interaction of Rbm with $Tra2\alpha$, whose function is in relation to RNA splicing. Interestingly, interaction between $Tra2\alpha$ and hnRNP K was also observed. In transfected tissue culture cells, Rbm and hnRNP K appeared to be co-localized in the nucleus.

Conclusions: Interactions of Rbm with hnRNP K and Tra2 α suggest its function in RNA-splicing during spermatogenesis. In fact, it was well documented that a number of transcripts are spliced alternatively in male germ cells in developmental stage-specific manner and play critical functions in spermatogenesis. Further studies to confirm Rbm interactions in vivo are in progress.

P-27 The Effect of Blocking of Occludin on Blastocoel Formation and Trophectoderm Differentiation in Mouse Preimplantation Embryos

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Tight junction (TJ) formation is critical for blastocoel formation in the mammailan embryos. Occludin, one of the TJ molecules, assembly occurs at late morula stage and leads to the establishment of a permeability seal to maintain the integrity of the cavitating blastocyst. In this study, the role of occludin on blastocyst morphogenesis was verified in mouse preimplantation embryos. Early and late morula were cultured in KSOM in the presence or absence of 0.05% sodium azide (SA), mouse seurm (6 μ g/ μ l), and/or occludin antibodies (6 μ g/ μ l) for 24 h and 14 h, respectively. Blastocoel formation was observed in the embryos cultured in control medium and the medium containing 0.05% SA + mouse serum, but not in the medium containing occludin antibodies (early morula 0%, late morula 46%). Blastocoel formation rate decreased in the presence of occludin antibodies in a dose-dependent manner. Expression of trophectoderm-specific gene (h19) was examined in the cultured embryos by RT-PCR. h19 transcript was not detected in the embryos cultured in the medium containing occludin antibodies. These results suggest that occludin engages in the blastocoel formation and contributes trophectoderm differentiation and that h19 gene is expressed after blastocoel formation.