Data in brief 29 (2020) 105165

Contents lists available at ScienceDirect

Data in brief

journal homepage: www.elsevier.com/locate/dib



Data Article

Data on genetic linkage of oxidative stress with cardiometabolic traits in an intercross derived from hyperlipidemic mouse strains



Daniela T. Fuller ^a, Andrew T. Grainger ^b, Ani Manichaikul ^{b, c}, Weibin Shi ^{a, b, *}

^a Department of Radiology & Medical Imaging, University of Virginia, Charlottesville, VA, USA

^b Biochemistry & Molecular Genetics, University of Virginia, Charlottesville, VA, USA

^c Center for Public Health Genomics, University of Virginia, Charlottesville, VA, USA

ARTICLE INFO

Article history: Received 18 December 2019 Received in revised form 8 January 2020 Accepted 14 January 2020 Available online 23 January 2020

Keywords: Genetic linkage Oxidative stress Cardiometabolic trait Atherosclerosis Mouse

ABSTRACT

The data presented here are related to the research article, entitled Genetic linkage of oxidative stress with cardiometabolic traits in an intercross derived from hyperlipidemic mouse strains, published in Atherosclerosis 2019 Dec 3;293:1–10 (D. Fuller, A.T. Grainger, A. Manichaikul, W. Shi). The supporting materials include original genotypic and phenotypic data obtained from 266 female F2 mice derived from an intercross between C57BL/6 (B6) and BALB/cJ (BALB) Apoe^{-/-} mice. F2 mice were fed 12 weeks of Western diet, starting at 6 weeks of age. Plasma levels of HDL, LDL cholesterol, triglycerides, glucose and malondialdehyde (MDA) and atherosclerosis in the aortic root and the left carotid artery were measured. 127 microsatellite markers across the entire genome were genotyped. The data is provided in the format ready for QTL analysis with J/qtl and MapManager QTX.

© 2020 The Author(s). Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http:// creativecommons.org/licenses/by-nc-nd/4.0/).

DOI of original article: https://doi.org/10.1016/j.atherosclerosis.2019.11.034.

* Corresponding author. University of Virginia Charlottesville, Virginia, 22908 USA. *E-mail address:* ws4v@virginia.edu (W. Shi).

https://doi.org/10.1016/j.dib.2020.105165

2352-3409/© 2020 The Author(s). Published by Elsevier Inc. This is an open access article under the CC BY-NC-ND license (http:// creativecommons.org/licenses/by-nc-nd/4.0/).

Specifications Table

Subject	Biochemistry, Genetics and Molecular Biology
Specific subject area	QTL analysis of oxidative stress
Type of data	Tables
How data were acquired	Plasma levels of malondialdehyde (MDA), total and HDL cholesterol, triglyceride, and glucose levels were measured with assay kits.
	Atherosclerotic lesion sizes in the aortic root and the left carotid artery were quantified with AxioVision version 4.8 software on oil red O stained sections.
	127 microsatellite markers across the entire genome were typed by PCR.
	J/qtl and Map Manager QTX software was used to find genetic loci for the traits.
Data format	Raw
	Analysed
Parameters for data collection	Female F2 mice were started with a western diet at 6 weeks of age and remained on the diet for 12 weeks
Description of data collection	a control 12 models and the second s
	Fasting blood was collected once before and once after 12 weeks of Western diet. The heart and adjacent aorta and the left carotid artery were harvested and processed for quantification of atherosclerosis.
Data source location	University of Virginia, Charlottesville, Virginia, USA
Data accessibility	With the article.
Related research article	Fuller DT, Grainger AT, Manichaikul A, Shi W. Genetic linkage of oxidative stress with cardiometabolic traits in an intercross derived from hyperlipidemic mouse strains. Atherosclerosis. 2019 Dec 3;293:1–10. https://doi.org/10.1016/j.atherosclerosis.2019. 11.034.

Value of the Data

• The raw data provided allows other researchers to make comparisons with their own data.

The data allows other researchers to conduct combined cross analyses of genetic loci for cadiometabolic traits in mice.
Plasma levels of total, HDL, non-HDL cholesterol, free cholesterol, and glucose in F2 mice fed a chow diet are also included.

