Twin Table Tennis Champions

Identical 14-year-old twins, Brandon and Brad Belle of Brooklyn, New York, have distinguished themselves as champion table tennis players in a very short time. The twins moved to Brooklyn from Guyana in 2007 and began playing table tennis shortly thereafter. Their coach recalls that they went from being nonplayers to tournament competitors in only 3 months. They began to win tournaments four months after that. The twins are physically matched, except for their hand preference — Brandon is left-handed and Brad is right-handed. Their next goal is to participate in the Olympic trials in January 2012.

References

Buckley, C. (2009, 20 April). Culinary twins, rarely separated since birth. *New York Times*.

Casci, T. (2002). A twin approach to disease. *Nature Reviews (Genetics), 3*, 732.

Choi, Y., Bishai, D., & Minokvitz, C. (2009). Multiple births are a risk factor for postpartum maternal depressive symptoms. *Pediatrics, 123*, 1208–1216.

Haque, F. N., Gottesman, I. I., & Wong, A. H. C. (2009). Not really identical: Epigenetic differences in monozygotic twins and implications for twin studies in psychiatry. *American Journal of Medical Genetics Part C (Seminars in Medical Genetics), 151C*, 136–141.

Holden, C. (2009). 'Banking on Twins.' *Science, 324,* 571.

Kimani, J. W., Shi, M., Daack-Hirsch, S., Christenen, K., Moretti-Ferreira, D., Marazita, M. L., Field, L. L., Canady, J. W., Murray, J. C. (2007). X-Chromosome inactivation patterns in monosygotic twins and sib pairs discordant for nonsyndromic cleft lip and/or palate. *American Journal of Medical Genetics Part A, 143A*, 3267–3272.

Kondo, S., Schutte, B. C., Richardson, R. J., Bjork, B. C., Knight, A. S., & Watanabe, Y. (2002). Mutations in IRF6 cause Van der Woude and popliteal pteryglum syndromes. *Nature Genetics, 32,* 285–289.

Mansilla, M. A., Kimani, J., Mitchell, L. E., Christensen, K., Boomsma, D. I., Daack-Hirsh,S., Nepomucena, B., Wyszynski, D. F., Felix, T. M., Martin, N. G., & Murray, J. C. (2005). Discordant MZ twins with cleft lip and palate: A model for identifying genes in complex traits. *Twin Research and Human Genetics, 8,* 39–46.

NYDailyNews.com (2009, 17 April). 'Meet Brooklyn's table tennis terrors.' Available at www.the2012londonolympics.com/forum/showthread.php?t=14069.

Pharoah, P. O. D., Glinianaia, S. V., & Rankin, J. (2009). Congenital anomalies in multiple births after early loss of a conceptus. *Human Reproduction, 24*, 726–731.

Seager, J. (2007a). 'Breastfeeding miracle.' Mothering, 142, 66–69. See also http://www.mothering.com/articles/new_baby/breastfeeding/breastfeeding-miracle.html

Seagher, J. (2007b). Beautiful chaos. *Canadian Living*, November, 97–102.

Vilska, S., Unkila-Kailo, L., Punamaki, R.-L., Poikkeus, P., Repokari, L., Sinkkonen, J., Tiitinen, A., & Tulppala, M. (2009). Mental health of mothers and fathers of twins conceived via assisted reproduction treatment: A 1-year prospective study. *Human Reproduction, 24*, 367–377. Heterotaxy syndrome with mirror-image anomalies in identical twins. *Congenital Heart Disease, 4*, 50–53.

Additional Abstract from the Genes for Health Meeting, Incorporating the Inaugural GRaPH-Int Conference and the 33rd HGSA Annual Scientific Meeting

Fremantle, Australia, May 3–6, 2009

HIGH THROUGHPUT SCREENING FOR THE PHARMACOLOGICAL THERAPY OF FRIEDREICH ATAXIA [192]

L. Li[1], S. van den Hengel[2], J. Cook[1], P.A. Ioannou[2,3], M. B. Delatycki[1,3], and J. P. Sarsero[1,2,3]

[1] *Bruce Lefroy Centre for Genetic Health Research, Murdoch Children's Research Institute, Royal Children's Hospital, Victoria, Australia*

[2] *Cell and Gene Therapy, Murdoch Children's Research Institute, Royal Children's Hospital, Victoria, Australia*

[3] *Department of Paediatrics, University of Melbourne, Murdoch Children's Research Institute, Royal Children's Hospital, Victoria, Australia*

Friedreich ataxia (FRDA) is characterized by neurodegeneration and cardiomyopathy. The presence of a GAA trinucleotide repeat expansion in the first intron of the FXN gene results in the inhibition of gene expression and an insufficiency of the mitochondrial protein, frataxin. There is a correlation between expansion length, the amount of residual frataxin and the severity of disease. As the coding sequence is unaltered, pharmacological upregulation of FXN expression may restore frataxin to therapeutic levels in patients. To facilitate screening of compounds that modulate FXN expression in a physiologically relevant manner, we have established a genomic reporter assay consisting of stable HeLa cells containing an FXN-EGFP fusion construct (in-frame fusion of the EGFP gene with the entire normal human genomic FXN locus on a BAC clone). The cell line was used to establish a fluorometric cellular assay for use in high throughput screening (HTS) procedures. A small chemical library of FDA-approved compounds and natural extracts has been screened and analysed. Compound hits identified by HTS have been further evaluated by flow cytometry in the cellular genomic reporter assay. The effects on FXN mRNA and frataxin protein levels have been measured in cell lines derived from individuals with FRDA. Any compound that specifically increases frataxin levels by several-fold in FRDA patients could serve as a potential pharmacological therapy for Friedreich ataxia.

https://doi.org/10.1375/twin.12.4.406 Published online by Cambridge University Press