

Fig. S1. *celf1* plays a crucial role in symmetric somitogenesis and LR asymmetric patterning. (A) *celf1_long*-MO and *celf1_short*-MO inhibited the expression of *celf1L*-GFP and *celf1S*-GFP, respectively. (B) Western blotting using anti-Celf1 antiserum. Left panel: co-injection of *celf1_long*-MO with *celf1_short*-MO (*celf1*-MOs) inhibited the expression of both forms of endogenous Celf1 in zebrafish embryos. The asterisk indicates a non-specific band. Right panel: Celf1 and Celf1^{Δlinker} proteins were detected in Celf1- and Celf1^{Δlinker}-overexpressing embryos, respectively. (C) Representative images of *myod* expression demonstrating normal (left; control-MO, *n*=105) or abnormal (right; *celf1*-MOs, *n*=88) somitogenesis in embryos at 24–28 hpf. Higher magnification images (lower panels) highlight somites. Knockdown of *celf1* resulted in a mild loss of the chevron shape of somites (94%). (D) Upregulation of *myod* expression in *celf1* morphants. qPCR assay revealed that *celf1* knockdown resulted in a 50% increase of *myod* expression in zebrafish embryos, suggesting that *myod* is a target of Celf1 not only in C2C12 (Lee et al., 2010) but also in zebrafish.

