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How heat stress drives the expression of LTR retrotransposons in the flatworm model organism *Macrostomum lignano*

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Motivation and Aim: There is a growing body of evidence suggesting that some transposable elements are activated under stress conditions. However, the exact molecular mechanism underlying the activation is rarely known. Recently, we have found that two groups of *Ty3/Gypsy* LTR retrotransposons (LTR-RTs) are highly expressed under heat shock (37 ºC) in the genome of a free-living flatworm *Macrostomum lignano*, a model species to study stem cell biology, regeneration and ageing [1]. Here, we present new evidence for the activation mechanism of these retrotransposons, confirming our previous hypothesis with the new experimental data.
Methods and Algorithms: We utilized the transgenesis technology [2] to investigate whether the long terminal repeat (LTR) sequence of the LTR-RTs alone can induce expression of the reporter fluorescent protein in *M. lignano* after heat shock, and used the promoter of the heat shock protein gene (*Hsp*) 20 as a positive control [3].
Results: Three new *M. lignano* transgenic lines were produced. The heat shock treatment resulted in the expression of the fluorescent reporter controlled both by the LTRs and the *Hsp20* promoters of the transgenic worms. The LTR-driven expression was mostly absent from gonads, while it was ubiquitous in the *Hsp20*-line.
Conclusion: The functional elements of the heat stress activation of the *M. lignano* LTR-RTs are located within their LTRs and regulated by the same mechanism as that for *Hsp* genes.
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References

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