ISSN- O: 2458 - 868X, ISSN–P: 2458 – 8687
National Library of Medicine - ID: 101731606

International Journal of Medical Science and Innovative Research (IJMSIR)
IJMSIR : A Medical Publication Hub
Available Online at: www.ijmsir.com
Volume – 11, Issue – 3, June – 2026, Page No. : 62 – 72

Symphony of Illusions - Organelle Dysfunction in Cancer: A Review

[1]Dr. Pramit Sen, Post Graduate Trainee, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata – 700114

[2]Prof. (Dr.) Mousumi Pal, HOD and Professor, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata –700114

[3]Prof. (Dr.) Sk. Abdul Mahmud, Professor, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata –700114

[4]Dr. Sanjeet Kumar Das, Associate Professor, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata –700114

Corresponding Author: Dr. Pramit Sen, Post Graduate Trainee, Department of Oral and Maxillofacial Pathology, Guru Nanak Institute of Dental Sciences and Research, Panihati, Kolkata – 700114

Citation this Article: Dr. Pramit Sen, Prof. (Dr.) Mousumi Pal, Prof. (Dr.) Sk. Abdul Mahmud, Dr. Sanjeet Kumar Das, "Symphony of Illusions - Organelle Dysfunction in Cancer: A Review", IJMSIR – June – 2026, Vol – 11, Issue – 3, P. No. 62 – 72.

Type of Publication: Review Article

Conflicts of Interest: Nil

Abstract

Cancer development involves complex molecular and cellular alterations that extend beyond genetic mutations. Increasing evidence suggests that intracellular organelles play crucial roles in tumour initiation, progression, and metastasis. These subcellular structures form an interconnected network responsible for protein synthesis, metabolic regulation, intracellular signalling, and genomic integrity. In malignant cells, organelles undergo structural reorganization and functional disruption that support the hallmarks of cancer. Endoplasmic reticulum stress responses enable tumour cells to survive in metabolically hostile environments. Alterations in Golgi apparatus organization influence vesicular trafficking, glycosylation patterns, and cellular polarity, thereby promoting metastatic behaviour. Mitochondrial dysfunction drives metabolic reprogramming, reactive oxygen species production, and resistance to apoptosis. Dysregulated ribosome biogenesis enhances translational capacity and contributes to oncogene activation. Nuclear abnormalities including chromatin remodelling, nuclear envelope alterations, and DNA ploidy changes further facilitate malignant transformation. Understanding the role of organelle dysfunction provides valuable insight into cancer biology and highlights novel targets for therapeutic intervention. This review discusses the molecular mechanisms through which intracellular organelles contribute to tumour development and emphasizes their potential relevance in cancer diagnosis and treatment.

Keywords: Cancer Biology, Organelle Dysfunction, ER Stress, Mitochondrial Metabolism, Reactive Oxygen

Species, Ribosome Biogenesis, Golgi Trafficking, Nuclear Dysfunction, Unfolded Protein Response.

Introduction

Multicellular organisms maintain physiological balance through coordinated communication among specialized cells. This coordination relies on tightly regulated cellular processes such as proliferation, differentiation, metabolic activity, and programmed cell death. Any disruption in these regulatory mechanisms can lead to pathological conditions, including cancer [1].

Cancer is a multifactorial disease characterized by uncontrolled cellular proliferation, resistance to apoptosis, altered metabolism, and the ability to invade surrounding tissues. Traditionally, cancer research focused primarily on genetic mutations and dysregulated signalling pathways. However, emerging evidence indicates that subcellular organelles play a pivotal role in shaping the malignant phenotype [1].

Organelles are not isolated structures; instead, they form an integrated network that coordinates cellular homeostasis. In cancer cells, this network undergoes profound reorganization to meet the metabolic and biosynthetic demands of rapid cell division. Structural changes, metabolic adaptations, and altered communication between organelles allow tumour cells to thrive in conditions that would normally be incompatible with cell survival. [2]

Recent studies demonstrate that organelle dysfunction contributes directly to many fundamental characteristics of cancer, including metabolic reprogramming, resistance to stress, immune evasion, and metastatic potential. Understanding these alterations is essential for deciphering the complex biology of tumour progression.[2]

This review explores the role of major intracellular organelles—including the Endoplasmic Reticulum, Golgi Apparatus, Mitochondria, Ribosomes, and Nucleus in cancer development. Emphasis is placed on the molecular mechanisms through which these organelles influence oncogenic transformation and tumour progression.

