

P036 Pachytene asynapsis drives meiotic sex chromosome inactivation and results in post-meiotic repression in spermatids

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Transcriptional silencing of the sex chromosomes during male meiosis (MSCI) is conserved among organisms with limited sex chromosome synapsis, including mammals. Since the 1990's the prevailing view has been that MSCI in mammals is transient, with sex chromosome reactivation occurring as cells exit meiosis. Recently, we found that any chromosome region unsynapsed during pachytene of male and female mouse meiosis is subject to transcriptional silencing (MSUC) and hypothesised that MSCI is an inevitable consequence of this more general meiotic silencing mechanism. Here, we provide direct evidence that asynapsis does indeed drive MSCI, and that MSCI is essential for progression through pachytene. We also show that a substantial degree of transcriptional repression of the sex chromosomes is retained post-meiotically, and provide evidence that this post-meiotic repression is a downstream consequence of MSCI/MSUC. While this post-meiotic repression is after the loss of MSUC-related proteins at the end of prophase, other histone modifications associated with transcriptional repression have by then become established.