These data can be used by others to conduct combined cross analysis of QTLs for these traits and power calculation to estimate the number of animals needed for similar experiments.

1. Data description

The Supplementary data provided here includes both raw data and analysed data used in the Atherosclerosis article [1]. The raw data was obtained from 266 female F2 mice derived from an intercross between C57BL/6 (B6) and BALB/cJ (BALB) Apoe^{-/-} mice as reported [2] and was formatted for analyses using J/qtl and Map Manager QTX.

2. Experimental design, materials, and methods

2.1. Mice

266 female F2 mice were generated from an intercross between B6-Apoe^{-/-} and BALB-Apoe^{-/-} mice as reported [2]. Mice were weaned onto a chow diet at 3 weeks of age and started on a Western diet at 6 weeks of age. After 12 weeks of Western diet, mice were euthanized. Fasting blood was collected under isoflurane anaesthesia, once before and once after 12 weeks of Western diet.

2.2. Phenotypic analyses

Plasma MDA was measured with a Cayman Thiobarbituric Acid Reactive Substances (TBARS) kit (Cat. # 10009055). Plasma glucose was measured with a Sigma assay kit (Cat. # GAHK20). Cholesterol and triglyceride concentrations were measured enzymatically using Thermo DMA (Louisville, CO) kits. Non-HDL cholesterol concentrations were calculated as the difference between total and HDL

cholesterol levels. Atherosclerotic lesion sizes in the aortic root and the left carotid artery of F2 mice were measured on oil red O stained sections as we reported [3–7].

2.3. Genotypic analysis

DNA was prepared from tail clips of mice and genotyped as described [8–10]. 127 microsatellite markers across the entire genome at an average interval of 12 cM were typed.

2.4. Statistical analysis

QTL analysis was performed using J/qtl and Map Manager QTX as reported [11–15]. One thousand permutations were run to define the genome-wide LOD (logarithm of odds) score thresholds for significant or suggestive linkage of each trait. Loci exceeding the LOD score threshold of 0.05 were significant (p < 0.05) and those exceeding the threshold of 0.63 were suggestive (p < 0.63). Regression analysis was performed to dissect the contribution of a metabolic trait to the variation in plasma MDA levels of F2 mice.

Acknowledgments

The work was supported by NIH grants R01 DK116768 and HL112281 and Commonwealth Health Research Board (CHRB) Virginia.

Conflict of Interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.dib.2020.105165.

