Philosophy of Medicine
Perspective

Rethinking Cancer: Process Ontology, Creativity, and Cancer as a Chaotic Concrescence

Mesut Tez[1]

1 Department of Surgery, University of Health Sciences, Ankara City Hospital, Çankaya, Ankara, Turkey.
Email: mesuttez@yahoo.com

Abstract

This article redefines cancer through Alfred North Whitehead's process philosophy. Challenging models that frame cancer as a genetic error or stochastic mutation, I draw on Whitehead's principle that "creativity is the ultimate causality." Cancer thus emerges as a chaotic creative process: a concrescence wherein cells, dissociated from the organism's integrated social order, construct a new relational nexus, guided by a deviant subjective aim. This framework explains therapy resistance, heterogeneity, and metastasis, and suggests therapeutic strategies should redirect pathological creativity, rather than target static entities.

1. Introduction

Cancer has long been dominantly conceptualized as a genetic disease, the deterministic output of specific mutations in oncogenes and tumor suppressor genes. This reductionist paradigm, crystallized in the "somatic mutation theory," has undeniably driven significant progress. It has enabled the identification of key molecular drivers and facilitated the development of targeted therapies designed as "magic bullets" against these genetic lesions.

However, despite decades of research and clinical investment, durable cures remain elusive for most advanced cancers. Tumors consistently adapt, develop resistance, and recur. This recurring therapeutic impasse suggests, in my view, not a failure of medical science per se, but a failure of conceptualization. The prevailing model treats cancer as a static "thing"—a fixed entity, defined by its genetic blueprint. Yet the clinical reality reveals cancer as something far more dynamic: a process. It is a dynamic, adaptive strategy by which a cellular population strives to maintain its internal order (negentropy) against the pervasive pull of entropy, often at the expense of the host organism's higher-level organization.

This paper argues that a more adequate understanding of cancer requires an ontological shift. I turn to Alfred North Whitehead's process philosophy and Ilya Prigogine's theory of

ISSN 2692-3963 | Date Received 21-01-2026 | Date Accepted 10-03-2026 | Date First Published 28-04-2026
Philosophy of Medicine | DOI 10.5195/pom.2026.279 | Volume 7 | Issue 1 | pp. 1–5

dissipative structures to reframe cancer as a continuous state of *becoming*. At the heart of this reframing, I propose that cancer is a pathological, chaotic, yet fundamentally creative process—a *concrescence*. Recognizing cancer as *process,* rather than *phenotype,* is, I contend, essential for achieving transformative therapeutic progress.

2. Heterogeneity, Resistance, and the Limits of the Genetic Determinism Paradigm

The foundational assumption of genetic determinism is challenged by the overwhelming heterogeneity uncovered in cancer genomes. Large-scale sequencing efforts, such as The Cancer Genome Atlas (TCGA), have revealed a landscape where, aside from a few recurrent mutations (for example, in *TP53* or *PIK3CA*), the vast majority of genomic alterations are rare, patient specific, or even unique to subclones within a single tumor (Cancer Genome Atlas Network 2012; Nik-Zainal et al. 2016). This profound heterogeneity destabilizes the notion of cancer as the stable, deterministic output of a defined genetic program. If a specific set of mutations invariably caused cancer, mutational concordance across tumors would be significantly higher.

The clinical translation of the gene-centric model—targeted therapies against specific driver mutations—has produced striking but transient successes. Drugs targeting estimated glomerular filtration rate (eGFR), anaplastic lymphoma kinase (ALK), the BRAF gene, and so on are often hailed as breakthroughs and can induce dramatic initial regressions. Yet, therapeutic resistance emerges with near inevitability (Boumahdi and de Sauvage 2021). Crucially, this resistance frequently originates not from secondary *genetic* mutations in the drug target but from nongenetic adaptive mechanisms (Marine et al. 2020; Hangauer et al. 2017). Tumor cells exhibit phenotypic plasticity, dynamically rewiring signaling pathways, altering their metabolic state, adopting drug-tolerant persister phenotypes, or co-opting the microenvironment to survive. The tumor behaves not as a passive entity waiting to be struck down but as a resilient, adaptive system.

