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Decoding biofilm tolerance in clinical infections: Matrixome architecture, metabolic zonation, and therapeutic implications

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Metabolic zonation

Therapeutic implications

ABSTRACT

Biofilm-associated infections continue to pose a major challenge to clinical and public health due to their high resistance to antimicrobial agents and host immune defense. Accordingly, for up to 50% of chronic infections, biofilms are the primary cause of treatment failure. Biofilms are complex, organized communities of microorganisms that are embedded in a self-produced extracellular matrix. Biofilms release toxins from structural proteins synthesized by the microorganisms and contribute to therapeutic resistance by the community. The matrix serves as structural and physiological barrier of infection, encapsulating metabolic wastes, environmental stressors (antibiotic efflux), and transport-based protection through bioactive efflux pumps. The unique complexity along with its extensive network, functional architecture promotes the development of the biofilm. The biofilm is a highly organized community of microorganisms. Despite advances in understanding biofilms, a significant gap remains in translation due to the reliance on planktonic-based diagnosis and treatment strategies. In this review, we discuss the role of the biofilm in clinical infection. We suggest defining novel 'biofilm-centric' clinical approaches that encompass systemic investigations, advanced imaging tools, and matrix degradation therapies, including nanotechnology, phage therapy, and quorum sensing inhibition. To prevent global health to emerge from this infection, interdisciplinary collaboration and accurate diagnosis are essential. Including pathogen through a biofilm-related control strategy is crucial for controlling emerging chronic and invasive diseases.

1. Introduction

Biofilm-associated infections continue to pose clinical and public health challenges. Despite advances in antimicrobial therapy, biofilms are structural microbial communities that enable organisms to adhere to surfaces, evade environmental resistance, and antimicrobial interventions, thereby contributing to chronic infections, particularly in medical devices and healthcare-related environments (Costa et al., 2020; Veng et al., 2021). Biofilms are complex communities of microorganisms, including bacteria, fungi, archaea, and eukaryotes, and contribute to global health issues. Biofilms are highly organized communities of microorganisms that are embedded in a self-produced extracellular matrix. Biofilms release toxins from structural proteins synthesized by the microorganisms and contribute to therapeutic resistance by the community. The matrix serves as structural and physiological barrier of infection, encapsulating metabolic wastes, environmental stressors (antibiotic efflux), and transport-based protection through bioactive efflux pumps. The unique complexity along with its extensive network, functional architecture promotes the development of the biofilm. The biofilm is a highly organized community of microorganisms. Despite advances in understanding biofilms, a significant gap remains in translation due to the reliance on planktonic-based diagnosis and treatment strategies. In this review, we discuss the role of the biofilm in clinical infection. We suggest defining novel 'biofilm-centric' clinical approaches that encompass systemic investigations, advanced imaging tools, and matrix degradation therapies, including nanotechnology, phage therapy, and quorum sensing inhibition. To prevent global health to emerge from this infection, interdisciplinary collaboration and accurate diagnosis are essential. Including pathogen through a biofilm-related control strategy is crucial for controlling emerging chronic and invasive diseases.

2. Biofilm formation and tolerance

Biofilms are complex communities of microorganisms that are embedded in a self-produced extracellular matrix. Biofilms release toxins from structural proteins synthesized by the microorganisms and contribute to therapeutic resistance by the community. The matrix serves as structural and physiological barrier of infection, encapsulating metabolic wastes, environmental stressors (antibiotic efflux), and transport-based protection through bioactive efflux pumps. The unique complexity along with its extensive network, functional architecture promotes the development of the biofilm. The biofilm is a highly organized community of microorganisms. Despite advances in understanding biofilms, a significant gap remains in translation due to the reliance on planktonic-based diagnosis and treatment strategies. In this review, we discuss the role of the biofilm in clinical infection. We suggest defining novel 'biofilm-centric' clinical approaches that encompass systemic investigations, advanced imaging tools, and matrix degradation therapies, including nanotechnology, phage therapy, and quorum sensing inhibition. To prevent global health to emerge from this infection, interdisciplinary collaboration and accurate diagnosis are essential. Including pathogen through a biofilm-related control strategy is crucial for controlling emerging chronic and invasive diseases.

