cultural shift in

publishing. F1000Research, 4, 134.

Piran R, et al. (2014) Pharmacological induction of pancreatic islet cell transdifferentiation: relevance to type I diabetes. Cell death & disease, 5(7), e1357.

Metzger DE, et al. (2014) Grg3/TLE3 and Grg1/TLE1 induce monohormonal pancreatic ?-cells while repressing ?-cell functions. Diabetes, 63(5), 1804.

Lenz A, et al. (2014) Redifferentiation of adult human ? cells expanded in vitro by inhibition of the WNT pathway. PloS one, 9(11), e112914.

Brafman DA, et al. (2013) Regulation of endodermal differentiation of human embryonic stem cells through integrin-ECM interactions. Cell death and differentiation, 20(3), 369.

Fradkin JE, et al. (2013) Celebrating 30 years of research accomplishments of the diabetes control and complications trial/epidemiology of diabetes interventions and complications study. Diabetes, 62(12), 3963.

Fradkin JE, et al. (2013) Diabetes research: a perspective from the National Institute of Diabetes and Digestive and Kidney Diseases. Diabetes, 62(2), 320.

Capito C, et al. (2013) Mouse muscle as an ectopic permissive site for human pancreatic development. Diabetes, 62(10), 3479.