KIFC1 AND SPINDLE INSTABILITY IN HUMAN OOCYTES

Human eggs, in contrast to those in many other mammalian species, are highly prone to aneuploidy. Recombination failure[1] (posted on December 25, 2020) is considered to be one of the major causes.
In an article published in Science[2], So et al. point to the lack of expression of the KIFC1 gene as another important cause of aneuploidy. This gene codes for a key protein involved in spindle stabilization in other mammalian oocytes and cancer cells. However, surprisingly, its expression is (almost) absent in human oocytes. The artificial depletion of this motor protein in mouse and bovine oocytes results in unstable spindles, as in humans. Conversely, the delivery of the KIFC1 protein into human oocytes reduces spindle instability.

One wonders: first, why is such a basic biological function, the assembly of the spindle apparatus, not well conserved in mammals. Second, why was this deficit in spindle assembly not rejected by natural selection?

1- https://www.cell.com/ajhg/fulltext/S0002-9297(20)30407-9
2- https://www.science.org/doi/10.1126/science.abj3944