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Sustaining innovation requires consistent investment in top scientific talent. When funding is this constrained, risk-taking and long-term discovery inevitably suffer

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Ramon Parsons, Director of the Mount Sinai Tisch Cancer Center, reflects on four decades of progress in oncology â?? from molecular genetics to immunotherapy and cellular therapies â?? and the critical role of comprehensive cancer centres in translating science into care. He highlights relapse, resistance and funding constraints as key challenges, calling for sustained investment, patient-centred research and closer collaboration between academia, industry and government.

Could you begin by introducing yourself and outlining your role at the Mount Sinai Tisch Cancer Center?

I am Ramon Parsons, Director of the Mount Tisch Cancer Center, which is an NCI-designated Comprehensive Cancer Center. The center encompasses clinical care, clinical research, basic research, and population health research across oncology.

My background is both clinical and scientific. I trained as an MD-PhD in the 1980s, at a time when our understanding of the molecular genetics of cancer was only just beginning to take shape. When I entered medical school, we knew very little about the genetic drivers of cancer. That period coincided with the early emergence of cancer virology and molecular biology, which ultimately became foundational to my career.

I initially studied oncogenic viruses during my PhD research and then moved to Johns Hopkins where I worked on colon cancer genetics. This included the discovery of genes responsible for Lynch syndrome, the most common hereditary colon cancer syndrome. I then went on to lead a laboratory at Columbia University where our focus was on the discovery of cancer genes.

In 2013, I joined the Icahn School of Medicine at Mount Sinai as Chair of Oncological Sciences. I led the cancer mechanisms programme at our cancer center during our first NCI designation application. We became NCI-designated in 2015, I was appointed deputy director in 2016, and then director in 2017. I have now served as director for over eight years.

From your long perspective in the field, what do you see as the most significant disruptors in oncology over the past few decades?

When I entered the field nearly 40 years ago, oncology looked very different. There were long periods with almost no new FDA approvals, and most therapies were variations of chemotherapy, such as taxanes or topoisomerase inhibitors. From when progress was once slow, there have been major strides and incredible momentum in the field within just the past decade or so. Understanding how far we have come really requires a long-term perspective.

Decades of work in cancer virology laid critical foundations for this transformation, from Peyton Rousâ??s early discoveries to the identification of Kaposi sarcoma virus in the 1990s. As DNA sequencing and hybridisation technologies began to emerge, it became clear that many viral cancer genes originated from the host genome itself. This helped reveal that cancer is driven by corrupted, mutated versions of normal cellular genes.

The development of DNA sequencing in the 1970s, followed by cloning technologies and polymerase chain reaction (PCR) in the early 1980s, laid the groundwork for modern molecular biology. These tools enabled us to identify cancer-causing genes and mutations, which is a process that reached maturity around the early 2010s.

Today, we have a comprehensive understanding of the genetic alterations that drive cancer. This has enabled the identification of actionable targets and the development of targeted therapies. While these therapies have meaningfully improved outcomes, resistance remains common, because tumours contain millions of cells, and it takes only one resistant clone for treatment failure.

As a result, combination therapy has re-emerged as a critical strategy. We learned decades ago that combination chemotherapy is more effective than single agents, and we are now relearning that lesson in the era of targeted and molecular therapies, as we work to combine agents that address multiple cooperating drivers within tumours.

How have targeted cancer therapies evolved over time, and what key limitations still remain?

Most targeted therapies today fall into two broad categories: small-molecule inhibitors and monoclonal antibodies. These approaches have transformed care for diseases driven by dominant oncogenic pathways, such as androgen receptor signalling in prostate cancer, oestrogen receptor signalling in breast cancer, EGFR-mutant lung cancer, and BCR-ABL-driven leukaemias.

At the same time, many oncogenic pathways tend to cooperate rather than act in isolation. Targeting a single pathway often leaves residual disease, which remains a significant limitation. Drug

development itself is also inherently slow. Some targets, such as the oestrogen receptor, have been known for more than a century. The first major therapeutic, tamoxifen, emerged decades ago, and we continue to refine and improve upon these agents today.

Taken together, this long arc of development suggests that our therapies will continue to improve with sustained research investment. As long as we maintain scientific momentum, our ability to precisely target cancer will continue to advance.

The tumour microenvironment has become a major focus in oncology research. Why has this area proven to be so important?

Approximately 25 years ago, it became clear that tumours do not exist in isolation. They actively shape their surrounding microenvironment, recruiting blood vessels, fibroblasts, and immune cells to support their growth and survival.

Tumours behave almost like conductors, orchestrating multiple non-cancerous cell types to create a permissive environment. Immune cells, such as macrophages, can paradoxically promote tumour growth, while tumours themselves secrete factors that stimulate angiogenesis and immune suppression.

This evolving understanding led to renewed interest in immunotherapy. While immune-based cancer treatments had been explored for more than a century, their effects were often inconsistent, in part because the complexity of the immune system made it difficult to identify reliable intervention points.

The discovery of immune checkpoints marked a turning point. Understanding how tumours suppress T-cell activation led to the development of immune checkpoint inhibitors, which have now been in clinical use for over a decade. These therapies have produced remarkable benefits in cancers such as melanoma and lung cancer, although responses remain variable across tumour types.

Cellular therapies are often described as the next frontier in cancer treatment. How do you see their impact to date?

Cellular therapies, particularly CAR T-cell therapy, represent a major advance in the field. These approaches involve engineering or enriching T cells so they can recognise specific cancer antigens and persist within the body.

They have shown extraordinary efficacy in certain haematological malignancies, including leukaemia, lymphoma, and multiple myeloma. These diseases are particularly amenable to cellular therapy because malignant cells express lineage-specific surface markers that allow for precise targeting.