Endoplasmic Reticulum Dysfunction in Cancer

- **Structure and Functional Role**

The Endoplasmic Reticulum (ER) is an extensive membranous organelle that forms a continuous network within the cytoplasm. It exists in two structural forms: The Rough Endoplasmic Reticulum (RER), which contains ribosomes attached to its surface, and the Smooth Endoplasmic Reticulum (SER), which lacks ribosomes [34]. The RER is primarily responsible for the synthesis of secretory proteins, membrane proteins, and lysosomal enzymes. Newly synthesized polypeptides undergo folding and post-translational modifications within the ER lumen. The SER participates in lipid biosynthesis, steroid metabolism, detoxification reactions, and intracellular calcium storage [34].

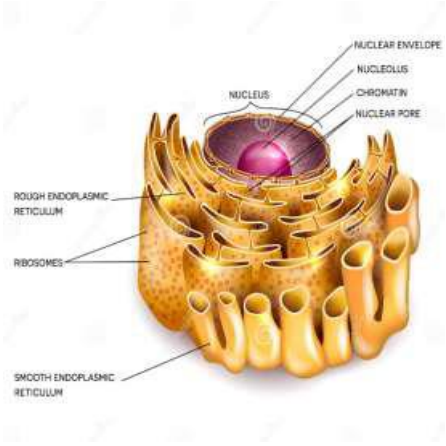

Figure 1: Diagram showing the structural relationship between the Nucleus and Endoplasmic Reticulum, including Rough ER with ribosomes, Smooth ER, nuclear envelope, nucleolus, chromatin, and nuclear pores involved in protein synthesis and intracellular regulation.

By regulating protein folding and intracellular signalling pathways, the ER acts as a central hub for maintaining cellular homeostasis.

- **ER Stress and Activation of the Unfolded Protein Response**

Cancer cells frequently experience conditions that disturb normal ER function. Environmental stressors such as hypoxia, nutrient deprivation, oxidative stress, and acidic microenvironments lead to accumulation of unfolded or misfolded proteins within the ER lumen.

This condition is referred to as Endoplasmic Reticulum Stress (ER stress) [1]. To restore equilibrium, cells activate a complex signalling network known as the Unfolded Protein Response (UPR). The UPR functions through three principal sensors: PERK, IRE1, and ATF6. Activation of these sensors leads to multiple adaptive responses, including suppression of global protein synthesis, increased production of molecular chaperones, and enhanced degradation of misfolded proteins [2,3].

These mechanisms collectively increase the folding capacity of the ER and allow cells to adapt to stressful conditions.

Endoplasmic reticulum (ER): a dynamic tubular network involved in metabolic processes including gluconeogenesis, lipid synthesis, and the biogenesis of autophagosomes and peroxisomes. It is also the major intracellular calcium reservoir.
ER stress: a cellular condition generated when misfolded proteins accumulate inside the ER.
ER-associated degradation (ERAD): a pathway to eliminate misfolded proteins along which proteins are transported from the ER to the cytosol for further degradation by the proteasome.
Proteostasis: a portmanteau of the words protein and homeostasis. Refers to the concept of integrated biological pathways within cells that control the biogenesis, folding, trafficking, and degradation of intracellular and extracellular proteins.
Proteotoxicity: pernicious condition generated by an exacerbated increase of proteins or the presence of misfolded proteins.
Regulated IRE1-dependent decay (RIDD): the degradation of a subset of mRNAs encoding proteins located in the ER and microRNAs through the activation of the endoRNase domain of IRE1.
Unfolded protein response (UPR): a series of adaptive mechanisms triggered by ER stress to cope with protein-folding alterations through the transcriptional regulation of proteins involved in folding and clearance to restore ER proteostasis.

Figure 2: Key concepts related to endoplasmic reticulum (ER) stress and proteostasis, including ER-associated degradation (ERAD), unfolded protein nresponse (UPR), regulated IRE1- dependent decay (RIDD), and mechanisms involved in maintaining intracellular protein homeostasis.

- **Contribution to Tumour Survival**

The tumour microenvironment is often characterized by hypoxia and limited nutrient availability. Under such conditions, activation of the UPR becomes critical for tumour cell survival. Persistent ER stress signalling promotes angiogenesis through upregulation of Vascular Endothelial Growth Factor (VEGF) and other pro-angiogenic mediators [4].

