Abstract/Session Information for Program Number 131

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Abstract Content

Program Nr: **131**

Characterization of the Synthetic Multivulva Suppressor *isw-1*, the *C. elegans* Homolog of the Chromatin-Remodeling ATPase ISWI. **Erik C. Andersen**, Xiaowei Lu, Scott Clark, Bob Horvitz. HHMI, Dept. Biology, MIT, Cambridge, MA.

The synthetic Multivulva (synMuv) genes are grouped into two functionally redundant classes, A and B, that negatively regulate the induction of vulval cell fates. Animals with a mutation in one or more genes within the same class are non-Muv. By contrast, animals with mutations in both a class A and a class B gene are Muv. Among some of the identified class B gene products are counterparts of a transcriptional repression complex: Rb (LIN-35), the Rb-associated protein RbAp48 (LIN-53), the heterodimeric transcription factor DP/E2F (DPL-1/EFL-1), and the chromatin-regulating proteins HDAC (HDA-1), Mi-2 (LET-418), and HP1 (HPL-2).

To understand better the synMuv A and B pathways, two screens for synMuv suppressors were performed. In these screens, 166 suppressors of the synMuv phenotype of *lin-15AB(n765*ts*)* and 43 suppressors of the *lin-53(n833); lin-15A(n767)* synMuv phenotype were isolated. One *lin-53(n833); lin-15A(n767)* synMuv suppressor was cloned and found to encode a homolog of the chromatin-remodeling ATPase ISWI. RNAi or a presumptive null allele of this gene, which we call *isw-1*, suppresses the Muv phenotype of most, if not all, synMuv mutant combinations. The loss-of-function Vul phenotype of mutants in the RTK/Ras pathway (except *lin-3* EGF) is epistatic to the synMuv phenotype. Loss of *isw-1* function cannot suppress the Muv phenotype of *lin-1* mutants or a gain-of-function mutation in *let-60*. We conclude that *isw-1* is acting downstream of or in parallel to one or both synMuv classes and upstream of or in parallel to the RTK/Ras pathway.

ISWI has been biochemically purified from *Drosophila* embryo extracts as a member of multiple complexes, including the Nucleosome Remodeling Factor (NURF), the Chromatin Accessibility Complex (CHRAC), and the ATP-utilizing Chromatin assembly and remodeling Factor (ACF). RNAi of *C. elegans* homologs of NURF, CHRAC, and ACF complex members led us to identify a NURF301 homolog, *F26H11.2*, as a gene that likely acts with *isw-1* to regulate vulval development. NURF301 is the largest subunit of the NURF complex and is implicated in complex formation, stability and interactions with transcription factors. We are seeking a deletion allele of *F26H11.2* to further characterize the activity of the putative NURF complex in antagonism of the synMuv phenotype. Furthermore, we are mapping and cloning additional mutationally-defined suppressors of the synMuv phenotype in an effort to identify other factors that may act with ISW-1 or a putative *C. elegans* NURF complex.

Session Information

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