Dataset of TWIST1-regulated genes in the cranial mesoderm and a transcriptome comparison of cranial mesoderm and cranial neural crest

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This article contains data related to the research article entitled “Transcriptional targets of TWIST1 in the cranial mesoderm regulate cell-matrix interactions and mesenchyme maintenance” by Bildsoe et al. (2016) [1]. The data presented here are derived from: (1) a microarray-based comparison of sorted cranial mesoderm (CM) and cranial neural crest (CNC) cells from E9.5 mouse embryos; (2) comparisons of transcription profiles of head tissues from mouse embryos with a CM-specific loss-of-function of Twist1 and control mouse embryos collected at E8.5 and E9.5; (3)
ChIP-seq using a TWIST1-specific monoclonal antibody with chromatin extracts from TWIST1-expressing MDCK cells, a model for a TWIST1-dependent mesenchymal state. © 2016 The Authors. Published by Elsevier Inc. This is an open access article under the CC BY license (http://creativecommons.org/licenses/by/4.0/).

### Specifications Table

| Subject area | Biology          | More specific subject area | Developmental Biology |
|--------------|------------------|----------------------------|-----------------------|
| Type of data | Tables           | Data format                | Analyzed              |
| How data was acquired | Illumina Mouse WG-6 v2 arrays; Chromatin-immunoprecipitation and next generation sequencing | Experimental factors | Samples for microarray analysis were collected from either FACS sorted GFP-positive embryonic head tissues, or whole embryo heads. Chromatin for ChIP-seq was collected from MDCK cells over-expressing human TWIST1. |
| Experimental features | Transcriptome comparison between sorted E9.5 cranial mesoderm (CM) and neural crest cells. Twist1 conditional knockout and control tissues (E8.5 & E9.5). TWIST1 genomic binding sites in MDCK cells. | Data source location | Children's Medical Research Institute, Sydney Medical School, University of Sydney, Australia |
| Data accessibility | The microarray and ChIP-sequencing data within this article are accessible in GEO under accession number GEO: GSE80663. http://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE80663 |

### Value of the data

- The data set provides an important reference for all studies investigating Twist1 function in the context of development and cancer.
- By comparing the transcriptome of the cranial mesoderm and cranial neural crest, the data set provide a useful tool for studying the complex process of craniofacial development.
- The data set potentially contributes to the identification of genes that control the mesenchymal cell state in development and cancer.

### 1. Data

Dissociated craniofacial tissues that were FACS-sorted by GFP expression reporting either Mesp1-Cre or Wnt1-Cre activity were compared using microarrays (Supplementary Tables 1 and 2). Dissected embryo heads of control (Twist1\(^{\text{flox/+}}\)), heterozygote (Twist1\(^{\text{del/+}}\)), mesoderm heterozygote (Twist1\(^{\text{flox/+}}\); Mesp1\(^{\text{Cre/+}}\)) and conditional knockout (Twist1\(^{\text{floxdel}}\); Mesp1\(^{\text{Cre/+}}\)) (Supplementary Tables 3 and 4) genotypes were compared using microarrays. Chromatin immunoprecipitation using an anti-TWIST1 antibody was performed on MDCK cells that express human TWIST1 (Supplementary Table 5).

### 2. Experimental design, materials and methods

#### 2.1. Isolation and analysis of CM and CNC populations

Embryo were collected at E9.5 from Mesp1-Cre x Z/EG (for CM) and Wnt1-Cre x Z/EG (for CNC) [2–4]. Heads were dissected below the first branchial arch, dissociated and prepared for cell sorting as
described [2]. Each sample yielded 4000–18,000 GFP-positive cells, which were stored at −80 °C.

RNA was extracted and amplified using Illumina TotalPrep (Ambion) and labeled using MessageAmp II aRNA (Ambion) as described elsewhere [1].

2.2. Transcriptomic analysis of Twist1-conditional mutant embryos

Embryos in which Twist1 was specifically ablated in the anterior mesoderm were generated using Mesp1-Cre [1,3,5–7]. Embryo heads were collected at E8.5 (5–7 somites) and E9.5 (18–20 somites). Four genotypes were analyzed: CM-KO (Twist1^flox/del; Mesp1^Cre/+), CM-Het (Twist1^flox/wt; Mesp1^Cre/+), Het (Twist1^flox/del; Mesp1^-/-) and Control (Twist1^flox/wt; Mesp1^-/+). RNA labeling and hybridization was carried out by the Australian Genome Research Facility.

2.3. Chromatin Immunoprecipitation

ChIP was carried out using extracts of TWIST1-expressing MDCK cells [8]. Cross-linking in 1% formaldehyde, lysis and sonication were carried out as described [1]. Extracts were pre-cleaned by incubation with A/G magnetic beads (Dynal) for 3 hrs and incubated with an anti-TWIST1 monoclonal antibody (Abcam ab50887) overnight at 4 °C, before adding blocked beads and subsequent washing steps in RIPA buffer, RIPA/NaCl buffer and LiCl buffer [1]. Sequencing was carried out by the Australian Genome Research Facility.

2.4. Data analysis

Raw microarray data were log2 transformed, quantile normalized and differential expression analyzed using the Linear Models for Microarray (LIMMA, [9] implementation within Gene Pattern. Differentially expressed genes were filtered on a false discovery rate (FDR) of 0.05.

For ChIP-Seq data, 50 bp reads were trimmed using Cutadapt [10], filtered by quality score and aligned to the CanFam3 dog genome using bowtie2 [11] as described [1]. Peaks were called using MACS2 [12] and IDR analysis performed using an IDR cut-off of 0.05. Peak coordinates from two replicates were merged, using the most extreme start and end positions of the two replicates. The equivalent mouse genome (mm10) peak genomic locations were determined using Liftover (NCBI) annotated using the R library ChipSeeker.

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Transparency document. Supporting information

Transparency data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2016.09.001.
Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.dib.2016.09.001.

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