

Resource Summary Report

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Linkage Disequilibrium Analyses for Quantitative and Discrete Traits

RRID:SCR_013365

Type: Tool

Proper Citation

Linkage Disequilibrium Analyses for Quantitative and Discrete Traits (RRID:SCR_013365)

Resource Information

URL: <http://www.sph.umich.edu/csg/abecasis/QTDT/>

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Description: How is association mapping going to help me find genes? During the past decade, the genes for a large number of rare mendelian traits have been identified. However, traditional linkage analyses lack power and precision when applied to complex disease. Association mapping, which compares the effects of different chromosomal variants, may be more successful at identifying genes of small effect. How does QTDT help association mapping? Association mapping can produce misleading results when the study population is not homogeneous, but includes individuals with different genetic backgrounds. Family based association tests, commonly referred to as TDTs (Transmission Disequilibrium Tests), do not produce misleading results in these circumstances. QTDT can use all the information in a pedigree to construct powerful tests of association that are robust in the presence of stratification. What does the Q stand for? Q stands for Quantitative. Quantitative traits provide effective descriptions of many complex diseases, including asthma. For many of these conditions, all or nothing definitions of disease are arbitrary and unsatisfactory. QTDT incorporates variance components methodology in the analysis of family data and includes exact estimation of p-values for analysis of small samples and non-normal data. The QTDT abbreviation (for Quantitative Transmission Disequilibrium Tests) was first used by David Allison in his 1997 paper. This research was supported in part by the intramural program of the National Eye Institute and by National Institutes of Health Grants EY016862, EY007758, EY09859, EY012118, P30-EY014801, EY-014458, EY014467, HL084729, and HG002651, by the Foundation Fighting Blindness, the Macula Vision Research Foundation, the American Health Assistance Foundation, Research to Prevent Blindness, the Pew Charitable Trusts, the Mayo Clinic Foundation, the Casey Macular Degeneration Center

Fund, the Marion W. and Edward F. Knight AMD Fund, the Harold and Pauline Price Foundation, National Genotyping Centre of Spain, and the Elmer and Sylvia Sramek Foundation. The Center for Inherited Disease Research, fully funded through a federal contract (HHSN268200782096C) from National Institutes of Health to

Synonyms: QTDT

Resource Type: database, software application, software resource, data or information resource

Funding:

Resource Name: Linkage Disequilibrium Analyses for Quantitative and Discrete Traits

Resource ID: SCR_013365

Alternate IDs: nif-0000-31385

Record Creation Time: 20220129T080315+0000

Record Last Update: 20250428T053750+0000

Ratings and Alerts

No rating or validation information has been found for Linkage Disequilibrium Analyses for Quantitative and Discrete Traits.

No alerts have been found for Linkage Disequilibrium Analyses for Quantitative and Discrete Traits.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 6 mentions in open access literature.

Listed below are recent publications. The full list is available at [FDI Lab - SciCrunch.org](#).

Bohossian N, et al. (2014) Single-marker and multi-marker mixed models for polygenic score analysis in family-based data. BMC proceedings, 8(Suppl 1), S63.

Knez J, et al. (2014) Left ventricular diastolic function associated with common genetic variation in ATP12A in a general population. BMC medical genetics, 15, 121.

Xiong DH, et al. (2009) Genome-wide association and follow-up replication studies identified

ADAMTS18 and TGFBR3 as bone mass candidate genes in different ethnic groups. American journal of human genetics, 84(3), 388.

Huang RS, et al. (2007) Identification of genetic variants contributing to cisplatin-induced cytotoxicity by use of a genomewide approach. American journal of human genetics, 81(3), 427.

Zhang F, et al. (2006) HDC gene polymorphisms are associated with age at natural menopause in Caucasian women. Biochemical and biophysical research communications, 348(4), 1378.

Ferreira R, et al. (2005) Heritable factors shape natural human IgM reactivity to Ro60/SS-A and may predispose for SLE-associated IgG anti-Ro and anti-La autoantibody production. Journal of autoimmunity, 25(2), 155.