Additionally, ER stress facilitates Epithelial-Mesenchymal Transition (EMT), a biological process through which epithelial cells lose their polarity and acquire migratory characteristics.

EMT plays a key role in tumour invasion and metastasis[5]. An emerging concept known as transmissible ER stress suggests that tumour cells can transmit ER stress signals to surrounding immune cells, thereby suppressing antitumor immune responses and enhancing tumour survival [6].

Golgi Apparatus Alterations in Cancer

- **Structural Organization and Normal Functions**

The Golgi apparatus consists of a series of flattened membranous sacs known as cisternae. Positioned between the endoplasmic reticulum and the plasma membrane, the Golgi complex performs essential roles in protein modification, sorting, and intracellular transport [35].

Proteins synthesized in the ER are transported to the Golgi apparatus, where they undergo glycosylation, sulfation, and proteolytic processing. The Golgi also determines the final destination of proteins by packaging

them into vesicles targeted for secretion or delivery to specific intracellular compartments [35].

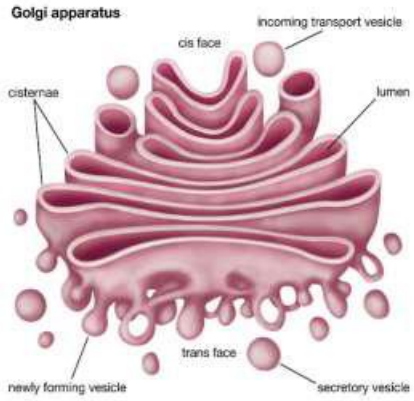

Figure 3: Structural organization of the Golgi apparatus showing stacked cisternae with the cis face (receiving side), trans face (shipping side), transport vesicles, and secretory vesicles involved in protein modification, sorting, and intracellular trafficking.

- **Dysregulation of Vesicular Transport**

Cancer cells frequently manipulate vesicular transport pathways to support tumour progression.

Enhanced secretion of enzymes such as matrix metalloproteinases contributes to degradation of extracellular matrix components, thereby facilitating tumour invasion [7].

Members of the Rab and ARF families of small GTPases regulate vesicular trafficking within the Golgi network. Mutations or overexpression of these proteins have been detected in multiple malignancies.

For instance, Rab40b has been shown to regulate the trafficking of MMP-2 and MMP-9 during invadopodia formation in breast cancer cells [8].

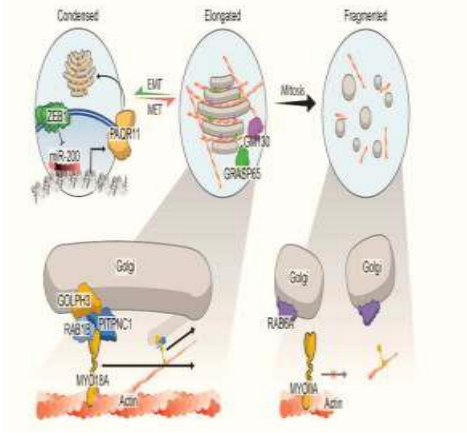

Figure 4: Dynamic structural changes of the Golgi apparatus during epithelial–mesenchymal transition (EMT) and mitosis, illustrating Golgi condensation, elongation, and fragmentation, along with associated molecular regulators that influence vesicular trafficking and cell migration.

- **Golgi Reorientation and Metastasis**

Cell migration requires reorganization of intracellular trafficking pathways. During epithelial mesenchymal transition, the Golgi apparatus undergoes a spatial reorientation that directs secretory vesicles toward the leading edge of migrating tumour cells [9].

This reorientation enhances directional secretion of proteins involved in cell adhesion, extracellular matrix degradation, and cell motility. The process significantly contributes to metastatic dissemination [9].

Alterations in glycosylation patterns of cell surface proteins further influence tumour cell interactions with vascular and lymphatic endothelium, thereby promoting metastatic spread[11].

Mitochondrial Dysfunction and Cancer Metabolism

- **Role of Mitochondria in Cellular Physiology**

Mitochondria are double-membrane organelles responsible for ATP production through oxidative phosphorylation. Beyond their role in energy metabolism, mitochondria participate in biosynthetic

pathways, calcium signalling, and regulation of programmed cell death [36].

The mitochondrial electron transport chain generates the majority of cellular ATP and also produces reactive oxygen species as metabolic byproducts[36].