3. Matrixome architecture and metabolic zonation

The extracellular polymeric substance (EPS) matrix acts as a barrier, limiting antimicrobial penetration and reducing immune effectiveness. While the biofilm environment, cells exist in diverse metabolic states, showcasing the adaptability of metabolic communities and the challenges in addressing biofilm-related infections. Additionally, subpopulations of persister cells can survive antibiotic treatments, causing recurrent infections. Biofilms also impact host immune responses, contributing to chronic inflammation and disease progression. To prevent global health to emerge from this infection, interdisciplinary collaboration and accurate diagnosis are essential. Including pathogen through a biofilm-related control strategy is crucial for controlling emerging chronic and invasive diseases.

4. Therapeutic implications

Consequently, an individual treatment strategy is ineffective against established biofilms, thus it is urgent need for an understanding of biofilm immune mechanisms and new anti-biofilm

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A parallel-group randomised controlled trial**

ORIGINAL ARTICLE

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**Dose-response evaluation of intravenous carbetocin for
prevention of postpartum haemorrhage during elective caesarean
delivery: A parallel-group randomised controlled trial**

Riya Abraham, Rajesh Prabhu Chandrasekaran, Pradeesh Johny, Akshaya Manimaran, C L Sabu Kumar, Ezhilraja Vaithiyalingam

Authors and Affiliations

Indian Journal of Anaesthesia 76(6) p 749-756, June 2026 | doi:10.4103/ij_a_391_26

OPEN

Abstract

Background and Aims:

Postpartum haemorrhage (PPH) is a major cause of maternal morbidity and mortality. Although 100 µg intravenous carbetocin is commonly administered for uterotonic prophylaxis at caesarean delivery, the optimal dose remains uncertain. The primary objective was to evaluate the dose-response relationship of intravenous carbetocin in achieving adequate uterine tone during elective caesarean delivery.

Methods:

In this randomised, parallel-group, dose-response trial, 100 parturients scheduled for elective caesarean delivery were assigned into five groups to receive intravenous carbetocin at doses of 20 µg, 40 µg, 60 µg, 80 µg, or 100 µg (n = 20 per group) immediately after foetal delivery. The primary outcome was satisfactory uterine tone at 2, 5, and 10 minutes. Secondary outcomes included blood loss, requirement for additional uterotonics, PPH incidence, blood transfusion, haemodynamic variables, and adverse events. Trend analysis assessed dose-response relationships.

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Expression and Methylation of Insulin-Like Growth Factor Binding Protein-1 Gene in Cord Blood of Intrauterine Growth Restricted Neonates

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Abstract

Objectives
To compare the insulin-like growth factor binding protein-1 (IGFBP-1) gene expression and expression in the umbilical blood among intrauterine growth restricted (IUGR) and appropriate for gestational age (AGA) neonates and find their association with anthropometric parameters.

Methods
Cord blood samples from IUGR and AGA neonates were collected. Anthropometric measurements i.e., weight, length and head circumference of the neonates were measured. RT-PCR was performed to estimate IGFBP-1 gene expression and DNA methylation analysis by bisulfite sequencing (BS). The results were analyzed by using Mann-Whitney U-test and Pearson correlation.

Results
IUGR neonates had significantly lower anthropometry (weight, length and head circumference) measurements in birth compared to AGA neonates. Gene expression analysis showed significantly higher IGFBP-1 gene expression among IUGR compared to AGA neonates and gene expression level correlated with anthropometric measurements. There was no difference in IGFBP-1 gene methylation levels between the two groups of neonates and no correlation was observed with anthropometry.

Conclusions
IGFBP-1 gene expression in cord blood was significantly higher among IUGR neonates and was associated with reduced birth weight, birth length and head circumference while the gene methylation was similar. This suggests involvement of multiple factors in occurrence of fetal growth restriction.

