Editorial

IT Medical Team
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International Journal of Drug Development and Research
1791-809X

2022
Vol. 14 No. 6: 962

Vector-Borne Parasites with Drug Resistance and Intercellular Cancer Transfer's Effects on Evolution Extracellular Vesicle-Based Drug Resistance Characteristics

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Citation: Moreira R (2021) Vector-Borne Parasites with Drug Resistance and Intercellular Cancer Transfer's Effects on Evolution Extracellular Vesicle-Based Drug Resistance Characteristics. Int J Drug Dev Res J , Vol.14 No. 6: 962.

Received: 01-Jun-2022, Manuscript No. IJDDR-22-12811; **Editor assigned:** 03-Jun-2022, PreQC No. IJDDR-22-12811; **Reviewed:** 25-Jun-2022, QC No. IJDDR-22-12811 **Revised:** 30-Jun-2022, Manuscript No. IJDDR-22-12811(R); **Published:** 08-Jul-2022, DOI: 10.36648/1791-809X.22.16.962

Keywords: Extracellular Vesicles; Phospholipid Bilayer; Drug Therapy; Antibiotic Resistance; Intratumoral Heterogeneity; Protozoan

Editorial

Drug Resistance

Pathogens that are resistant to medications develop more quickly than new drugs originate from drug research pipelines. Therefore, it is important to better safeguard the treatment options available today and in the future. To do this, it is important to comprehend the factors that lead to the development of drug resistance over time[1]. For the majority of bacteria, many of these parameters are fairly well recognised, but for parasites carried by vectors, the situation is more complicated. We explore how drug resistance can develop, disseminate, and persist using three major models (Plasmodium, Leishmania, and Schistosoma)[2]. We present a variety of scenarios that are clearly a result of the biological diversity of the various organisms, as well as the various routes of action of the drugs used, the unique ecology of the parasites within and between hosts, and environmental factors that could have direct or indirect effects. [3] Given the effects of antibiotic resistance on human health, clinical researchers are very interested in studying this issue. In addition, one of the few evolution instances that can be tracked in real time is antibiotic resistance. Therefore, clinical professionals, evolutionary biologists, and ecologists are interested in understanding the general principles involved in the acquisition of antibiotic resistance. [4] Environmental microorganisms are the source of the genes that human infections now have that make them resistant to antibiotics. The study of both clinical ecosystems and natural habitats is necessary to understand how antibiotic resistance evolves.[5] The founder effect, ecological connection, and fitness costs are significant bottlenecks that control the transfer of resistance from ambient microbes to diseases, according to newly available evidence on the evolutionary mechanisms behind resistance. Extracellular vesicles (EVs) are regarded as crucial mediators of intercellular communication. They are nanoparticles (100–1000 nm) surrounded by a phospholipid bilayer. [6] Numerous studies have been conducted on the function of EVs in oncobiology, including their participation in the horizontal transfer of drug resistance from drug-resistant to drug-sensitive cancer cells. Specifically, drug-efflux pumps, miRNAs, long non-coding RNAs (lncRNAs), and other mediators are the subject of this review's discussion of the EVs cargo that is accountable for this intercellular transmission of drug resistance. [7] Also highlighted are the known chemical properties and mechanisms of this transfer. In order to completely comprehend and prevent the intercellular transfer of drug resistance mediated by EVs, which is a developing area of research, we emphasise areas that still require additional research. The biggest challenge to the effectiveness of cancer chemotherapies is multi-drug resistance (MDR). While the mechanisms of MDR and methods for testing MDR have been found, they remain poorly understood. [8] This review discusses the in vivo and in vitro methods for the laboratory detection of MDR as well as the mechanisms of MDR in malignancies. This study also looks ahead to future advancements in MDR's clinical and therapeutic uses in the treatment of cancer. Future medicines for the treatment of cancer are anticipated to combine current therapies with medications derived from MDR processes, such as anti-cancer stem cell medications, anti-miRNA medications, or anti-epigenetic medications.[9] Finding novel biomarkers and developing new evaluation methods

before drug resistance develops will be difficult for clinical MDR identification. The delivery of cancer curative medicines is still significantly hampered by drug resistance. The overexpression of drug transporters, modifications to drug kinetics, or amplified drug targets have historically been linked to drug resistance. The development of resistance in patients receiving novel targeted therapy has, however, revealed significant details about the intricacies underlying cancer drug resistance. Intratumoral heterogeneity is now recognised as a key factor in treatment resistance, according to recent findings. Human tumours are remarkably varied, as shown by single cell sequencing studies that discovered numerous genetically different variants within them. The four main causes of intratumoral heterogeneity are (i) genetic diversity, (ii) stochastic processes, (iii) the microenvironment, and (iv) cell and tissue plasticity. [10] The sensitivity to drugs is affected by each of these parameters. Drug therapy is essential for the treatment of viral, bacterial, fungal, and protozoan diseases as well as the management of human cancer. The prevalence of resistance is endangering the efficacy of therapy. We investigate and contrast the mechanisms of drug resistance in these various biological systems (using HIV and Plasmodium falciparum as examples of viral and protozoan pathogens, respectively), and we discuss how variables like mutation rates, fitness effects of resistance, epistasis, and clonal interference affect the evolutionary trajectories of drug-resistant clones. We discuss similarities and differences in resistance development that could inform tactics for enhancing treatment efficiency and creating a new class of medications.

Acknowledgement

The author would like to acknowledge his Department of Pharmacogenomics, Center for Applied Medical Research from the University of Navarra for their support during this work.

Conflict of Interest

The author has no known conflicts of interested associated with this paper.

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