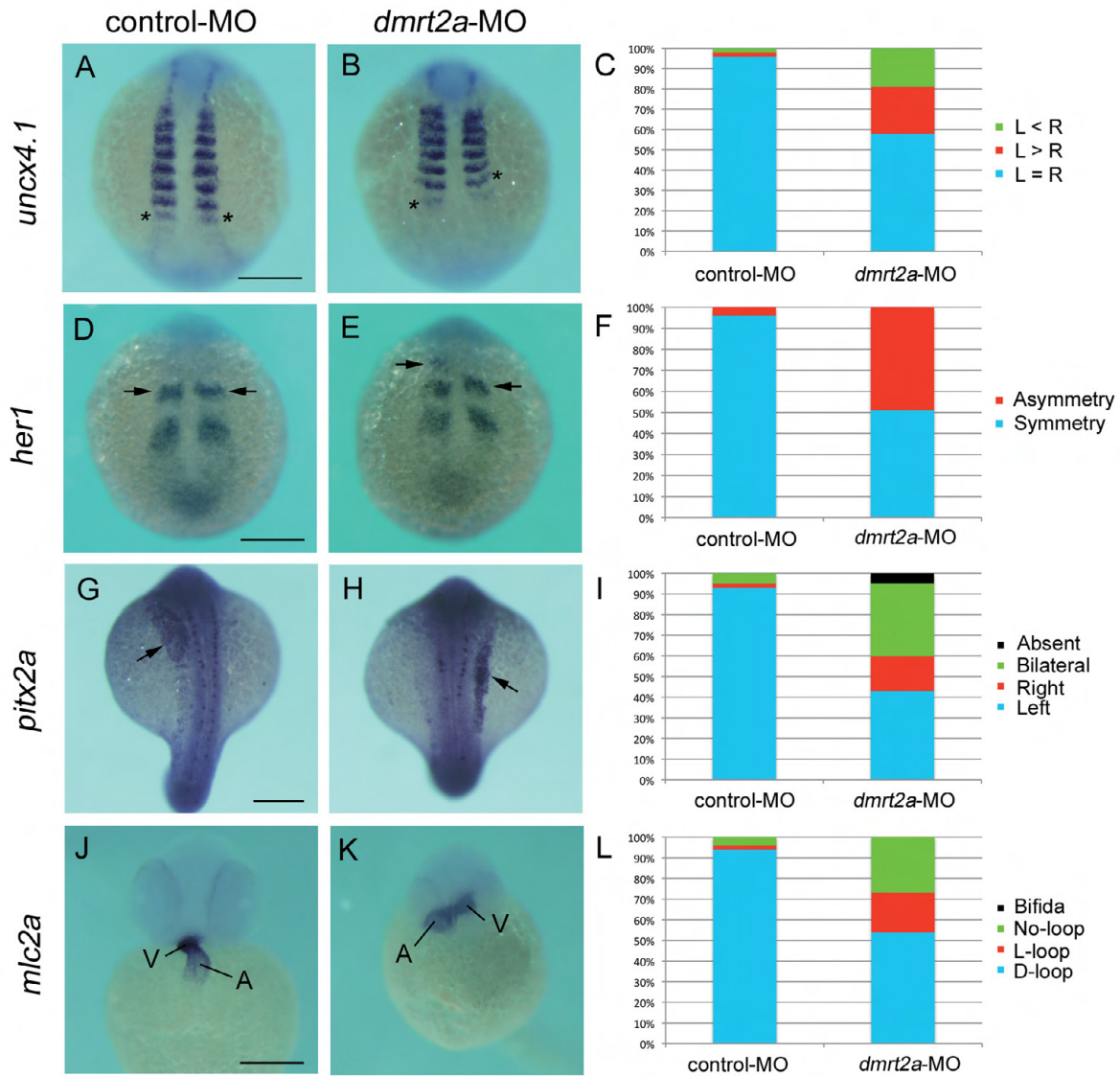


Fig. S2. Knockdown of *dmrt2a* yields defects similar to those seen in *celf1*-overexpressing embryos. (A,B,D,E,G,H,J,K) In situ hybridization for *uncx4.1* (A,B), *her1* (D,E), *pitx2a* (G,H) or *mlc2a* (J,K) in control-MO-injected (A,D,G,J) or *dmrt2a*-MO-injected (B,E,H,K) embryos. Asterisks in A and B mark the last-formed somite. Arrows in D, E, G and H indicate the position of the anterior strip of *her1* and *pitx2a* expression in the lateral plate mesoderm, respectively. A, atrium; V, ventricle. Scale bar: 200 μ m. (C) Percentages of symmetric (L=R), left-biased (L>R) or right-biased (L<R) asymmetric somitogenesis in embryos injected with control-MO ($n=44$) or *dmrt2a*-MO ($n=53$). (F) Percentages of symmetric and asymmetric *her1* oscillation in embryos injected with control-MO ($n=48$) or *dmrt2a*-MO ($n=51$). (I) Percentages of left-sided, right-sided, bilateral, or no (absent) expression of *pitx2a* in embryos injected with control-MO ($n=59$) or *dmrt2a*-MO ($n=46$). (L) Percentages of D-loop, L-loop, no-loop or cardia bifida of the heart in embryos injected with control-MO ($n=58$) or *dmrt2a*-MO ($n=78$).

