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From Precision Cancer Therapy to Neuroprotection: Highlights of Four Recent Advances in Medicine and Biology

— A Research Brief on Frontier Progress, October 2026

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Abstract

This communication highlights four recent advances in medicine and biology: circulating tumour DNA-based biomarkers that predict benefit from perioperative nivolumab in resectable non-small-cell lung cancer; a binding-to-release strategy that overcomes the internalization barrier of targeted anticancer drug conjugates; sugar-rich diets that exacerbate antibiotic-induced gut microbiome disruption; and an intracellular, complement-independent neuroprotective role of factor H in multiple sclerosis. Together, these studies exemplify the accelerating translation of basic biomedical research into clinical precision practice.

Keywords: non-small-cell lung cancer; antibody-drug conjugates; gut microbiome; multiple sclerosis; biomarkers

Editorial

A recent crop of international publications spans several frontiers in medicine and biology, ranging from perioperative cancer therapy, targeted drug delivery and gut microbial ecology to neuroimmunology. This research brief selects four studies with particularly close relevance to clinical and basic research, and offers concise commentary on each, in the hope of providing readers with a panoramic view of current progress in these fields.

1. Predictive biomarkers of perioperative nivolumab benefit in resectable non-small-cell lung cancer

Immune checkpoint inhibitors have become a key component of perioperative therapy for resectable non-small-cell lung cancer (NSCLC), yet identifying the patients who truly benefit remains a clinical challenge. Cascone and colleagues report an exploratory biomarker analysis of the phase III CheckMate 77T trial (NCT04025879), in which pre-surgical circulating tumour DNA (ctDNA) clearance, post-surgical molecular residual disease (MRD) status and pathological complete response (pCR) together constituted key indicators of benefit from perioperative nivolumab. Among nivolumab-treated patients, 66% achieved

pre-surgical ctDNA clearance (versus 38% with placebo), and 50% of those who cleared ctDNA attained pCR; all patients whose MRD status converted from negative to positive during adjuvant therapy experienced disease recurrence. In addition, patients harbouring single or co-alterations in KEAP1, STK11, CDKN2A and/or SMARCA4 derived significantly longer event-free survival from nivolumab than from placebo (hazard ratio 0.48). These findings suggest that ctDNA dynamics-based liquid biopsy could become an important tool for perioperative immunotherapy decision-making.

Commentary: The "one-size-fits-all" era of perioperative immunotherapy is giving way to biomarker-guided precision strategies. ctDNA clearance and MRD monitoring add a quantifiable time dimension to response assessment and relapse prediction; although the analysis covered only 41% of randomized patients, its translational value merits close attention.[1]

2. A "binding-to-release" strategy overcomes the internalization bottleneck of targeted anticancer drug conjugates

Antibody-drug conjugates (ADCs) rely on receptor-mediated internalization to release their payloads, and roughly 90% of potential targets internalize poorly, limiting the reach of conjugate-based drugs. Wen and colleagues introduce a binding-to-release (BTR) strategy that positions an electrophile within the target protein's binding pocket so that a proximal nucleophilic residue can directly cleave and release the payload, thereby decoupling drug release from endocytosis. The team developed a phosphorus(V)-phenol exchange (PhoPEx) chemistry platform; in a fibroblast activation protein (FAP) model, the BTR small-molecule-drug conjugate achieved a 5.9-fold higher intratumoural exposure of monomethyl auristatin E than internalization-dependent counterparts, together with a 14.7-fold more favourable tumour-to-blood exposure ratio, and induced near-complete tumour regression across multiple models. The strategy is extensible to other targets such as PD-L1, offering a general framework that broadens the target space of therapeutic and diagnostic conjugates.

Commentary: The BTR strategy bypasses the "internalization ceiling" of ADC development, bringing a large pool of previously "undruggable" membrane targets within range of conjugate therapy, while its markedly reduced off-target release may widen the therapeutic window. This design concept could represent a paradigm shift in the ADC field comparable to site-specific conjugation.[2]

3. Sugar-rich diets exacerbate antibiotic-induced gut microbiome disruption

Antibiotics disrupt gut microbial homeostasis while eradicating pathogens, but the role of dietary factors in this process has been unclear. Dai and colleagues precisely tracked 9,419 meals consumed by 173 hospitalized patients undergoing allogeneic haematopoietic stem-cell transplantation for haematological malignancies, with longitudinal stool microbiome analysis in 158 of them. During broad-spectrum antibiotic exposure, each additional 100 g of sugar intake was associated with a further 20.6%-24.1% decline in gut microbial alpha diversity, accompanied by marked expansion of the pathobiont Enterococcus; sugar intake alone, without antibiotics, showed no such effect. Mouse experiments confirmed that sucrose supplementation increased and prolonged carbapenem-induced Enterococcus expansion by 16.3-fold on day 3 and 33.4-fold on day 6. The findings suggest that limiting simple sugars during antibiotic treatment may mitigate microbiota disruption.

Commentary: This "diet-drug-microbiome" interaction study offers a low-cost, actionable intervention cue, particularly for immunocompromised patients undergoing intensive chemotherapy and transplantation. Notably, the conclusions derive from a specific inpatient population, and whether they generalize to routine outpatient antibiotic use awaits validation in independent cohorts and prospective interventional trials.[3]

4. An intracellular, complement-independent neuroprotective role of factor H in CNS inflammation

Neurodegeneration is the principal driver of disability in chronic inflammatory diseases of the central nervous system (CNS) such as multiple sclerosis (MS), yet the neuron-intrinsic mechanisms of resilience remain poorly understood. Through single-nucleus RNA-sequencing of retinal ganglion cells from donors with MS, Mayer and colleagues found that complement factor H (CFH) expression correlated strongly with resistance to degeneration. Mechanistically, CFH was induced in retinal and other CNS neurons under inflammatory and oxidative stress, localized to the endoplasmic reticulum—a major site of lipid peroxidation during neuronal ferroptosis—and limited reactive oxygen species accumulation and lipid peroxidation. This protective activity depended on its C-terminal SCR20 domain but was independent of CFH secretion and of complement component C3. The study uncovers a previously unrecognized intracellular function of CFH and identifies it as a key mediator of neuronal resilience across the CNS in mice and humans, with implications for neuroprotective therapies in MS and beyond.

Commentary: The complement system has long been cast as a "villain" of CNS inflammation; this work reveals the neuron-intrinsic protective face of a complement regulator, providing a new conceptual framework for neuron-immune interaction research and opening avenues for ferroptosis-targeted neuroprotection.[4]

Concluding Remarks

Taken together, these studies exemplify the accelerating translation of basic biomedical research into clinical practice: from ctDNA-anchored precision perioperative immunotherapy, to chemistry-driven expansion of the conjugate-drug target space; from dietary modulation that helps preserve the gut microbial ecosystem, to complement factor-centred insights into neuronal intrinsic resilience. They suggest that future medical breakthroughs will increasingly rely on interdisciplinary strategies—the cross-fusion of genomics, chemical biology, microbiomics and neuroimmunology is reshaping the landscape of disease prevention and treatment.

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