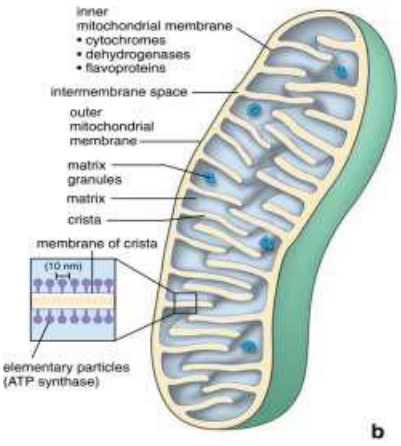

Figure 5: Ultrastructural organization of the mitochondrion showing the outer membrane, inner membrane with cristae, intermembrane space, and matrix containing enzymes involved in oxidative phosphorylation and ATP synthesis.

- **Metabolic Reprogramming in Tumor Cells**

One of the defining features of cancer is metabolic reprogramming. Tumour cells modify their metabolic pathways to support rapid proliferation and survival under stressful conditions. Alterations in mitochondrial oxidative phosphorylation, fatty acid metabolism, and glutamine utilization have been reported in various malignancies [13].

Mutations affecting mitochondrial DNA and nuclear genes encoding components of the tricarboxylic acid cycle further contribute to these metabolic adaptations [14].

- **Mitochondrial DNA Alterations**

Mitochondrial DNA (mtDNA) is particularly susceptible to damage due to its proximity to the electron transport chain and limited repair mechanisms. Somatic mutations and variations in mtDNA copy number have been identified in several cancers [16].

Reduced mtDNA content can influence tumour cell differentiation and proliferation, while certain mtDNA mutations are associated with aggressive tumour behaviour and poor prognosis [16].

- **Reactive Oxygen Species and Tumour Progression**

Mitochondrial dysfunction often leads to increased production of Reactive Oxygen Species (ROS). Although excessive ROS can induce cell death, moderate levels function as signalling molecules that promote tumour growth [20]. ROS activate signalling pathways involved in cell proliferation, migration, and angiogenesis. They also contribute to genomic instability and facilitate immune evasion mechanisms within the tumour microenvironment [21,22].

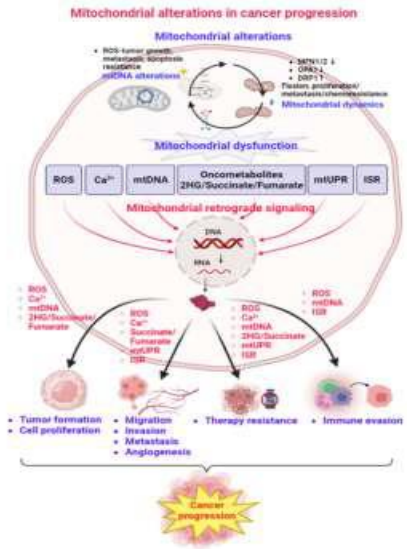

Figure 6: Schematic representation of mitochondrial alterations in cancer progression, highlighting mitochondrial dysfunction, reactive oxygen species (ROS) production, mtDNA alterations, and retrograde signalling pathways that contribute to tumour growth, metastasis, therapy resistance, and immune evasion.

Ribosome Biogenesis and Cancer

- **Ribosomal Structure and Function**

Ribosomes are ribonucleoprotein complexes responsible for translating messenger RNA (mRNA) into functional

proteins. They consist of a large and a small subunit composed of ribosomal RNA (rRNA) and ribosomal proteins [37].

Protein synthesis is essential for cellular growth and proliferation. Therefore, precise regulation of ribosome biogenesis and translational activity is critical for maintaining normal cellular function [37].

- **Translational Control in Tumour Development**

Cancer cells require enhanced protein synthesis to sustain rapid proliferation. Dysregulation of ribosome biogenesis and translational control mechanisms has therefore emerged as an important aspect of tumour biology [23].

Oncogenes such as MYC stimulate transcription of ribosomal RNA and ribosomal protein genes, leading to increased ribosome production. This process significantly enhances the translational capacity of cancer cells [24].