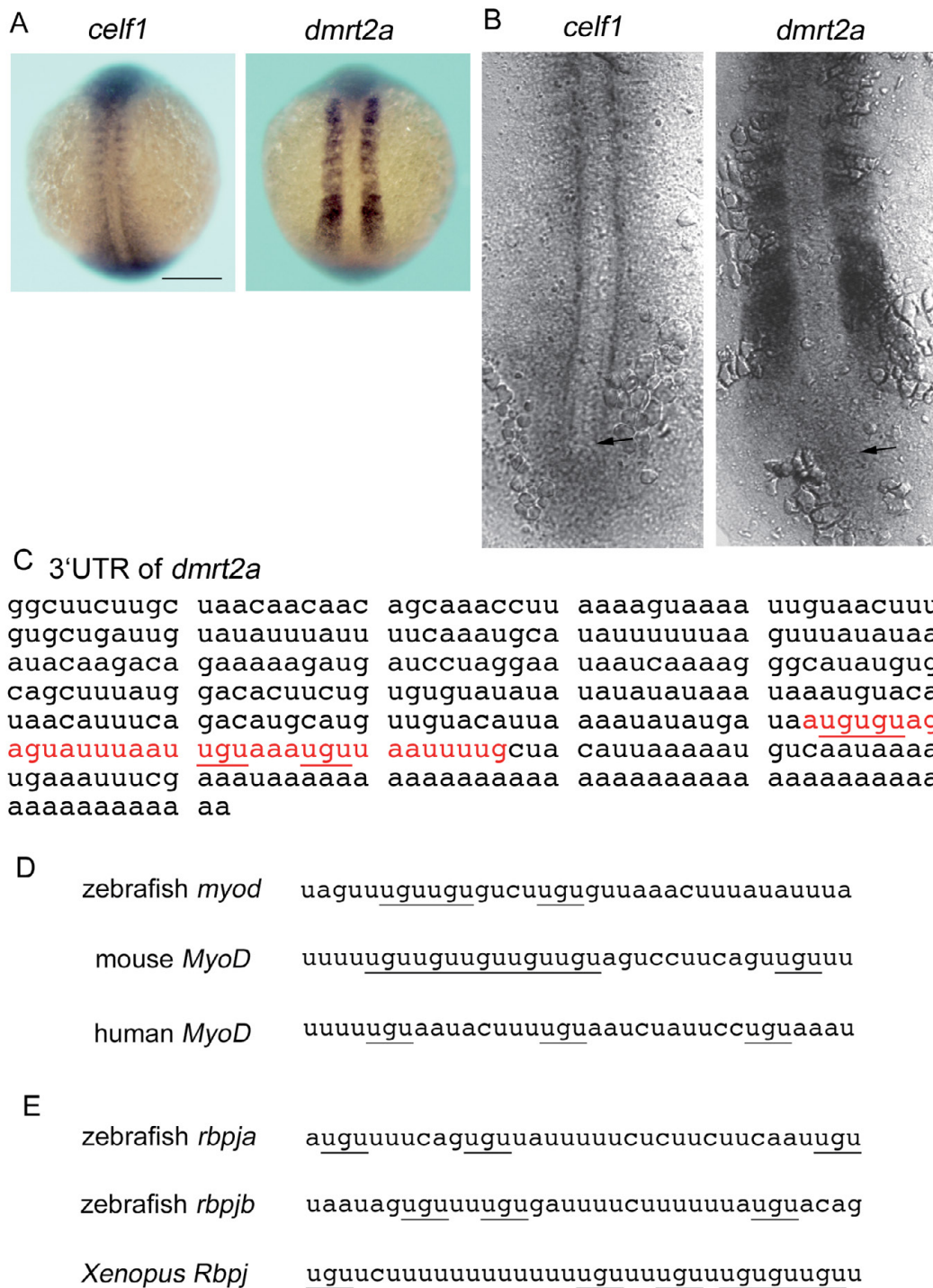


Fig. S3. *dmrt2a* mRNA is a target of Celf1. (A,B) *celf1* (left) or *dmrt2a* (right) expression in zebrafish embryos at 12-14 hpf. (A) Dorsal view of whole-mount embryos. Scale bar: 200 μ m. (B) Dorsal view of flat-mounted embryos. Arrows mark the position of Kupffer's vesicle. (C) Sequence of the 3'UTR of *dmrt2a* mRNA. Red letters are the putative Celf1-binding site including UGU repeats (underlined), and U- and A-rich sequences. The 35 nucleotides were used as a probe named *dmrt2a* wildtype for an in vitro binding assay (see also Fig. 4A). (D,E) The putative Celf1-binding sites including UGU repeats (underlined) and U- and A-rich sequences were found within 3'UTR of *myod* (D) or *rbpj* (E) mRNA, but sequence homology was low among species.

