

Supplemental figure 1: *sip1a* splice morpholino is effective in eliminating wild-type *sip1a* mRNA. RT-PCR of mRNA from staged *sip1a* splice MO injected embryos with *sip1a* specific primers reveals that the smaller misspliced product is detected by dome stage (4.3 hpf). By 75% epiboly (8 hpf), no wild-type mRNA is detected.

Supplemental figure 2: Comparison of zebrafish Sip1a and Sip1b proteins. Genedoc software is used to align the predicted protein sequences. Predicted domains of Sip1a and Sip1b is lined with colored bars: C2H2 type zinc fingers present in both Sip1a and Sip1b (red), C2H2 type zinc finger region that is absent from Sip1a and Sip1b short forms (blue). Identical and similar amino acids conserved among all proteins are shown in black and dark gray boxes, respectively. Lighter shades of gray or no shading represent low levels of amino acid conservation and the lack of conservation, respectively.

Supplemental figure 3: *sip1a* splice variant targeting morpholinos efficiently eliminate short and long forms. Diagram of the alternative splicing within the 3' region of the *sip1a* pre-mRNA (A). Alternative splicing of exon 8 eliminates one zinc finger (dark blue box) which is present in the longer form. Sip1a MO3 (orange bar) targeted to alternative splice site in exon 8 (denoted by black arrow) blocks the alternating splicing event and eliminates production of the short form. Sip1a MO2 (pink bar) blocks the pre-mRNA splicing event needed to generate the long form by targeting the splice site at the 3' end of exon 8. (B) RT-PCR of mRNA from staged *sip1a* splice MO2 and MO3 injected embryos with *sip1a* full length specific primers (B, green arrows in A) and *sip1a* short form specific primers (C, orange arrows in A). *sip1a* splice MO2 efficiently altered splicing so that the full length message is eliminated and only the shorter form was produced (B) while *sip1a* splice MO3 blocked production of the shorter form (C).

Supplemental figure 4: Pace of the “molecular clock” is not significantly altered in ChCh-compromised embryos. Living wild type and ChCh-compromised embryos beginning at the 7 somite stage. All views are dorsal, anterior to the top (A-J). Duration of somite formation in both control (A-E) and ChCh-compromised embryos (F-J) is approximately 45 mins at 23°C. Black arrows denote already formed somite boundaries and white arrows denote newly forming segmentation furrow.

Supplemental figure 5: Somite malformation in ChCh and Sip1a compromised embryos can be partially rescued by FGF antagonist Spry4. Bar graph representation of *spry4* rescue assay in *chch* and *sip1a* ATG morphants. Injection of *spry4* sense RNA into *chch* and *sip1a* ATG morphants (blue bar) reduced the penetrance of the somite phenotype with respect to their *lacZ* injected siblings (gray bar).