Editorial Note

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CNAs (Circulating Nucleic Acids) in a New Light

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Editorial

In 1948, Circulating Nucleic Acids (CNAs) were found in the plasma of healthy and diseased people. This discovery, however, did not pique the scientific community's curiosity at the time. The discovery of significant quantities of circulating DNA in individuals with systemic lupus erythematous was only made in the 1960s, when study in this field was restarted.

CNAs are segments of genomic, mitochondrial, or viral DNA, RNA, and microRNA (miRNA) discovered in the circulation, according to the definition. Apoptosis and necrosis, as well as the spontaneous release of nucleic acids from cells, are thought to be methods by which CNAs are released into circulation. Cell-free CNAs, on the other hand, are primarily caused by apoptosis or necrosis. CNAs are also thought to be released in the bloodstream by Circulating Tumour Cells (CTCs). CNAs derived from viruses like as EBV, HPV, and hepatitis B virus have been detected in healthy people's plasma and serum, as well as in cases of malignancies linked to viral infections.

CNAs are found not just in animals, but also in plants and bacteria. CNAs are fragmented DNA and chromatin with a size range of 100 bp-1000 bp, a half-life of 10-15 minutes, and are eventually eliminated by the liver. Several research have compiled data suggesting the diagnostic value of these CNAs, notably in cancer screening and monitoring the success of anticancer therapies, throughout the last decade.

Until date, a lot of study has been done to look into the diagnostic potential of CNAs. However, there are few studies that look at the pathophysiological activities of CNAs. In 1999, some fascinating results suggested that circulating DNA may be taken up by cells, resulting in visible gene expression. CNAs (DNA and chromatin fragments) isolated from the blood of healthy volunteers and cancer patients have recently been shown to be avidly taken up by cells in culture, resulting to fast accumulation in cell nuclei and eventual interaction with cell chromosomes.

These intracellular CNAs activated DDR and apoptotic pathway proteins, triggering a DNA-Damage-Repair-response (DDR) that up-regulated several DNA damage and repair pathways, allowing them to integrate into the host cell genome. This is the first concrete proof that CNAs can behave like mobile genetic elements in vitro and in vivo, changing the expression levels of particular genes in otherwise healthy cells, promoting invasion and metastasis. This new theory could provide an intriguing mechanism for age-related mutation. CNAs may be a novel, endogenous source of genomic instability, contributing to greater genome mosaicism as people get older. There is evidence that CNAs grow more prevalent in the circulation as old and injured cells become more vulnerable to cell death. As a result, activation of the DDR could greatly increase genomic instability, thereby leading to age-related degenerative illnesses like cancer and inflammation.

CNAs' cellular entrance and acquisition of biological features have recently been revealed to be dependent on their size. Small pieces of DNA (300 bp-3000 bp) from a variety of sources, such as malignant and non-cancerous human cells, bacteria, and plants, can not only infiltrate other cells indiscriminately across species and kingdom boundaries, but also integrate into their genomes and activate biological processes. As a result, fragmented DNA produced after cell death may operate as mobile genetic elements and may have evolutionary implications as a result of their involvement in horizontal gene transfer.

DNase I and II, two enzymes found in circulation, are known to destroy DNA. However, when their inhibitors are produced, such as in malignant disorders, reduced activity of DNase I and II have been discovered, explaining why higher DNA levels in circulation are reported. Because CNAs released from dying cells can enter healthy cells' genomes and cause DNA damage, apoptosis, and inflammation, it's possible that a large part of the toxicity of chemotherapy is due to the release of large amounts of CNAs from dying cells, as well as toxicity caused by the drugs themselves. Concurrent treatment with CNAs neutralising or degrading agents, such as anti-histone antibody complexed nanoparticles, DNase I, and Resveratrol-Cu, could decrease chemotherapy toxicity caused by CNAs produced from dying cells. Many naturally occurring DNA cleaving agents can also be investigated for use in preventing chemotherapy-induced harm. The potential for CNAs to operate as mobile genetic elements,

as well as their subsequent role in genomic instability and evolutionary processes, will open up new avenues for medication development.

Chemotherapy-derived CNAs from dying cells may promote genomic instability in cancer patients. CNAs neutralising or degrading agents taken concurrently with chemotherapy may be able to prevent these chemotherapy adverse effects. If this strategy is successfully translated, it may be possible to prevent chemotherapy-related deaths. As a result, the confirmed function of CNAs in genomic instability and evolutionary processes could reveal a new treatment method for age-related degenerative disorders.