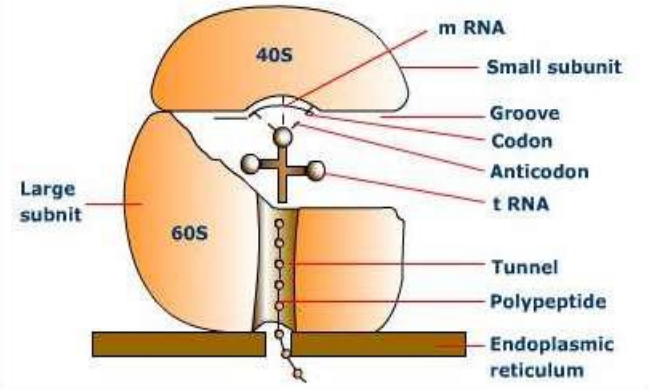

Figure 7: Structural diagram of the ribosome showing the large (60S) and small (40S) subunits, mRNA binding site, tRNA interaction, and the nascent polypeptide chain emerging during protein synthesis.

- **Ribosomal Protein Mutations and Tumorigenesis**

Mutations or altered expression of ribosomal proteins can disrupt ribosomal assembly and trigger nucleolar stress responses. These changes influence tumour suppressor pathways, particularly the p53 signalling axis [25,26].

Several ribosomal proteins have been implicated in specific malignancies. For example, ribosomal protein S2 is overexpressed in hepatocellular carcinoma, while multiple ribosomal proteins are upregulated in colorectal cancer [28,29].

These findings highlight the role of ribosomes not only as protein synthesis machinery but also as regulators of oncogenic signalling pathways.

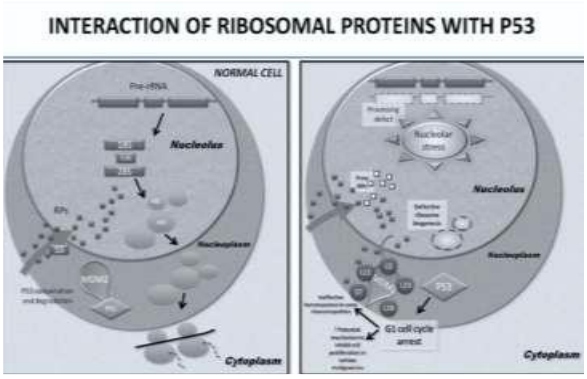

Figure 8: Illustration showing the interaction between ribosomal proteins and the p53 pathway, where defective ribosome biogenesis induces nucleolar stress, leading to stabilization of p53 and subsequent cell cycle arrest.

Nuclear Alterations In Cancer

- **Structural Changes in Malignant Nuclei**

The nucleus serves as the central repository of genetic material and regulates transcriptional activity. Morphological abnormalities of the nucleus are among the most prominent histological features of malignant cells[32]. These abnormalities include nuclear pleomorphism, irregular nuclear membranes, enlarged nucleoli, and abnormal chromatin distribution [32].

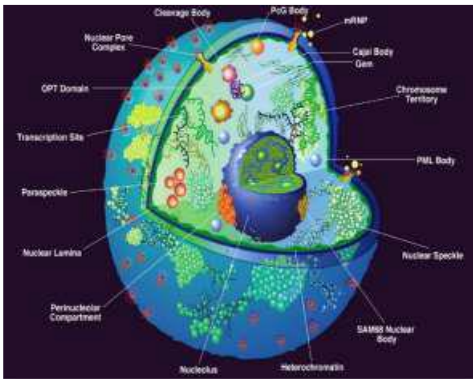

Figure 9: Detailed schematic of the cell nucleus illustrating nuclear substructures including the nucleolus, nuclear lamina, nuclear pore complex, chromatin

organization, and various nuclear bodies involved in gene regulation and RNA processing.

- **Nuclear Lamina Alterations**

The nuclear lamina provides structural support to the nuclear envelope and plays a critical role in chromatin organization. Alterations in lamina proteins have been observed in several malignancies, including lung and colorectal cancers [32].

These alterations may influence gene expression by modifying the spatial organization of chromatin within the nucleus [33].

- **Chromatin Remodelling and Epigenetic Regulation**

Chromatin remodelling represents a key mechanism through which cancer cells alter gene expression patterns. Remodelling can occur through ATP-dependent repositioning of nucleosomes or through covalent modifications of histone proteins [32].

Histone acetylation, methylation, and phosphorylation regulate the accessibility of DNA to transcriptional machinery. Dysregulation of these processes contributes to oncogene activation and tumor suppressor gene silencing [33].