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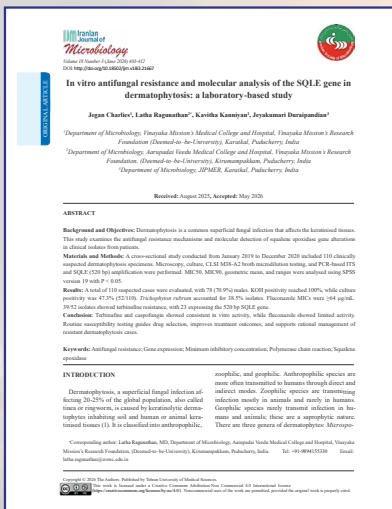
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In vitro antifungal resistance and molecular analysis of the SQLE gene in dermatophytosis: a laboratory-based study



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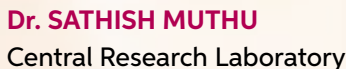
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A Study Assessing the Effectiveness of Lateral Partial Turbinatectomy versus Submucosal Conchoplasty for Concha Bullosa - A Randomized Controlled Trial

A Study Assessing the Effectiveness of Lateral Partial Turbinatectomy versus Submucosal Conchoplasty for Concha Bullosa - A Randomized Controlled Trial

Open Access | Published 1 June 2024
Volume 76 | Page 2571-2581 | DOI: 10.1007/s00034-024-02000-0

Abstract

Introduction

Concha bullosa, a prevalent anatomical variation of the middle turbinate, can cause significant symptoms like nasal obstruction and headaches due to sinus drainage blockage. Surgical approaches exist, with lateral partial turbinectomy (LPT) and submucosal conchoplasty (SMC) being common endoscopic procedures.

Aims

To assess the effectiveness of Lateral Partial Turbinectomy (LPT) and Submucosal Conchoplasty (SMC) in providing relief of nasal symptoms in patients diagnosed with concha bullosa.

Methods

This single-blinded randomized controlled trial, conducted at a tertiary healthcare centre, aimed to assess the effectiveness of LPT versus SMC in achieving clinical outcomes and to compare their postoperative complication profiles in patients with concha bullosa. Forty adult patients (20-65 years) with confirmed concha bullosa were recruited. Inclusion criteria: unilateral or bilateral concha bullosa diagnosed by either CT or MRI scans (10 per group). Primary outcomes included symptom relief assessed by the Nasal Obstruction Symptom Evaluation (NOSE) and Visual Analogue Scale (VAS), along with diagnostic nasal endoscopy at specified follow-up intervals (1 week, 12 weeks, 12 months). Complications like bleeding, infection, crusting, and speech were also monitored.

Results

Both groups showed significant improvement in pain and quality of life over 12 weeks ($p < 0.005$ for both). At 12 weeks, LPT demonstrated a lower post-operative pain control ($p < 0.0001$) and quality of

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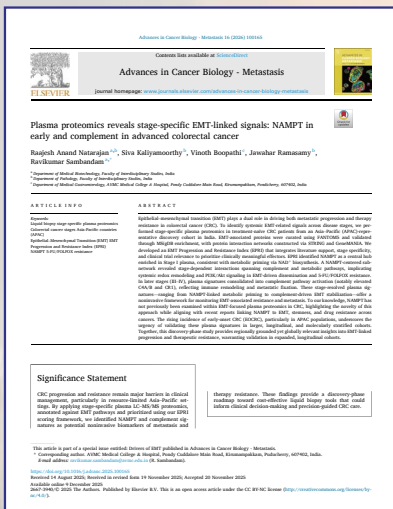
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Plasma proteomics reveals stage-specific EMT-linked signals: NAMPT in early and complement in advanced colorectal cancer



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Molecular investigation of Clustered Differentiation 40 gene in comparison with anti-cyclic citrullinated peptide autoantibodies in positive and negative Rheumatoid arthritis patients

Research Journal of Biotechnology

Vol. 23 (04) August (2024)
ISSN: 2349-2603

Molecular investigation of Clustered Differentiation 40 gene in comparison with anti-cyclic citrullinated peptide autoantibodies in positive and negative Rheumatoid arthritis patients

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⁴ Department of Microbiology, Sri Ramakrishna Dental College and Hospital, Coimbatore, INDIA