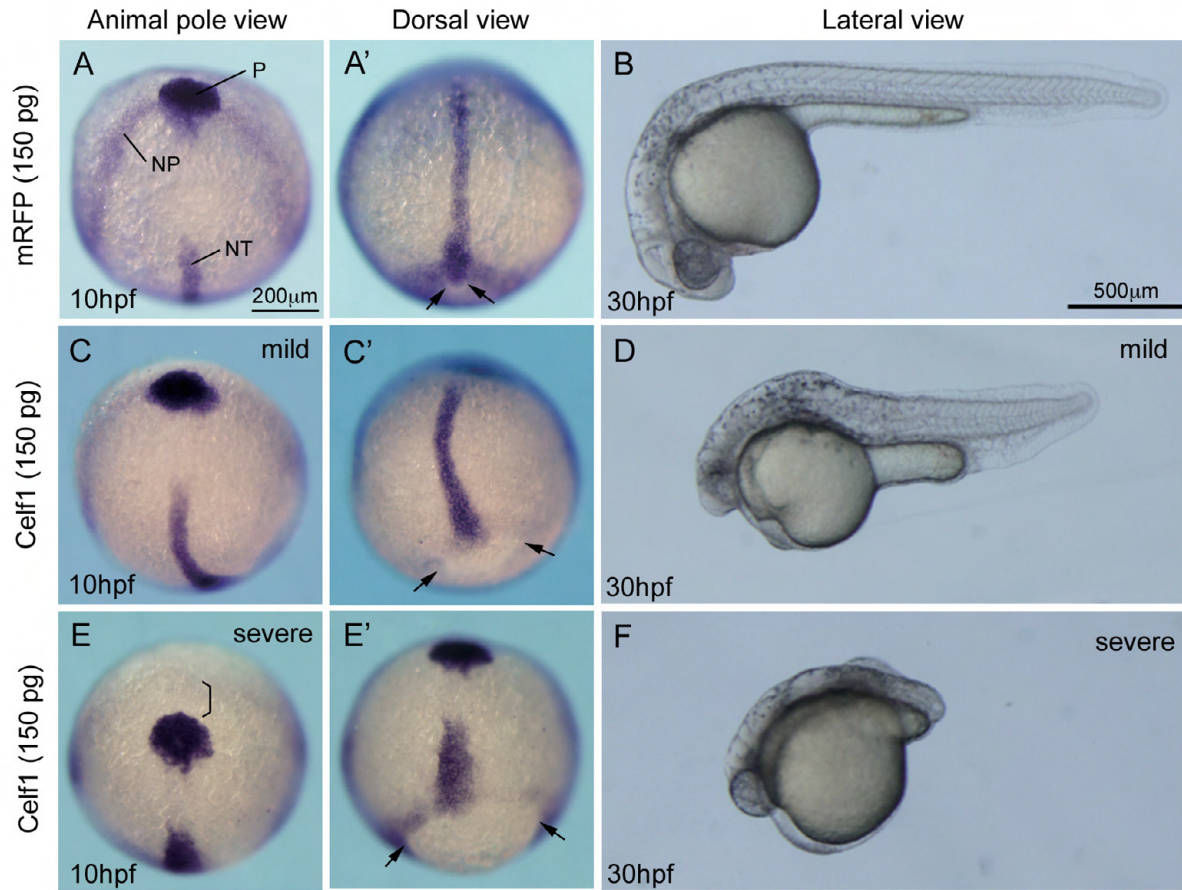


Fig. S4. Higher amounts of *celf1* mRNA lead to additional defects in zebrafish. (A,C,E) Expression of *hgg1* (P, polster), *dlx3* (NP, anterior edge of the neural plate) or *ntl* (NT, notochord) in embryos injected with *mRFP* (A) or *celf1* (C,E) mRNAs (150 pg). Animal pole view. Scale bar: 200 μ m. (A',C',E') Dorsal view of the embryo, anterior to the top. Injection of 150 pg *celf1* mRNA resulted in the formation of bended (C') or short (E') notochord. The polster did not reach the anterior edge of the neural plate (bracket in E). Epiboly was incomplete (C',E'). Arrows in A', C' and E' mark edge of the yolk plug. (B,D,F) Lateral view of embryos injected with *mRFP* (B) or *celf1* (D, F) mRNAs (150 pg) at 30 hpf. Various phenotypes, such as short tails, segmentation defects, small eyes, small heads and less pigmentation, were observed (D,F).