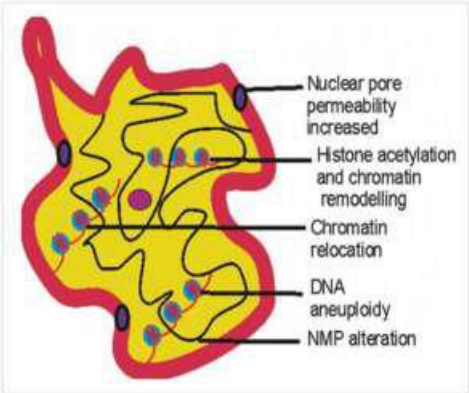

Figure 10: Diagram illustrating nuclear alterations in malignant cells, including increased nuclear pore permeability, chromatin remodeling, chromatin relocation, DNA aneuploidy, and nuclear membrane protein alterations associated with tumor progression

- **DNA Ploidy and Nuclear Pore Alterations**

Changes in DNA content, referred to as aneuploidy, are frequently observed in malignant cells. Abnormal DNA ploidy reflects genomic instability and correlates with tumour aggressiveness. Increased permeability of nuclear pores may also alter nucleocytoplasmic transport of regulatory molecules, thereby influencing cellular proliferation and survival [32].

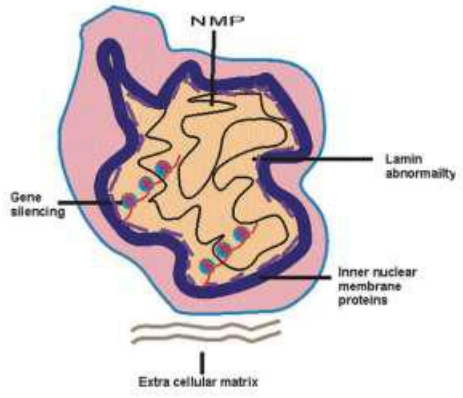

Figure 11: Illustration showing nuclear lamina abnormalities in cancer, including alterations in nuclear membrane proteins (NMP), lamin defects, and associated gene silencing mechanisms that influence chromatin organization and tumour progression.

Inter-Organelle Communication in Cancer

Modern cell biology recognizes that organelles operate as components of a highly interconnected network rather than isolated units. Physical contact sites between organelles allow exchange of metabolites, lipids, and signalling molecules [32].

For example, interactions between the endoplasmic reticulum and mitochondria regulate calcium signalling and apoptosis. Similarly, communication between the Golgi apparatus and cytoskeleton controls directional cell migration [32].

Disruption of these inter-organelle interactions can profoundly influence tumour cell behaviour and contribute to several hallmarks of cancer [33].

Therapeutic Implications

Targeting organelle dysfunction offers promising opportunities for the development of novel anticancer therapies. Pharmacological agents that modulate ER stress pathways, inhibit mitochondrial metabolism, or disrupt vesicular trafficking are currently being investigated.

Similarly, inhibitors of ribosome biogenesis and epigenetic regulators affecting nuclear chromatin organization represent emerging therapeutic strategies [2].

Understanding organelle-specific vulnerabilities may allow selective targeting of tumour cells while minimizing damage to normal tissues.

Table 1: Major intracellular organelles involved in cancer progression, summarizing their key molecular alterations, and associated oncogenic consequences.

ORGANELLE	MAJOR DYSFUNCTIONS IN CANCER	KEY MOLECULAR MEDIATORS	ONCOGENIC CONSEQUENCES
Endoplasmic Reticulum (1-6)	Accumulation of misfolded proteins leading to ER stress and activation of the unfolded protein response (UPR); altered proteostasis	PERK, IRE1, ATF6, GRP78/BiP, CHOP	Tumor survival under hypoxia, increased angiogenesis, epithelial–mesenchymal transition (EMT), immune suppression
Golgi Apparatus (7-11)	Dysregulated vesicular trafficking, abnormal glycosylation, Golgi fragmentation and reorientation during EMT	Rab GTPases (Rab40b, Rab27b), ARF proteins, PAQR11	Increased tumour invasion, enhanced metastasis, extracellular matrix degradation, altered cell polarity
Mitochondria (13-22)	mtDNA mutations, altered mitochondrial metabolism, reduced mtDNA copy number, increased ROS production	Cytochrome c, SMAC/DIABLO, TFAM, mitochondrial respiratory chain enzymes	Metabolic reprogramming, resistance to apoptosis, increased metastatic potential, immune evasion
Ribosomes (23-31)	Dysregulated ribosome biogenesis, altered translational control, mutations or abnormal expression of ribosomal proteins	MYC oncogene, ribosomal proteins (RPS2, RPS6, RPL19), MDM2–p53 pathway	Enhanced protein synthesis, tumour cell proliferation, nucleolar stress, altered tumour suppressor regulation
Nucleus (9,26)	Nuclear membrane irregularities, chromatin remodelling, DNA aneuploidy, altered nuclear pore permeability	Lamin proteins, histone modifiers, nuclear membrane proteins (NMPs), nuclear pore complexes	Genomic instability, abnormal gene expression, uncontrolled cellular proliferation
Inter-organelle communication (3,22)	Disruption of ER–mitochondria contact sites and altered calcium signalling	MAM proteins, ROS signalling pathways	Metabolic adaptation, tumour survival, therapy resistance

