Toxicology: Open Access

Short Communication Open Access

Microrna: A Review of Emerging Experimental and Epidemiological Data as a Hugely Beneficial Technique in Agricultural Toxicity Testing Screening

Abdo Manuell*

Department of Biochemistry, College of Colchester, Honduras

Abstract

Many biological processes, including development, cell differentiation, proliferation, apoptosis, and tumour growth, are regulated by miRNAs. They can also act as precise, reliable, and sensitive indicators of chemical exposure, according to numerous studies. Current experimental and epidemiological evidence showing dysregulation in miRNA expression in response to fungicides, insecticides, or herbicides were examined in this study. According to Venn's diagrams, let-7, miR-155, miR-181, and miR-21 were found to be often deregulated by at least three different insecticides. In contrast, miR-363 and miR-9 deregulation is connected with fungicide exposure in vitro and in vivo. Additionally, three different herbicides frequently disrupted the expression of let-7, miR-30, miR-126, miR-181, and miR-320.

Keywords: MiRNA; Toxicology; Apoptosis

Introduction

Single-stranded, non-coding RNA molecules called microRNAs (miRNAs), which have 19 to 24 nucleotides, regulate gene expression by having an impact on many biological processes and fine-tuning the abundance and translation ratio of mRNAs. MiRNAs control a variety of cellular and developmental processes in eukaryotic species. Several mRNAs may be the targets of a single miRNA, which by base pairing completes the messenger's 3′ UTR. Partial or complete coupling is possible, however for mRNA recognition; a miRNA sequence called the "Seed sequence"—consisting of 2–8 nucleotides from 3′ to 5′ —is thought to be adequate [1,2].

Methods

Expression of miRNAs and fungicide exposure

Chemical substances known as fungicides prevent or eliminate the growth of fungi and their spores. They are made up of a lot of different chemicals with different chemical structures that are frequently used to protect crops from fungus and moulds. The use of fungicides has grown essential in recent decades in the agricultural system since it is believed that fungal infections globally reduce crop yields by around 20% [3, 4].

MiRNA expression and herbicide exposure

Herbicides are substances that are applied to plants to prevent them from growing or to kill them, including weeds, invasive species, and agricultural pests. Herbicides have less acute toxicity than insecticides in non-target species, such as humans, because most methods of herbicidal action involve metabolic processes that are specific to plants [5, 6].

For instance, the herbicide glyphosate works by preventing plants from making aromatic amino acids, which is one of its main modes of action [7].

In order to investigate the potential value of miRNAs as biomarkers in pesticide toxicological risk assessment, this article summarises recent experimental and epidemiological data examining dysregulation in miRNA expression in response to fungicides, insecticides, or herbicide exposure. In order to determine if there is a direct pesticide effect or whether dysregulation is the outcome of the cellular response to pesticide exposure, we also sought to understand the mechanisms relating to the deregulation of miRNAs upon pesticide exposure [8, 9].

Conclusions

MiRNAs can act as precise, reliable, and sensitive indicators of chemical exposure, according to numerous studies. Current experimental and epidemiological data that demonstrate dysregulation in miRNA expression in response to fungicides, insecticides, or herbicides in vitro and in vivo were examined in this study. It was discovered that the fungicides triadimefon and propiconazole exclusively deregulated the miRNA miR-363 [10].

Acknowledgments

We appreciate Ana Rosa Flores Márquez's support, Makso Herman's assistance with the English editing of the paper, and Ilse Bonilla's work on the Graphical Abstract.

Declaration of competing interest

The authors affirm that they have no known financial or interpersonal conflicts that would have appeared to have an impact on the research presented in this study.

References

1. Sarin SK, Chandan K, Zaigham A (2014) Acute-on-chronic liver failure: consensus recommendations of the Asian Pacific Association for the study of the liver (APASL) Hepatol. Int 8: 453-471.

2. Sarin SK, Kumar A (2009) Acute-on-chronic liver failure: consensus recommendations of the Asian Pacific Association for the study of the liver (APASL) Hepatol. Int 3(269): 282.

3. Choudhury A, Jindal A, Maiwall R, Sharma M K, Sharma B C, et al. (2017)

***Corresponding author:** Abdo Manuell, Department of Biochemistry, College of Colchester, Honduras, E-mail: AbdoMa@hotmail.com

Received: 03-Mar-2023, Manuscript No: tyoa-23-91164; **Editor assigned:** 06-Mar-2023, Pre-QC No: tyoa-23-91164 (PQ); **Reviewed:** 20-Mar-2023, QC No: tyoa-23-91164; **Revised:** 22-Mar-2023, Manuscript No: tyoa-23-91164 (R); **Published:** 29-Mar-2023, DOI: 10.4172/2476-2067.1000208

Citation: Manuell A (2023) Microrna: A Review of Emerging Experimental and Epidemiological Data as a Hugely Beneficial Technique in Agricultural Toxicity Testing Screening. Toxicol Open Access 9: 208.

Liver failure determines the outcome in patients of acute-on-chronic liver failure (ACLF): comparison of APASL ACLF research consortium (AARC) and CLIF-SOFA models. Hepatology International 11(5): 461-471.

4. Atkins D, Best D, Briss PA (2004) Grading quality of evidence and strength of recommendations. BMJ 328: 1490.
5. O'Grady JG, Schalm SW, Williams R (1993) Acute liver failure: redefining the syndromes. Lancet 342: 273-275.
6. Jalan R, Pavesi M, Wendon J (2015) CANONIC Study Investigators; EASLCLIF Consortium et al. The CLIF Consortium Acute Decompensation score (CLIF-C ADs) for prognosis of hospitalised cirrhotic patients without acute-on-chronic liver failure. J Hepatol 62(4): 831–840.
7. Sen S, William R, Jalan R (2002) The pathophysiological basis of acute-on-chronic liver failure. Liver 22(Suppl 2): 5-13.
8. Wlodzimirow KA, Eslami S, Abu-Hanna A (2013) A systematic review on prognostic indicators of acute on chronic liver failure and their predictive value for mortality. Liver Int 33(1): 40-52.
9. Bajaj JS, Moreau R, Kamath PS, Vargas HE, Arroyo V (2018) Acute-on-chronic liver failure: getting ready for prime time? Hepatology 68(4): 1621-1632.
10. Ferenci P, Lockwood A, Mullen K (2002) Hepatic encephalopathy- definition, nomenclature, diagnosis and quantification: final report of the working party at the 11th World Congress of Gastroenterology, Vienna 1998. Hepatology 25(3): 716-721.