References

- D.T. Fuller, A.T. Grainger, A. Manichaikul, W. Shi, Genetic linkage of oxidative stress with cardiometabolic traits in an intercross derived from hyperlipidemic mouse strains, Atherosclerosis 293 (2020) 1–10, https://doi.org/10.1016/j. atherosclerosis.2019.11.034.
- [2] Z. Zhang, J.S. Rowlan, Q. Wang, W. Shi, Genetic analysis of atherosclerosis and glucose homeostasis in an intercross between C57BL/6 and BALB/cJ apolipoprotein E-deficient mice, circulation, Cardiovasc. Genet. 5 (2012) 190–201, https://doi. org/10.1161/CIRCGENETICS.111.961649.
- [3] W. Shi, Z. Zhang, M.H. Chen, J.F. Angle, A.H. Matsumoto, Genes within the MHC region have a dramatic influence on radiation-enhanced atherosclerosis in mice, Circulation, Cardiovasc. Genet. 3 (2010) 409–413, https://doi.org/10.1161/ CIRCGENETICS.110.957449.
- [4] A. Manichaikul, Q. Wang, Y.L. Shi, Z. Zhang, N. Leitinger, W. Shi, Characterization of Ath29, a major mouse atherosclerosis susceptibility locus, and identification of Rcn2 as a novel regulator of cytokine expression, Am. J. Physiol. Heart Circ. Physiol. 301 (2011) H1056–H1061, https://doi.org/10.1152/ajpheart.00366.2011.
- [5] Q. Li, Y. Li, Z. Zhang, T.R. Gilbert, A.H. Matsumoto, S.E. Dobrin, W. Shi, Quantitative trait locus analysis of carotid atherosclerosis in an intercross between C57BL/6 and C3H apolipoprotein E-deficient mice, Stroke – J. Cerebr. Circ. 39 (2008) 166–173, https://doi.org/10.1161/STROKEAHA.107.492165.
- [6] A.T. Grainger, M.B. Jones, M.H. Chen, W. Shi, Polygenic control of carotid atherosclerosis in a BALB/cJ x SM/J intercross and a combined cross involving multiple mouse strains, G3 (Bethesda, Md.) 7 (2017) 731–739, https://doi.org/10.1534/g3.116. 037879.
- [7] A.T. Grainger, M.B. Jones, J. Li, M.H. Chen, A. Manichaikul, W. Shi, Genetic analysis of atherosclerosis identifies a major susceptibility locus in the major histocompatibility complex of mice, Atherosclerosis 254 (2016 Nov) 124–132, https://doi. org/10.1016/j.atherosclerosis.2016.10.011.
- [8] Z. Su, Y. Li, J.C. James, M. McDuffie, A.H. Matsumoto, G.A. Helm, J.L. Weber, A.J. Lusis, W. Shi, Quantitative trait locus analysis of atherosclerosis in an intercross between C57BL/6 and C3H mice carrying the mutant apolipoprotein E gene, Genetics 172 (2006) 1799–1807, https://doi.org/10.1534/genetics.105.051912.
- [9] Z. Yuan, H. Pei, D.J. Roberts, Z. Zhang, J.S. Rowlan, A.H. Matsumoto, W. Shi, Quantitative trait locus analysis of neointimal formation in an intercross between C57BL/6 and C3H/HeJ apolipoprotein E-deficient mice, Circulation, Cardiovasc. Genet. 2 (2009) 220–228, https://doi.org/10.1161/CIRCGENETICS.108.792499;10.1161/CIRCGENETICS.108.792499.

- [10] Z. Yuan, Z. Su, T. Miyoshi, J.S. Rowlan, W. Shi, Quantitative trait locus analysis of circulating adhesion molecules in hyperlipidemic apolipoprotein E-deficient mice, Mol. Genet. Genom.: MGG. 280 (2008) 375–383, https://doi.org/10.1007/ s00438-008-0371-0.
- [11] Z. Su, Y. Li, J.C. James, A.H. Matsumoto, G.A. Helm, A.J. Lusis, W. Shi, Genetic linkage of hyperglycemia, body weight and serum amyloid-P in an intercross between C57BL/6 and C3H apolipoprotein E-deficient mice, Hum. Mol. Genet. 15 (2006) 1650–1658, https://doi.org/10.1093/hmg/ddl088.
- [12] J.S. Rowlan, Z. Zhang, Q. Wang, Y. Fang, W. Shi, New quantitative trait loci for carotid atherosclerosis identified in an intercross derived from apolipoprotein E-deficient mouse strains, Physiol. Genom. 45 (2013) 332–342, https://doi.org/10. 1152/physiolgenomics.00099.2012;10.1152/physiolgenomics.00099.2012.
- [13] S.W, J.S. Rowlan, Q. Li, A. Manichaikul, Q. Wang, A.H. Matsumoto, Atherosclerosis susceptibility loci identified in an extremely atherosclerosis-resistant mouse strain, J. Am. Heart Assoc. 2 (2013), e000260. https://doi:10.1161/JAHA.113. 000260.
- [14] N.E. Garrett 3rd, A.T. Grainger, J. Li, M.H. Chen, W. Shi, Genetic analysis of a mouse cross implicates an anti-inflammatory gene in control of atherosclerosis susceptibility, Mamm. Genome: Off. J. Int. Mamm. Genome Soc. 28 (2017) 90–99, https:// doi.org/10.1007/s00335-016-9677-0.
- [15] Q. Wang, A.T. Grainger, A. Manichaikul, E. Farber, S. Onengut-Gumuscu, W. Shi, Genetic linkage of hyperglycemia and dyslipidemia in an intercross between BALB/cJ and SM/J Apoe-deficient mouse strains, BMC Genet. 16 (2015) 133, https:// doi.org/10.1186/s12863-015-0292-y.