While toxicity remains a consideration, it is increasingly manageable. Overall, the success of cellular therapies demonstrates what is possible when biology, engineering, and clinical infrastructure align.

How do comprehensive cancer centres enable the translation of these advances into routine clinical care?

Translating advances such as combination regimens and cellular therapies into routine clinical practice requires far more than scientific validation. It demands an integrated ecosystem with the expertise, infrastructure, and governance needed to operationalise complexity. That is where comprehensive academic cancer centres, such as the Mount Sinai Tisch Cancer Center, become indispensable.

At a minimum, these centres integrate three core pillars: basic research, population health research, and clinical research. From an implementation perspective, the clinical research infrastructure is especially critical. At Mount Sinai, this is anchored by a dedicated Clinical Trials Office, which is responsible not only for running trials, but also for opening them, maintaining regulatory compliance, ensuring appropriate staffing, and subjecting every study to both scientific and safety review.

Scale and subspecialisation also matter. We have around 150 staff supporting cancer clinical trials, reflecting the reality that oncology is no longer a single discipline. Care and research are organised into disease-specific teams, including breast, genitourinary, haematological, lung, gastrointestinal, and others. These teams are led by clinician-scientists working alongside specialised nurses and clinical research coordinators. The coordinators, in particular, are essential to implementation, ensuring informed consent, protocol adherence, and high-quality data capture.

Importantly, academic centres do not operate in isolation. While some discovery science occurs internally, most oncology therapies emerge from a broader academic and industrial ecosystem. Pharmaceutical companies are essential partners in scaling development, given the cost and complexity involved. Our clinician-scientists continuously engage with the basic, population, and clinical literature, assess how therapies perform in real-world patient populations, and work closely with industry to refine and advance them.

What distinguishes centres like the Mount Sinai Tisch Cancer Center is how patient data are translated into insight. Beyond imaging, we use circulating tumour DNA, single-cell sequencing, and immune profiling to understand response, resistance, and tumour biology in real time. These capabilities are supported by shared institutional resources across the Icahn School of Medicine at Mount Sinai in genomics, immune monitoring, and flow cytometry, and are complemented where necessary by external laboratories.

Crucially, these data directly inform the next generation of trials. Observations from patients drive new hypotheses, investigator-initiated studies, and industry-partnered trials. In that sense, academic cancer centres function as the translational engine of oncology, turning complex science into deliverable therapies and ensuring that innovation can be implemented safely, effectively, and at scale in everyday clinical practice.

Some trials are investigator-initiated and supported through grants or philanthropy, while others are industry-sponsored when companies recognise the strategic value. The process is iterative and patient-driven: clinical observations guide scientific questions, which in turn shape the next generation of trials.

Looking ahead, what do you see as the biggest priorities for sustaining innovation in oncology?

The overarching priority is to achieve sustained, meaningful improvements in patient outcomes. Alongside prevention and early detection, which remain essential, the development of new treatments is central to maintaining innovation and adaptability in cancer care.

The single biggest scientific gap we still face is relapse. Across many cancers, patients may respond well initially, only to relapse months or even a decade later. In many cases, we do not understand why one patient is cured while another relapses, or how the relapsed tumour has rewired itself to evade therapy. This is not only a question of tumour mutations; it applies equally to the immune response. Immunotherapy has delivered remarkable successes, but many patients still do not respond, and we need to treat those failures as signals that point to where our efforts should focus next.

Addressing relapse is not trivial. Tumours consist of millions of heterogeneous cells, capable of rapid genetic and epigenetic adaptation. Even when a drug effectively hits its intended target, the system can re-route around it. We are increasingly able to observe these changes, but translating observation into effective intervention remains a major challenge, and arguably the most important one facing the field.

A second priority is continued innovation in drug development and therapeutic modalities. Progress in oncology has always been driven by technology, and that will not change. AI-informed structural biology, for example, is opening new possibilities by revealing the multiple conformational states that proteins can adopt, exposing targets we could not previously see. Understanding how driver pathways adapt, mask themselves, or bypass inhibition will be essential if we are to stay ahead of resistance.

Finally, we need to study cancer in patients where it actually exists, while continuing to invest in foundational biology. Carefully designed clinical trials, combined with deep biochemical, cell biological, and immunological analysis, are now where much of the action lies. At the same time, animal models remain indispensable. Mouse models have underpinned virtually every FDA-approved cancer therapy and continue to enable experiments that simply cannot be done in humans.

Do you have any final reflections concerning the support of continuous, long-term innovation?

I will briefly step into the policy arena because it directly affects innovation. National Cancer Institute R01 funding rates are currently around the fourth percentile. Securing an NCI grant has always been difficult, but at these levels it becomes a serious barrier to progress. Talented scientists are losing funding and, in some cases, their ability to remain in the field.

Sustaining innovation requires consistent investment in top scientific talent. When funding is this constrained, risk-taking and long-term discovery inevitably suffer. A second priority is greater public support for exploratory clinical trials that use human tissue and blood. With so many new drugs emerging, often from small and underfunded companies, not all promising therapies receive the rigorous early evaluation they deserve. Publicâ??private partnerships could play an important role in identifying which approaches are most likely to succeed.

Pharmaceutical companies are also forced to make difficult choices because clinical trials are expensive. Even when several candidates appear promising, companies often have to advance only one programme, which creates a real risk of backing the wrong option. This is a structural limitation of the current system.

To move forward, we need stable research funding, stronger early-stage collaboration between government, academia, and industry, and clinical trial infrastructures that allow promising ideas to be tested efficiently rather than lost due to resource constraints.

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