

Resource Summary Report

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TIDE BC

RRID:SCR_003924

Type: Tool

Proper Citation

TIDE BC (RRID:SCR_003924)

Resource Information

URL: <http://www.tidebc.org/>

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Description: A collaborative care and research initiative with a focus on prevention and treatment of Intellectual disability (ID) that is due to inborn errors of metabolism (IEM), which can be treated with diet or drugs. Health care policy and institutional culture is still operating under the old premise that all ID is incurable and thus, many children born with treatable ID are at risk of not being treated. To acknowledge the multidisciplinary scope and the ways in which health care professionals and researchers will collaborate, the goals of the TIDE BC project are demonstrated within a framework of 7 Work Packages: * Implementation of a new Protocol for diagnostic evaluation of ID, focusing of treatable conditions; * Development of infrastructure to facilitate implementation, evaluation and sustainability of the Protocol; * Investments into next generation genomic technologies; * Improving evidence of and access to treatments; * Evaluation and health economy; * Knowledge dissemination; * Education and Mentoring. The objectives addressed in all Work Packages reflect a highly integrated cluster combining clinical care, research, evaluation, and knowledge dissemination.

Abbreviations: TIDE

Synonyms: TIDE-BC, Treatable Intellectual Disability Endeavor in B.C.

Resource Type: data or information resource, portal

Keywords: child, prevention, treatment, pediatric, genetic

Related Condition: Intellectual disability, Inborn error of metabolism

Funding: BC Childrens Hospital Foundation

Resource Name: TIDE BC

Resource ID: SCR_003924

Alternate IDs: nlx_158289

Record Creation Time: 20220129T080221+0000

Record Last Update: 20260729T044050+0000

Ratings and Alerts

No rating or validation information has been found for TIDE BC.

No alerts have been found for TIDE BC.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 27 mentions in open access literature.

Listed below are recent publications. The full list is available at [kravitz2](#) .

Carter MT, et al. (2023) Genetic and metabolic investigations for neurodevelopmental disorders: position statement of the Canadian College of Medical Geneticists (CCMG). Journal of medical genetics, 60(6), 523.

Li L, et al. (2023) Bioinformatics construction and experimental validation of a cuproptosis-related lncRNA prognostic model in lung adenocarcinoma for immunotherapy response prediction. Scientific reports, 13(1), 2455.

Luff DH, et al. (2021) PI3K γ Forms Distinct Multiprotein Complexes at the TCR Signalingosome in Naïve and Differentiated CD4⁺ T Cells. Frontiers in immunology, 12, 631271.

Boycott KM, et al. (2020) The Canadian Rare Diseases Models and Mechanisms (RDMM) Network: Connecting Understudied Genes to Model Organisms. American journal of human genetics, 106(2), 143.

Tarailo-Graovac M, et al. (2019) De novo pathogenic DNMT1L variant in a patient diagnosed with atypical hereditary sensory and autonomic neuropathy. Molecular genetics & genomic medicine, 7(10), e00961.

Lee JJY, et al. (2019) Development and user evaluation of a rare disease gene prioritization workflow based on cognitive ergonomics. Journal of the American Medical Informatics Association : JAMIA, 26(2), 124.

O'Byrne JJ, et al. (2018) The genotypic and phenotypic spectrum of MTO1 deficiency. Molecular genetics and metabolism, 123(1), 28.

- Lee JJY, et al. (2018) Text-based phenotypic profiles incorporating biochemical phenotypes of inborn errors of metabolism improve phenomics-based diagnosis. *Journal of inherited metabolic disease*, 41(3), 555.
- Shyr C, et al. (2017) Correction to: FLAGS, frequently mutated genes in public exomes. *BMC medical genomics*, 10(1), 69.
- Tarailo-Graovac M, et al. (2017) Assessment of the ExAC data set for the presence of individuals with pathogenic genotypes implicated in severe Mendelian pediatric disorders. *Genetics in medicine : official journal of the American College of Medical Genetics*, 19(12), 1300.
- Tarailo-Graovac M, et al. (2017) Identification of a large intronic transposal insertion in SLC17A5 causing sialic acid storage disease. *Orphanet journal of rare diseases*, 12(1), 28.
- Sinclair GB, et al. (2016) A three-tier algorithm for guanidinoacetate methyltransferase (GAMT) deficiency newborn screening. *Molecular genetics and metabolism*, 118(3), 173.
- Santra S, et al. (2016) Cytosolic phosphoenolpyruvate carboxykinase deficiency presenting with acute liver failure following gastroenteritis. *Molecular genetics and metabolism*, 118(1), 21.
- Horvath GA, et al. (2016) Secondary neurotransmitter deficiencies in epilepsy caused by voltage-gated sodium channelopathies: A potential treatment target? *Molecular genetics and metabolism*, 117(1), 42.
- Sass JO, et al. (2016) Unravelling 5-oxoprolinuria (pyroglutamic aciduria) due to bi-allelic OPLAH mutations: 20 new mutations in 14 families. *Molecular genetics and metabolism*, 119(1-2), 44.
- Zaharieva IT, et al. (2016) Loss-of-function mutations in SCN4A cause severe foetal hypokinesia or 'classical' congenital myopathy. *Brain : a journal of neurology*, 139(Pt 3), 674.
- Shyr C, et al. (2016) Dynamic software design for clinical exome and genome analyses: insights from bioinformaticians, clinical geneticists, and genetic counselors. *Journal of the American Medical Informatics Association : JAMIA*, 23(2), 257.
- Lee JJY, et al. (2016) Further Validation of the SIGMAR1 c.151+1G>T Mutation as Cause of Distal Hereditary Motor Neuropathy. *Child neurology open*, 3, 2329048X16669912.
- van Karnebeek CD, et al. (2015) Health economic evaluation of plasma oxysterol screening in the diagnosis of Niemann-Pick Type C disease among intellectually disabled using discrete event simulation. *Molecular genetics and metabolism*, 114(2), 226.
- Sirrs S, et al. (2015) Defects in fatty acid amide hydrolase 2 in a male with neurologic and psychiatric symptoms. *Orphanet journal of rare diseases*, 10, 38.