

Resource Summary Report

Generated by NIF on Sep 16, 2026

FRAXA Research Foundation

RRID:SCR_003125

Type: Tool

Proper Citation

FRAXA Research Foundation (RRID:SCR_003125)

Resource Information

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

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Description: FRAXA's mission is to accelerate progress toward effective treatments and ultimately a cure for Fragile X, by directly funding the most promising research. FRAXA also supports families affected by Fragile X and raises awareness of this important but relatively unknown disease. FRAXA was founded in 1994 by three parents of children with Fragile X, Katie Clapp, Michael Tranfaglia MD, and Kathy May, to support scientific research aimed at finding a treatment and a cure for Fragile X. Fragile X research is drastically underfunded, considering its high prevalence, prospects for a cure, and the promise that this research holds for advancing understanding of other disorders like autism, Alzheimer's disease, and X-linked mental retardation. FRAXA funds grants and fellowships at universities all over the world. We have funded more than \$17 million dollars in top-notch science. FRAXA's management expenses have always been just 4% or less of income, as we have just one full-time staff, three part time staff, and hundreds of volunteer parents. Since FRAXA was founded, the Fragile X field has grown tremendously, due in large part to our grass-roots efforts. You can help us accomplish much more. FRAXA is a 501c3 tax-exempt organization; Tax ID 04-3222167

Abbreviations: FRAXA

Synonyms: FRAXA

Resource Type: data or information resource, disease-related portal, funding resource, portal, topical portal

Keywords: research, treatment, cure

Related Condition: Fragile X syndrome

Availability: Free, Freely available

Resource Name: FRAXA Research Foundation

Resource ID: SCR_003125

Alternate IDs: nif-0000-30561

Record Creation Time: 20220129T080217+0000

Record Last Update: 20260912T200009+0000

Ratings and Alerts

No rating or validation information has been found for FRAXA Research Foundation.

No alerts have been found for FRAXA Research Foundation.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 12 mentions in open access literature.

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

Kumar V, et al. (2024) mGluR7 allosteric modulator AMN082 corrects protein synthesis and pathological phenotypes in FXS. EMBO molecular medicine, 16(3), 506.

Jung S, et al. (2023) FMRP deficiency leads to multifactorial dysregulation of splicing and mislocalization of MBNL1 to the cytoplasm. PLoS biology, 21(12), e3002417.

Saraf TS, et al. (2021) Evaluation of lorcaserin as an anticonvulsant in juvenile Fmr1 knockout mice. Epilepsy research, 175, 106677.

Lee AW, et al. (2018) Clinical Development of Targeted Fragile X Syndrome Treatments: An Industry Perspective. Brain sciences, 8(12).

Enriquez-Barreto L, et al. (2016) The PI3K signaling pathway as a pharmacological target in Autism related disorders and Schizophrenia. Molecular and cellular therapies, 4, 2.

Schaefer TL, et al. (2015) Emerging pharmacologic treatment options for fragile X syndrome. The application of clinical genetics, 8, 75.

Westmark CJ, et al. (2014) Soy infant formula and seizures in children with autism: a retrospective study. PloS one, 9(3), e80488.

Joyce C, et al. (2014) Transforming our approach to translational neuroscience: the role and impact of charitable nonprofits in research. Neuron, 84(3), 526.

Lucá R, et al. (2013) The fragile X protein binds mRNAs involved in cancer progression and modulates metastasis formation. *EMBO molecular medicine*, 5(10), 1523.

Kroon T, et al. (2013) Investigating mechanisms underlying neurodevelopmental phenotypes of autistic and intellectual disability disorders: a perspective. *Frontiers in systems neuroscience*, 7, 75.

Cruz-Martín A, et al. (2012) Glutamate induces the elongation of early dendritic protrusions via mGluRs in wild type mice, but not in fragile X mice. *PloS one*, 7(2), e32446.

Westmark CJ, et al. (2011) Reversal of fragile X phenotypes by manipulation of A?PP/A? levels in Fmr1KO mice. *PloS one*, 6(10), e26549.