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Commentary

Molecular Disruptions Shaping the Course of Ovarian Cancer: Genetic and Epigenetic Contributions to Progression and Recurrence

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DESCRIPTION

Ovarian cancer continues to be a significant health concern for women globally, with high mortality rates primarily due to late-stage diagnosis and frequent recurrence after initial therapy. Despite surgical interventions and advances in chemotherapy, long-term disease control remains difficult. To better manage this condition, researchers have focused on the molecular alterations that contribute to its development, progression, and return after treatment. Both genetic mutations and epigenetic shifts play a vital role in modifying cellular behaviour in ovarian tissue, leading to malignancy and resistance to therapy.

Among the most well-known genetic changes linked with ovarian cancer are mutations in the *BRCA1* and *BRCA2* genes. These genes are involved in repairing damaged DNA, particularly through homologous recombination. When their function is lost or compromised due to inherited or acquired mutations, cells accumulate errors in their DNA, which can initiate malignant growth. Women with these mutations face a significantly increased lifetime risk of developing ovarian cancer, especially the high-grade serous subtype. However, these same changes also influence response to certain treatments, particularly those that exploit weaknesses in DNA repair pathways.

Beyond *BRCA* genes, alterations in other DNA repair genes such as *RAD51C, RAD51D,* and *PALB2* have also been found in a subset of ovarian tumors. These changes further highlight how disruptions in genomic stability contribute to the disease. Additionally, mutations in *TP53* are nearly universal in high-grade serous ovarian carcinomas, affecting cell cycle regulation and contributing to unchecked proliferation.

Yet, not all changes driving ovarian cancer progression are found in the DNA sequence itself. Epigenetic modifications, which alter gene activity without changing the underlying genetic code, are also deeply involved. These include DNA methylation, histone modification, and changes in non-coding RNA expression. In many cases, tumor suppressor genes are turned off due to hyper methylation of their promoter regions, effectively silencing their protective functions. This has been observed with genes such as *MLH1, RASSF1A*, and *PTEN*.

Conversely, hypomethylation of other genomic regions can lead to the activation of genes that encourage tumor growth and invasion. Such patterns can lead to genomic instability and increase the likelihood of recurrence, especially after initial treatment has induced partial tumor regression. Histone modifications also play a part by influencing how tightly DNA is wrapped around histone proteins, thereby affecting gene accessibility. Changes in enzymes that modify histones have been found in ovarian cancer cells, leading to altered expression of genes involved in proliferation, cell death, and invasion.

Non-coding RNAs, particularly MicroRNAs (miRNAs) and Long Non-Coding RNAs (lncRNAs), are increasingly being studied for their roles in ovarian cancer biology. These molecules can regulate the expression of multiple genes by interacting with messenger RNA or modifying chromatin structure. For instance, the loss of tumor-suppressive miRNAs or an increase in oncogenic miRNAs can tilt the balance toward malignancy. Some lncRNAs have been found to interfere with normal regulatory processes, creating an environment more conducive to disease persistence and spread.

One of the most difficult challenges in managing ovarian cancer is the tendency of the disease to return, often in a more aggressive and treatment-resistant form. Molecular studies suggest that recurrence is often driven by residual cancer cells that survive initial therapy, aided by either genetic mutation that confer drug resistance or epigenetic mechanisms that allow for adaptation. These surviving cells can remain dormant or minimally active before re-emerging, sometimes with altered molecular characteristics that make previous therapies less effective.

Another contributing factor to recurrence is tumor heterogeneity. Within a single patient, different areas of the tumor may carry distinct molecular signatures. Some of these

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subpopulations may be more resistant to treatment, allowing them to persist and eventually dominate the tumor landscape after the more sensitive cells have been eliminated. This diversity complicates treatment planning and highlights the need for repeat molecular testing over the course of disease.

Overall, both inherited and acquired genetic alterations, along with reversible epigenetic changes, shape the trajectory of ovarian cancer. These molecular events not only initiate tumor development but also contribute to recurrence and therapy resistance. As knowledge continues to expand, a more complete molecular understanding will be essential for improving detection, refining treatments, and managing disease recurrence in individuals facing this formidable illness.