

LETTER

In silico analysis of microRNA-510 as a potential oncomir in human breast cancer

Pawel Gaj and Radoslaw Zagodzón*

See related research by Guo *et al.*, <http://breast-cancer-research.com/content/15/4/R70>

In a recent issue of *Breast Cancer Research*, Guo and colleagues provided mechanistic evidence supporting the role for microRNA (miR)-510 as a novel oncomir playing a pivotal role in human breast cancer via regulation of expression of peroxiredoxin 1 [1]. Their study follows the original observation of elevated miR-510 expression in human breast tumor samples described by Findlay and colleagues in 2008 [2].

To acquire a deeper insight into the potential role of miR-510 in human breast cancer, we carried out an *in silico* analysis of the miR-510 abundance in clinical breast cancer samples using three independent publicly available datasets (described in Additional file 1). Conspicuously, we have found that expression of miR-510 was virtually nonexistent in clinical sample sets (Dataset 1 [3] and Dataset 2 [4]). Only seven out of 473 clinical breast cancer specimens altogether demonstrated the miR-510 miRNA-Seq reads (Figure 1A,B, left), all of which could be attributed to a very low spectrum of the whole expression range of all miRNAs expressed in the respective datasets (Figure 1A,B, right). Analysis of the same kind carried out in 11 normal tissue samples (included in Dataset 2 [4]) revealed one sample expressing a low level of the miR-510 gene (Figure 1C). Similarly, one out of six breast cancer cell

lines (included in Dataset 2) showed detectable expression of miR-510 (Figure 1D).

Analysis of an independent dataset (Dataset 3 [5]) showed expression of miR-510 in a substantial number of cases studied. One should, however, consider that, similar to the previously mentioned miRNA-Seq experiments, the expression of miR-510 in Dataset 3 could be attributed to the very low spectrum of the expression range (Figure 2A), and consider that microarray results are by their nature characterized by a very modest signal-to-noise separation, resulting in a low estimation confidence for the low readouts. Nevertheless, we did attempt to assess whether stratification of Dataset 3 by the median expression signal of miR-510 would correlate with the disease-free survival time (Figure 2B). Results of the analysis revealed that, although the higher strata of miR-510 expression showed a tendency to correlate with slightly worse disease-free survival times (Figure 2B), the correlation did not reach statistical significance ($P = 1.88 \times 10^{-1}$).

Considering our observations and acknowledging the high clarity of mechanistic observations by Guo and colleagues [1], we assume that miR-510 ought to be further evaluated in human cancer types other than breast cancer, especially those in which peroxiredoxin 1 seems to play a role.

* Correspondence: radoslaw.zagodzón@wum.edu.pl
Department of Immunology, Center of Biostructure Research, Medical University of Warsaw, Banacha 1A, 02-097 Warsaw, Poland

