Drosophila zinc finger protein CG9890 is localized on the promoters of active genes and involved in the regulation of both basal and inducible transcription.

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In previous studies, we showed that Drosophila zinc finger Protein CG9890 is localized in the nucleus and interacts with chromatin modifying and remodeling complexes SAGA and dSWI/SNF as well as with ORC complex, which is necessary for the positioning of the replication origins. ChIP-Seq of CG9890 protein revealed that the protein is localized mostly on the promoters of active genes. In this work we decided to investigate the role of CG9890 in transcription regulation, given that CG9890 interacts with main transcriptional complexes and was found predominantly on gene promoters including promoters of ecdysone-dependent genes. To this end we decreased the level of CG9890 in Drosophila S2 cells by RNA interference and analyzed the changes in the level of mRNA 21 of the CG9890-associated gene compared to the control samples. After knockdown of the CG9890 protein, the amount of mRNA of ten of these genes changed, including five ecdysone-dependent genes ftz-f1, hr39, CG15279, Eip78C and Eip75B. To investigate the role of CG9890 in the regulation of inducible transcription we have performed analysis of activation of ecdysone-dependent genes hr4 and dhr3 after 20-hydroxyecdysone treatment in cell upon RNA interference of CG9890 protein compared to the control cells. As expected, the mRNA level of both genes significantly increased after ecdysone induction (882 times for dhr3 and 148 times for hr4) in control cells. After knockdown of the CG9890 protein activation of hr4 and dhr3 genes was significantly lower (369 and 45 respectively). Thus, the CG9890 protein is a new member of the cell transcriptional network which is localized on active promoters, interacts with the main transcription and replication complexes, and is involved in the regulation of both basal and inducible transcription. This work was supported by the Russian Science Foundation (Grant No. 20-14-00269).