Recent research further demonstrates that cancer evolution can exhibit chaotic dynamics, where long-term outcomes become inherently unpredictable from initial conditions (Chattopadhyay et al. 2025). This unpredictability is not mere noise but an emergent property of complex adaptive systems. Together, these observations—extreme heterogeneity, nongenetic adaptation, and evolutionary unpredictability—point decisively away from cancer as a static genetic entity and toward its nature as a dynamic, adaptive process.

3. Cancer as Concrescence: A Whiteheadian Framework of Creativity, Purpose, and Chaos

Whitehead's process ontology provides a sophisticated metaphysical system to make sense of this dynamism. Its core tenet is that the ultimate constituents of reality are not static "substances" but dynamic "actual occasions" or "processes." Each actual occasion is an event of experience that arises through a process termed "concrescence"—becoming "concrete" (Whitehead 1978, 22). Three interrelated concepts are pivotal:

1) Prehension: An actual occasion does not exist in isolation. It "grasps" or prehends data from its entire past world—other occasions, relationships, possibilities. This is its raw material.
2) Subjective aim: Each concrescence is guided by an internal *subjective aim,* a telos or final cause that dictates *how* the prehended data will be integrated. It is the occasion's purpose.
3) Creativity: This is Whitehead's "Category of the Ultimate." Creativity is the fundamental principle whereby the many (the prehended past) become a novel one (the new actual occasion). It is the engine of novelty and the reason why process is never mere repetition: "Creativity is the ultimate causality" (Whitehead 1978, 22).

In this framework, cancer is not simply a biochemical "error" but can be reinterpreted as a *pathological yet creative concrescence*. We can delineate this process in three stages:

- *Stage 1: Deviation in final cause – the creative forging of a new aim*
  A healthy cell operates with a *subjective aim* aligned with the organism's holistic function (for example, tissue homeostasis). Carcinogenic stresses (genetic damage, inflammation, metabolic stress) disrupt this alignment. In the Whiteheadian moment, the affected cell *prehends* this disruption. Crucially, it does not respond deterministically. Applying its inherent creativity, it forges a new subjective aim. This new aim deviates radically from organismic service: Its purpose becomes self-preservation and expansion at any cost. This shift in *final cause* marks the inception of the cancerous process—the first malignant concrescence.
- *Stage 2: Pathological niche construction – the relational expansion of creativity*
  Now locked into its deviant aim, the nascent cancer cell (or cancer stem cell) externalizes its creativity. Its environment is no longer simply a source of data but becomes the relational medium for its concrescence. It actively *prehends* and *transforms* its surroundings: It co-opts fibroblasts into cancer-associated fibroblasts, educates immune cells toward a tolerogenic state, and hijacks angiogenesis. This active remodeling of the tumor microenvironment is a quintessential example of pathological niche construction. The cancer cell synthesizes the "many" elements of the local tissue into a "new one"—a supportive, pathological *nexus* centered on its own deviant aim. This aligns with Prigogine's concept of a "dissipative structure," a system that maintains its internal order by importing energy and matter from its environment, exporting entropy (Prigogine 1997; Prigogine and Stengers 1984).
- *Stage 3: Chaotic novelty in response to perturbation – therapy resistance and metastasis*
  Whiteheadian creativity is inherently novelty producing and nondeterministic. This imbues the cancer process with a chaotic character. Each cellular concrescence within the tumor involves micro-variations (*self-creativity*) in how data is processed. When a therapeutic agent—a powerful new datum—is introduced, the tumor nexus prehends it. The response is not programmed but creative. The system may generate unpredictable resistance strategies: upregulation of efflux pumps, activation of bypass signaling pathways, or a shift to a drug-tolerant persister state. This represents a nonlinear, chaotic system response sensitive to initial conditions. Metastasis, similarly, is the spatial dissemination of this creative process, as cells attempt to prehend and conquer the data of a distant organ, striving to initiate a new concrescence and build a novel, supportive nexus there.

This perspective coherently explains the observed evolutionary unpredictability of cancer (Chattopadhyay et al. 2025) and the epigenetic attractor landscapes that maintain cellular heterogeneity (Lopes et al. 2025) as natural expressions of a chaotic, context-sensitive creative process. Therapy resistance, on this view, is not an unfortunate bug but the expected manifestation of the tumor's creative capacity.

4. Conclusion and Therapeutic Implications: From Eradication to Redirection

The persistent failure of "miracle" gene-targeted drugs to provide durable cures signals an ontological crisis for the reductionist paradigm. Lasting progress in oncology demands a shift toward a *process ontology* that acknowledges cancer as a dynamic, adaptive, and creative concrescence.