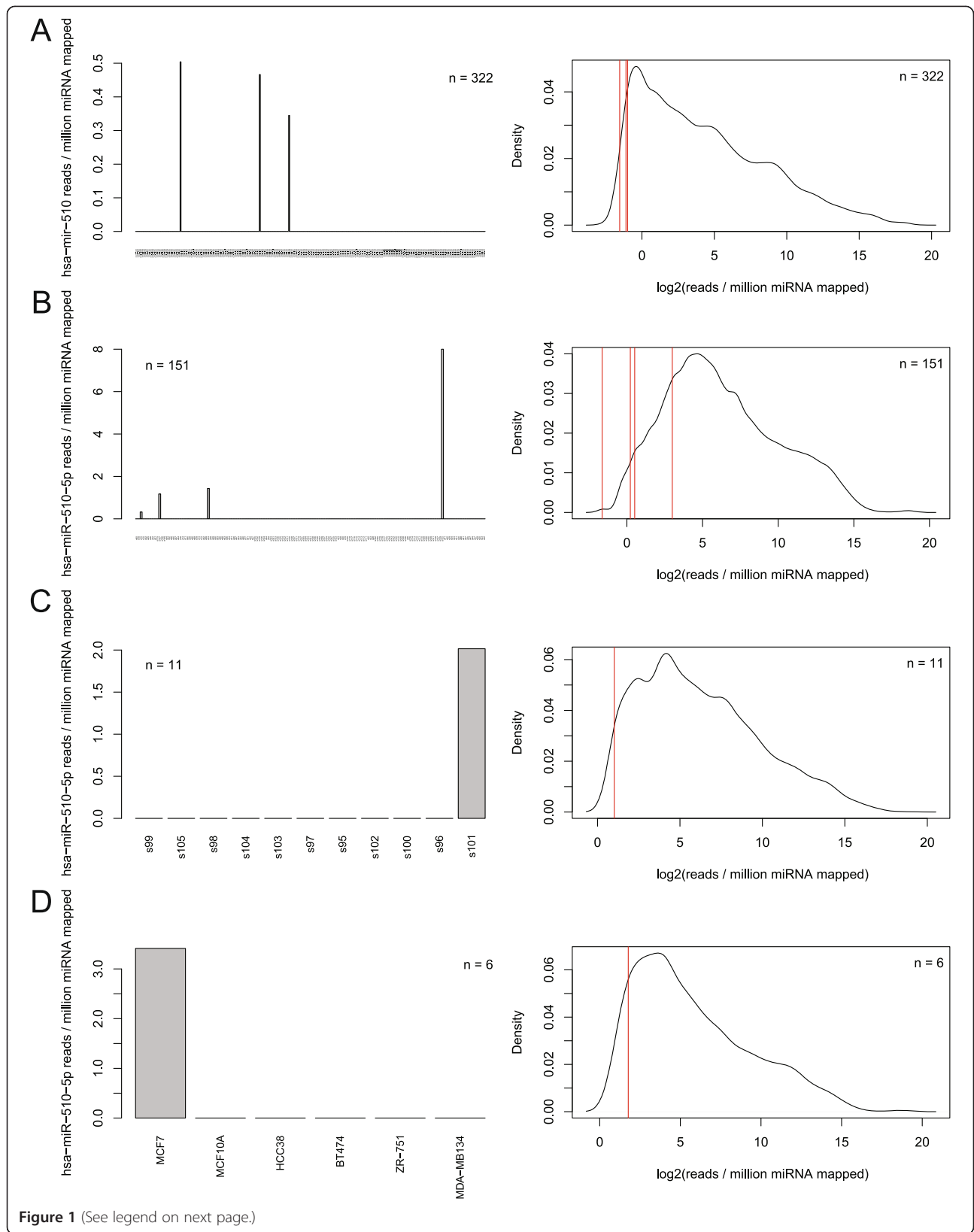


Figure 1 (See legend on next page.)

(See figure on previous page.)

Figure 1 Overview of miR-510 expression in clinical breast cancer samples, normal breast tissues and six breast cancer cell lines using miRNA-Seq experimental results from two independent studies. MicroRNA (miR)-510 expression from (A) Cancer Genome Atlas Network (Dataset 1 [3]) and (B-D) Farazi and colleagues (Dataset 2 [4]). Right: expression levels recorded for the miR-510-positive cases (red bars) in the context of the entire expression distribution of miRNA in the respective parts of the datasets.

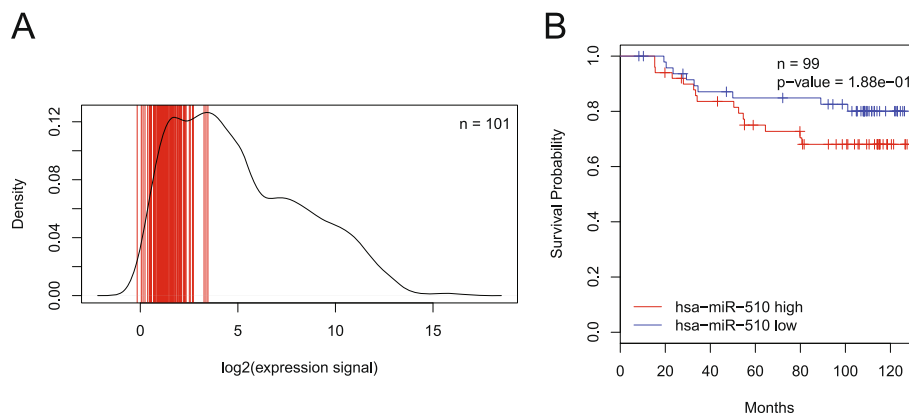


Figure 2 miR-510 expression signal derived from Agilent 4 × 44 K microarray dataset by Enerly and colleagues (Dataset 3 [5]). (A) MicroRNA (miR)-510 expression (red bars) presented against the expression signal distribution of the whole dataset. **(B)** Cox proportional hazard model of survival analysis of the groups stratified by the median value of the miR-510 expression signal. Note: two miR-510-positive cases did not have accompanying survival data assigned, hence the difference in number of cases shown in (A) and (B).

Additional file

Additional file 1: Is the supplemental methods.

Abbreviations

miR: microRNA.

Competing interests

The authors declare that they have no competing interests.

Acknowledgements

This work was supported by a grant from the European Commission 7th Framework Programme: FP7-REGPOT-2012-CT2012-316254-BASTION. The authors wish to express their gratitude to Prof. Jakub Golab and Prof. Krystian Jazdzewski of Medical University of Warsaw for their consultation and resourceful comments.

Published: 12 Mar 2014

References

1. Guo QJ, Mills JN, Bandurraga SG, Nogueira LM, Mason NJ, Camp ER, Larue AC, Turner DP, Findlay VJ: **MicroRNA-510 promotes cell and tumor growth by targeting peroxiredoxin1 in breast cancer.** *Breast Cancer Res* 2013, **15**:R70.
2. Findlay VJ, Turner DP, Moussa O, Watson DK: **MicroRNA-mediated inhibition of prostate-derived Ets factor messenger RNA translation affects prostate-derived Ets factor regulatory networks in human breast cancer.** *Cancer Res* 2008, **68**:8499–8506.
3. The Cancer Genome Atlas Network: **Comprehensive molecular portraits of human breast tumours.** *Nature* 2012, **490**:61–70.
4. Farazi TA, Horlings HM, Ten HJ, Mihailovic A, Halfwerk H, Morozov P, Brown M, Hafner M, Reyal F, van Kouwenhove M, Kreike B, Sie D, Hovestadt V, Wessels L, van de Vijver MJ, Tuschl T: **MicroRNA sequence and expression analysis in breast tumors by deep sequencing.** *Cancer Res* 2011, **71**:4443–4453.

5. Enerly E, Steinfeld I, Kleivi K, Leivonen S-K, Aure MR, Russnes HG, Rønneberg JA, Johnsen H, Navon R, Rødland E, Mäkelä R, Naume B, Perälä M, Kallioniemi O, Kristensen VN, Yakhini Z, Børresen-Dale A-L: **miRNA-mRNA integrated analysis reveals roles for miRNAs in primary breast tumors.** *PLoS One* 2011, **6**:e16915.

10.1186/bcr3624

Cite this article as: Gaj and Zagodzón: *In silico* analysis of microRNA-510 as a potential oncomir in human breast cancer. *Breast Cancer Research* 2014, **16**:403