JOURNAL OF RNAi AND Gene Silencing ISSN 1747-0854

ISSN: 1747-0854 J RNAi Gene Silencing, 2017, Vol 13, 544

EDITORIAL

piRNAs: the "Bodyguards" of Fertility

Valeria Specchia*

Department of Biological and Environmental Sciences and Technologies-University of Salento Ecotekne, SP Lecce - Monteroni, Lecce, Italy

*Correspondence to: Specchia V, E-mail: valeria.specchia@unisalento.it, Tel: +39 0832 29 8673

Received: 01 February 2017; Revised: 05 February 2017; Accepted: 10 February 2017; Published: 15 February 2017

EDITORIAL

Among the small non coding RNAs classes, piRNAs are of particular interest because of their role in allowing the normal reproduction of individuals, from Drosophila to mouse.

The reduction of piRNAs in the germ cells is sufficient to cause the sterility of fruit flies. This damaging effect arises from the spread of transposable elements, normally silenced by the piRNAs, into the genome. The DNA damages caused from mobile elements are directly related to the reproductive deficits.

piRNAs arise from genomic clusters that are composed from partial sequences of mobile elements' debris. They are loaded into Piwi clade proteins and make possible the silencing of transposons at the transcriptional and post-transcriptional level. A lot of different proteins cooperate with the Piwi proteins to regulate the transposons. In the Drosophila germ cells, the transposon transcripts exit the nucleus in a highly controlled manner, to reach a perinuclear region or nuage where a ping-pong mechanism cuts mobile elements' transcripts and amplifies the piRNAs amount. The Piwi proteins, Aubergine and Ago3, are key players in this mechanism. Interestingly, aubergine mutants are sterile. Piwi protein acts mainly at the transcriptional level and the transcriptional repression of mobile elements is performed through formation of heterochromatin. Many actors, such as the zinc finger protein Asterix, or Maelstrom and Panoramix, are crucial in the transcriptional repression.

Loading of piRNA precursors into Piwi proteins requires Heat shock protein 90 (Hsp90) an evolutionary conserved factor. The loss of Hsp90 deregulates transposons and leads to sterility and to an increment in the morphological abnormalities.

An interesting aspect is that in mice the Piwi pathway plays in the spermatogenesis and ensures male fertility and genome integrity. Although with mechanistic differences - mammals use DNA methylation as a silencing mark of transposons- the pathway appears conserved.

piRNAs are also produced in adult human testes and correspond to genes and mobile elements.

It will be interesting to investigate what role could have piRNAs in human infertility conditions.