My hypothesis—that cancer is a chaotic, creative process—carries profound implications for clinical thought and practice:

- *Redefining the therapeutic goal:* The objective must evolve from *eradicating a static target* to *destabilizing and redirecting a pathological process*. Successful intervention will likely require combinatorial strategies that target not only the cancer cell but also the relational network (nexus) it depends on—the dysregulated metabolism, the supportive signaling loops, and the co-opted microenvironment.
- *Anticipating and managing resistance as inevitability:* Resistance should be reconceived not as a treatment failure but as the system's creative adaptation. Therapeutic protocols must be designed to anticipate and outmaneuver this adaptability. Strategies like adaptive therapy, which modulates treatment pressure to suppress the emergence of resistant clones, embody this process-management philosophy (Gatenby et al. 2009).
- A deeper foundation for personalized medicine: If each tumor is a unique historical concrescence with its own *subjective aim,* true personalization must move beyond static genomic profiling. It requires dynamic assessment of the tumor's processual state—its active prehensions, its metabolic fluxes, and the structure of its ecological niche.

Ultimately, this ontological shift reframes the oncological mission. The task is not to wage a war of annihilation against a foreign entity but to guide a wayward process of becoming back toward alignment with the organism's holistic *final cause*. Cancer is a dark, pathological expression of the same creativity that underpins all life. We cannot and should not seek to destroy creativity itself, for it is the ultimate causality. Instead, we must learn to *redirect it*. This conceptual leap—from fighting a thing to redirecting a process—represents, in my view, the essential next step for oncology.

Disclosure Statement

No competing interest was reported by the author.

References

Boumahdi, Soufiane, and Frederic J. de Sauvage. 2021. "The Great Escape: Tumour Cell Plasticity in Resistance to Targeted Therapy." *Nature Reviews Drug Discov*ery 19, no. 1: 39–56. https://doi.org/10.1038/s41573-019-0044-1.

Cancer Genome Atlas Network. 2012. "Comprehensive Molecular Portraits of Human Breast Tumours." *Nature* 490, no. 7418: 61–70. https://doi.org/10.1038/nature11412.

Chattopadhyay, Subhayan, Jenny Karlsson, Michele Ferro, et al. 2025. "Evolutionary Unpredictability in Cancer Model Systems." *Scientific Reports* 15, no. 1, article 20334. https://doi.org/10.1038/s41598-025-07407-6.

Gatenby, Robert A., Ariosto S. Silva, Robert J. Gillies, and B. Roy Frieden. 2009. "Adaptive Therapy." *Cancer Research* 69, no. 11: 4894–4903. https://doi.org/10.1158/0008-5472.can-08-3658.

Hangauer, Matthew J., Vasanthi S. Viswanathan, Matthew J. Ryan, et al. 2017. "Drug-Tolerant Persister Cancer Cells Are Vulnerable to GPX4 Inhibition." *Nature* 551, no. 7679: 247–250. https://doi.org/10.1038/nature24297.

Lopes, Francisco, Bruno R.B. Pires, Alexandre A.B. Lima, Renata Binato, and Eliana Abdelhay. 2025. "NF-κB Epigenetic Attractor Landscape Drives Breast Cancer Heterogeneity." *npj Systems Biology and Applications* 11, no. 11, article 135. https://doi.org/10.1038/s41540-025-00611-0.

Marine, Jean-Christophe, Sarah-Jane Dawson, and Mark A. Dawson. 2020. "Non-Genetic Mechanisms of Therapeutic Resistance in Cancer." *Nature Reviews Cancer* 20, no. 12: 743–756. https://doi.org/10.1038/s41568-020-00302-4.

Nik-Zainal, Serena, Helen Davies, Johan Staaf, et al. 2016. "Landscape of Somatic Mutations in 560 Breast Cancer Whole-Genome Sequences." *Nature* 534, no. 7605: 47–54. https://doi.org/10.1038/nature17676.

Prigogine, Ilya. 1997. *The End of Certainty: Time, Chaos, and the New Laws of Nature*. Free Press.

Prigogine, Ilya, and Isabelle Stengers. 1984. *Order out of Chaos: Man's New Dialogue with Nature*. Bantam Books.

Whitehead, Alfred North. 1978. *Process and Reality: An Essay in Cosmology*. Free Press.