Abstract

Single nucleotide polymorphisms (SNPs) of the CD40 gene are expressed in normal B lymphocytes and endothelial cells and have been associated with susceptibility to rheumatoid arthritis (RA). Anti-cyclic citrullinated peptide (anti-CCP) antibodies are useful biomarkers for the early detection of RA, particularly in reverse clinical settings. This study aimed to evaluate the association between anti-CCP antibodies and CD40 gene polymorphisms in patients with and without RA. This cross-sectional study included 160 patients, comprising 80 symptomatic joint pain and swelling, participating in the study. Blood samples were tested for rheumatoid factor (RF), high-sensitivity C-reactive protein (hsCRP), and anti-CCP antibodies according to the manufacturer's instructions. Genomic DNA was extracted from EDTA blood samples for analysis of the CD40 gene.

Among 160 patients, males were predominant. Anti-CCP antibodies were detected in 52 (32.5%) in RF-positive and 19 (12.17%) in RF-negative patients. CD40 gene polymorphism was observed in 47.5% of RF-positive patients and 27% of RF-negative patients. A statistically significant association was observed between anti-CCP antibodies and the CD40 gene (p = 0.001). Anti-CCP antibodies and CD40 gene polymorphisms may play an important role in the early prediction of RA.

Keywords: Rheumatoid Arthritis, Cyclic citrullinated peptide, Rheumatoid Factor, IgG ELISA, Autoantibody.

Introduction

Early intervention of Rheumatoid arthritis (RA) is often associated with better long-term outcomes. RA is a chronic, systemic autoimmune disease, which primarily affects synovial joints and is marked by their inflammation, leading to chronic, worsening joint pain.^{1,2} It reduces the life span by average of 5-10 years by both disability as well as environmental factors that significantly decrease the

mobility of the joints.³ Early prediction of this disease helps in prevent and detect the disease dissemination and prevent complications. The disease is marked by detection of RA in early stages through various sensitive and specific diagnostic tests like Rheumatoid factor, anti-CCP antibodies etc.^{4,5}

In general, females is the most predominant than male for this disease. Autoantibodies against the citrullinated peptides are highly specific for the diagnosis of RA and are also slightly higher sensitive than RF. IgG antibody. Other subtypes of IgG is not elevated in general serum by using enzyme-linked immunosorbent assay (ELISA). It also suggested being a promising biomarker for the early diagnosis of RA.^{6,7}

Recently, CD40 gene is considered as a polymorphic gene to produce seropositive factors such as Rheumatoid growth factor (RFG) and interleukin-1 receptor antagonist (IL-1RA). The primary objective of this study is to correlate Anti-CCP antibodies levels with CD40 gene in patients with or without Rheumatoid Arthritis in Coimbatore population.

Material and Methods

This cross-sectional study was carried out after getting approval from the Institutional Human Ethics Committee (IHEC) (2020/04/18/CR/85), for the period of one year from April 2020 to April 2021. The study included 160 patients, comprising 80 symptomatic joint pain, swelling in the joints in various joints, chronic as well as laboratory data from Coimbatore District. After obtained consent from 160

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The Role of Indian Diabetic Risk Score In Predicting Metabolic Dysfunction Associated Statotoc Liver Disease In Non-Diabetic Population

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The Role Of Indian Diabetic Risk Score In Predicting Metabolic Dysfunction Associated Statotoc Liver Disease In Non-Diabetic Population

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Abstract

Background: Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD) represents a continuum of hepatic involvement ranging from uncomplicated fat accumulation to inflammatory injury, progressive fibrosis, cirrhosis, and ultimately hepatocellular carcinoma. In many non-diabetic individuals, MASLD remains clinically silent and is often driven by metabolic derangements such as abdominal adiposity, insulin resistance, and lipid abnormalities. Identifying individuals at risk at an early stage is essential to halt disease progression and reduce long-term hepatic and cardiovascular morbidity. The Indian Diabetic Risk Score (IDRS), a simple and validated screening instrument for diabetes, may also reflect underlying metabolic risk and thereby serve as a potential indicator for MASLD.

Aim: To assess the utility of the Indian Diabetes Risk Score as a predictor of MASLD in adults without diabetes.