Conclusion

Intracellular organelles are fundamental regulators of cellular physiology and play crucial roles in maintaining homeostasis. In cancer cells, these organelles undergo extensive structural and functional alterations that collectively support malignant transformation.

Dysfunction of the Endoplasmic Reticulum, Golgi Apparatus, Mitochondria, Ribosomes, and Nucleus contributes to metabolic adaptation, resistance to apoptosis, genomic instability, and metastatic potential. Increasing evidence suggests that interactions between these organelles form an integrated network that drives tumour progression.

Advancing our understanding of the molecular mechanisms underlying organelle dysfunction represents a critical frontier in modern cancer research. Deciphering these subcellular alterations not only deepens our insight into the intricate biology of tumour initiation and progression but also opens new avenues for the discovery of biomarkers enabling early diagnosis, the development of highly specific targeted therapies, and ultimately the achievement of improved patient prognosis.

In the era of precision medicine, the recognition of organelle-level alterations can no longer remain a purely theoretical or academic pursuit. Rather, it must be integrated into routine pathological practice as an essential component of modern diagnostic and therapeutic decision making.

References

1. Ruggero D. Translational control in cancer etiology. Cold Spring Harbor Perspectives in Biology. 2013 Feb 1;5(2):a012336.
2. Dejeans N, et al. Addicted to secrete—novel concepts and targets in cancer therapy. Trends in Molecular Medicine. 2014 May 1;20(5):242–250.
3. Hetz C. The unfolded protein response: controlling cell fate decisions under ER stress and beyond. Nature Reviews Molecular Cell Biology. 2012 Feb 1;13(2):89–102.
4. Bi M, et al. ER stress-regulated translation increases tolerance to extreme hypoxia and promotes tumor growth. The EMBO Journal. 2005 Oct 5;24(19):3470–3481.
5. Blais JD, et al. PERK-dependent translational regulation promotes tumor cell adaptation and angiogenesis in response to hypoxic stress. Molecular and Cellular Biology. 2006 Dec 1;26(24):9517–9532.
6. Clarke HJ, et al. Endoplasmic reticulum stress in malignancy. Cancer Cell. 2014 May 12;25(5):563–573.
7. Goldenring JR. A central role for vesicle trafficking in epithelial neoplasia: intracellular highways to carcinogenesis. Nature Reviews Cancer. 2013 Dec 1;13(12):813–820.
8. Jacob A, et al. Rab40b regulates trafficking of MMP2 and MMP9 during invadopodia formation and invasion of breast cancer cells. Journal of Cell Science. 2013 Oct 15;126(20):4647–4658.
9. Hanahan D, Weinberg RA. Hallmarks of cancer: the next generation. Cell. 2011 Mar 4;144(5):646–674.
10. Tan X, et al. Epithelial-to-mesenchymal transition drives a pro-metastatic Golgi compaction process through scaffolding protein PAQR11. The Journal of Clinical Investigation. 2017 Jan 3;127(1):117–131.
11. Lu HH, et al. Fucosyltransferase 4 shapes oncogenic glycoproteome to drive metastasis of lung adenocarcinoma. EBioMedicine. 2020 Jul 1;57:102846.
12. Stevenson NL, et al. Giantin-knockout models reveal a feedback loop between Golgi function and glycosyltransferase expression. Journal of Cell Science. 2017 Nov 15;130(22):4132–4143.
13. Hirst J. Mitochondrial complex I. Annual Review of Biochemistry. 2013 Jun 2;82:551–575.
14. Mandavilli BS, Santos JH, Van Houten B. Mitochondrial DNA repair and aging. Mutation Research. 2002 Jun 30;509(1–2):127–151.
15. Nouws J, et al. Assembly factors as a new class of disease genes for mitochondrial complex I deficiency: cause, pathology and treatment options. Brain. 2012 Jan 1;135(1):12– 22.
16. Lee HC, et al. Somatic alterations in mitochondrial DNA and mitochondrial dysfunction in gastric cancer

progression. World Journal of Gastroenterology. 2014 Apr 28;20(14):3950–3959.

