Microbes Culled from Patients Enabling Therapeutic Precision Oncology

Domenico Coppola*

Professor of Cancer Research, Department of biotechnologist, US

*Corresponding Author**

Domenico Coppola
Professor of Cancer Research, Department of biotechnologist, United States
E-mail: Coppola@gmail.com

Received: 15 Nov, 2022, Manuscript No. OCCRS-22-79840; **Editor assigned**: 16 Nov, 2022, **PreQC No** OCCRS-22-79840 (PQ); **Reviewed**: 18 Nov 2022, QC No. OCCRS-22-79840 (Q); **Revised**: 21 Nov 2022, Manuscript No. OCCRS-22-79840(R); **Published**: 30 Jan 2023, doi.10.37532/23.9.1.1

Abstract

It has been demonstrated that Patient-Derived Organoids (PDOs) and Patient-Derived Xenografts (PDXs) can simulate the clinical response to cancer treatment. Utilizing these models to help prompt clinical decisions for cancer patients is still difficult. Here, we combined temperature control, dead-volume minimization, and droplet emulsion microfluidics to quickly produce thousands of micro-organ spheres (MOSs) from low-volume patient tissues, which serve as the perfect patient-derived model for clinical precision oncology. Using a MOS-based precision oncology pipeline, a clinical investigation of patients with recently diagnosed advanced Colorectal Cancer (CRC) accurately assessed tumor medication response within 14 days, a time frame adequate for directing treatment decisions in the clinic. Additionally, MOSs isolate the original stromal cells and permit T cell penetration offers a clinical assay enabling PD-1 blockade, bispecific antibodies, and other Immuno-Oncology (IO) treatments to be tested on patient malignancies

Keywords Micro-organ sphere precision oncology • Colorectal cancer • Lung cancer • Droplet microfluidics • Immune-oncology • Bispecific antibody • Adoptive cell therapy • Precision medicine • Tumor sphere

Introduction

Pathogenic Variants (PV) inherited in the germline are thought to be responsible for 5%–10% of all cancers. Early detection of a cancer-predisposing germline PV is essential for high-risk families because genetic counselling that follows can help patients stick with risk-reducing measures. Germline PVs in DNA repair genes are known to not only raise cancer risk but are also important for determining how to treat cancer. There are numerous specialized pathways in cells that can repair various kinds of DNA lesions, and DNA repair is essential for genome stability. These pathways include Homologous Recombination Repair (HRR), non-Homologous End-Joining (NHEJ), Franconia Anemia (FA), Micro Homology-Mediated End Joining (MMEJ), Nucleotide Excision Repair (NER), Base Excision Repair (BER), Mismatch Repair (MMR), and replication repair. a concise explanation of the cancer risk and approved treatment biomarkers or medications. DNA damage can result from a variety of endogenous (metabolites, replication errors) or exogenous (irradiation, UV light, chemotherapy agents) sources. Replication errors, single-stranded breaks, and double-stranded breaks are just a few examples of the various types of DNA damage that can trigger the DNA damage response signaling and the checkpoint response. The sensory kinases ATM and ATR are activated as part of the signaling for the DNA damage response. The checkpoint kinases Chk1 and Chk2 amplify the signals from these sensory kinases, causing cells to arrest their cell cycle in a p53-dependent manner in order to either repair the damage or go through with apoptosis to kill the cells. Specialized DNA repair pathways repair various forms of DNA damage; the pathway members that are linked to an increased risk of cancer and/or are currently being examined by germline cancer gene panels are listed below. These specialized repair pathways are BER for single strand breaks, NER for large DNA adducts, MMR and replication-repair for base-base mismatches, FA, MMEJ, and NHEJ for double-strand breaks. Particularly sensitive to DNA-damaging treatments, such as radiation therapy and platinum-based chemotherapy, are tumors with defects in DNA repair.

Conclusions

The widespread application of next-generation sequencing technologies in clinical settings has uncovered a wide range of germline abnormalities in DNA repair genes. The clinical options for cancer patients have increased thanks to the availability of FDA-approved treatments that specifically target DNA repair defects, such as PARPis or ICIs that are beneficial in patients with DNA repair defects and have long-lasting effects. In light of the importance of DNA repair abnormalities for cancer risk and treatment response, it is crucial to ascertain in the future whether hereditary vulnerabilities in DNA repair pathway mediators might be used therapeutically. All things considered, the time is right to fully comprehend the therapeutic scope of germline vulnerabilities in DNA repair and evaluate their clinical use in cancer patients.

Cite this article: Coppola, D. Microbes Culled from Patients Enabling Therapeutic Precision Oncology. Oncol. Cancer Case Rep, 2023, 9(1), 1