Materials and Methods: A hospital-based cross-sectional study was carried out over an 18-month period at Aarupadai Veedu Medical College and Hospital, enrolling 97 non-diabetic adults aged 18-65 years. All participants were evaluated using the IDRS, along with detailed anthropometric measurements and biochemical investigations including fasting blood sugar, HbA1c, lipid profile, and liver function tests. Abdominal ultrasonography was performed to detect and grade hepatic steatosis. Statistical analysis involved chi-square testing and Pearson's correlation, with statistical significance defined as $p < 0.05$.

Results: The mean age of the study cohort was 42.5 years. The IDRS categorized 62.4% of participants. Higher IDRS categories showed a significant association with ultrasonographic evidence of fatty liver ($r^2 = 30.16, p = 0.01$), with most high-risk individuals demonstrating grade 1 or grade 2 steatosis. IDRS scores exhibited significant positive correlations with age ($r = 0.587$), fasting blood sugar ($r = 0.448$), HbA1c ($r = 0.570$), body mass index ($r = 0.545$), and waist circumference ($r = 0.451$). In addition, ultrasonographic abnormalities were significantly linked to advancing age and elevated fasting glucose levels.

Conclusion: The Indian Diabetes Risk Score emerges as a practical, non-invasive, and cost-effective tool for identifying non-diabetic individuals at increased risk of MASLD. Its use in routine clinical settings may facilitate early recognition and prompt lifestyle-based interventions, thereby helping to prevent progression to advanced liver disease and related metabolic complications.

Keywords: MASLD, IDRS, Ultrasonography, Non-Diabetic, Obesity, Metabolic Risk

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insights from the Mission Prison outreach initiative in India**



Volume 195, Issue
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<https://academic.oup.com/bjd/advance-article-abstract/doi/10.1093/bjd/ljab202/6600000>

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Abstract

Prison populations worldwide are medically underserved, with high dermatological morbidity driven by overcrowding, limited hygiene resources and restricted specialist access. Barriers such as structural inequity, inadequate on-site services, stigma and polycy concerns further discourage inmates from seeking timely care, leading to delayed diagnosis and a high untreated disease burden. The nationwide Medical Prison campaign led by the Indian Association of Dermatologists aims to bridge these gaps through increased outreach and education conducted across all states, providing a viable model relevant to prison healthcare systems worldwide. We evaluated dermatological disease burden, care gaps and the feasibility of delivering structured dermatology services in a prison setting. A 3-day dermatology camp was conducted in the central prison, Puducherry, India, where a team of 4 dermatologists evaluated 520 inmates (80% female, 20% male). The services provided included full-body skin examination, on-site treatment, counselling, sexually transmitted disease screening and laboratory testing for the chronic conditions. Demographic details and the spectrum of dermatological diagnoses were systematically recorded. A high prevalence of infectious aetiology, including scabies, fungal infections and pyoderma, followed by chronic inflammatory conditions, skin dermatoses and untreated pigmented disorders. Central diseases, including psoriasis, warts, suppurative infections, genital conditions, scabies, bacterial and herpes, were identified and treated. Many inmates presented with long-standing lesions. On-site treatment provided immediate relief, educational sessions improved hygiene and early case-seeking awareness, and dermatology enhanced continuity of care. This experience highlights significant dermatological care gaps in prison settings and underscores the importance of integrating structured dermatology camps into government prison healthcare programmes. Engagement of national professional societies, academic institutions and pharmaceutical partners can provide technical, educational and logistical support, creating a scalable and sustainable model to deliver equitable skin health services to medically underserved prison communities in India and beyond.

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Quercetin and Carvacrol Act Synergistically to Inhibit *Candida albicans* Biofilms In Vitro via Membrane Disruption and Oxidative Stress

Suganya Kannan^{1,2}, Jayashree Balasubramanian¹, Ananta Priya¹, Satyavani Kaliamurthi¹,
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Highlights

- Quercetin-carvacrol combination exhibited potent synergistic activity against *C. albicans* (FIC = 0.13).
- Combination treatment disrupted membrane integrity and enhanced intracellular leakage.
- ROS mediated apoptosis-like death was associated with nuclease gene activation and chromatin condensation.
- Key biofilm and virulence genes (*ALB1*, *HWP1*, *ECF1*, *LLP3*, *CAP1*, and *SDI1*) were significantly downregulated.
- Molecular docking supported multi-target interactions with virulence-associated proteins.