17. Mannick JB, et al. S-nitrosylation of mitochondrial caspases. Journal of Cell Biology. 2001 Sep 17;154(6):1111–1116.
18. Falkenberg M, et al. Mitochondrial transcription factors B1 and B2 activate transcription of human mtDNA. Nature Genetics. 2002 Jul 1;31(3):289–294.
19. Gaspari M, et al. The mitochondrial RNA polymerase contributes critically to promoter specificity in mammalian cells. The EMBO Journal. 2004 Dec 1;23(23):4606–4614.
20. Bourne HR, Sanders DA, McCormick F. The GTPase superfamily: a conserved switch for diverse cell functions. Nature. 1990 Nov 29;348(6297):125–132.
21. Spaargaren M, Bischoff JR, McCormick F. Signal transduction by Ras-like GTPases: a potential target for anticancer drugs. Gene Expression. 1995 Jan 1;4(6):345–356.
22. Dang CV, Semenza GL. Oncogenic alterations of metabolism. Trends in Biochemical Sciences. 1999 Feb 1;24(2):68–72.
23. Ruggero D, Pandolfi PP. Does the ribosome translate cancer? Nature Reviews Cancer. 2003 Mar 1;3(3):179–192.
24. Boon K, et al. N-myc enhances the expression of a large set of genes functioning in ribosome biogenesis and protein synthesis. The EMBO Journal. 2001 Apr 2;20(6):1383– 1393.
25. Lindstrom MS, Nister M. Silencing of ribosomal protein S9 elicits a multitude of cellular responses inhibiting the growth of cancer cells subsequent to p53 activation. PLoS One. 2010 Mar 9;5(3):e9578.
26. Deisenroth C, Zhang Y. Ribosome biogenesis surveillance: probing the ribosomal protein–MDM2–p53 pathway. Oncogene. 2010 Jul 8;29(30):4253–4260.
27. Ziemiecki A, et al. Oncogenic activation of the human trk proto-oncogene by recombination with the ribosomal large subunit protein L7a. The EMBO Journal. 1990 Jan 1;9(1):191–196.
28. Kowalczyk P, et al. Increased expression of ribosomal protein S2 in liver tumors, posthepatectomized livers, and proliferating hepatocytes in vitro. Acta Biochimica Polonica. 2002 Jan 1;49(3):615–624.
29. Pogue-Geile K, et al. Ribosomal protein genes are overexpressed in colorectal cancer: isolation of a cDNA clone encoding the human S3 ribosomal protein. Molecular and Cellular Biology. 1991 Aug 1;11(8):3842–3849.
30. Bee A, et al. Ribosomal protein L19 is a prognostic marker for human prostate cancer. Clinical Cancer Research. 2006 Apr 15;12(8):2061–2065.
31. Cheung MC, et al. An evolved ribosome-inactivating protein targets and kills human melanoma cells in vitro and in vivo. Molecular Cancer. 2010 Feb 2;9:28.
32. Dey P. Nuclear margin irregularity and cancer: a review. Analytical and Quantitative Cytology and Histology. 2009 Oct 1;31(5):345–352.
33. Dey P. Chromatin pattern alteration in malignant cells: an enigma. Diagnostic Cytopathology. 2005 Jan 1;32(1):25–30.
34. Schwarz DS, Blower MD. The endoplasmic reticulum: structure, function and response to cellular signaling. Cellular and Molecular Life Sciences. 2016 Jan 1;73(1):79–94.
35. Lowe M. Structural organization of the Golgi apparatus. Current Opinion in Cell Biology. 2011 Feb 1;23(1):85–93.

36. McBride HM, Neuspiel M, Wasiak S. Mitochondria: more than just a powerhouse. Current Biology. 2006 Jul 25;16(14):R551–R560.
37. Wilson DN, Cate JH. The structure and function of the eukaryotic ribosome. Cold Spring Harbor Perspectives in Biology. 2012 May 1;4(5):a011536.