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A Novel Ruthenium(II) Complex and its Synergistic Anti-Leukemia Activity with GidA Protein via Dual-Pathway Apoptosis

Thieme

Drug Research

Original Article

A Novel Ruthenium(II) Complex and its Synergistic Anti-Leukemia Activity with GldA Protein via Dual-Pathway Apoptosis

Antony K. T. Isanta S¹, Thandeeswaran Murugesan² , Arand Raj Dhanapal¹, Palaniwamy Mathusamy Angayarkanni Jayaraman¹

This study demonstrates the role of the anticancer activity of a Cu-synthetic complex integrating the Cu^{II} complex and the zinc-finger (ZF) complex against K562 human leukemia cells. Monotherapy evaluation revealed exceptional cytotoxicity, with half maximal inhibitory concentration values of 0.08 μ M for CuI (a novel topoisomerase II inhibitor) and 14.1 μ M for RuII. Subsequently, isobologram analysis of six combinations for CuI/ZF complex synergism in formations with elevated RuII concentrations (combinations 4 and 5, sum of the fractional inhibitory concentration = 0.73–0.86). All these synergistic combinations were evaluated in K562 cells. The results showed that the combination of CuI and RuII on the one side, methanolic distillate revealed dual potency. RuII triggered reactive oxygen species dependent apoptosis (2.8 fold reactive oxygen species surge at 20 nM ($p < 0.01$)). While CuI operated via reactive oxygen species independent mechanisms. This zinc-synthetic complex may promote RuII, potentially mediated by transporter receptor overexpression for targeted uptake. Also, combination of [4 + RuII] + CuI emerged as the lead combination, reducing effects observed and potentially mitigating resistance. The study also revealed that the CuI/ZF complex may be a promising anticancer agent with potential for in vivo application. Integrating a strategic solution to chemotherapy limitations through rational synergy.

Keywords: anticancer drugs, biopharmaceuticals, cancer pharmacology, drug research

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Abbreviations

Abbreviations	
TopoII	Topoisomerase
Ru	Ruthenium
ROS	Reactive Oxygen Species
TRK1	Transthienin Receptor
IC ₅₀	Half-Maximal Inhibitory Concentration
IC	Fractional Inhibitory Concentration
DMSO	Dimethyl Sulfoxide
SVN	Synovial
ADO	Additive

Introduction

Over the last five decades, cytotoxic chemotherapy and radiation therapy have formed the cornerstone of cancer treatment. While yielding profound benefits for hematological malignancies and certain solid tumors (e.g., germ cell and childhood cancers), these approaches have shown limited efficacy against the most prevalent malignancies.^{1,2} Dose escalation strategies have offered only mar-

ginal improvements, failing to constitute an effective therapeutic breakthrough.

The effectiveness of conventional cytotoxic treatments is inherently constrained by their deleterious side effects on normal tissues. Although supportive measures (e.g., bone marrow rescue, antibiotics, and growth factors) have mitigated some toxicities, limitations to cancer therapy persist.¹⁻⁴ Simultaneously, advances in understanding cellular growth regulation, stress responses, and the mechanisms of cytotoxic agents provide a conceptual framework for developing more targeted chemotherapies at the molecular level.⁵

A pivotal shift occurred with the discovery that many chemotherapeutic agents induce cancer cell death via apoptosis. Landmark studies demonstrated that etoposide, a topoisomerase II (Topo II) inhibitor, triggers internucleosomal DNA fragmentation characteristic of apoptosis.³⁻⁷ This finding expanded to include diverse agents like cisplatin, cyclophosphamide, cytosine arabinoside, and 4'- α -acyridinylamino-2-methylsulfonyl-*N*-imidazole, which induce apoptotic cell death in various solid tumors and leukemia, both in experimental models and patients.⁸ Apoptosis is recognized as a critical mechanism underlying successful chemotherapy.⁹

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