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March 6, 2026

Adam Cheong, BS or BA
Proof School
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Dear Adam Cheong,

On behalf of the Organizers, you are cordially invited to participate in the Population, Evolutionary, and Quantitative Genetics Conference to be held June 9-12, 2026 at Asilomar Conference Grounds in Pacific Grove, CA. The organizers greatly appreciate the important contribution that you will make to the meeting. The organizers greatly appreciate the important contribution that you will make to the meeting. All meeting attendees are required to pay a meeting registration fee and are responsible for all travel costs. The total cost of registration, housing and meals for the conference depends on the type of accommodations reserved and your registration category.

Your abstract entitled "How Cancer Cells Undergoing a Treatment Course Evolve to Survive Multiple Mass Extinctions" has been accepted and will be programmed. The Genetics Society of America is a professional membership organization for an international community of biologists advancing the field of genetics. The purposes of the Society are 1) to facilitate communication between geneticists, 2) to promote research that will bring new discoveries in genetics, 3) to foster the training of the next generation of geneticists so they can effectively respond to the opportunities provided by our discoveries and the challenges posed by them, and 4) to educate the public and their government representatives about advances in genetics and the consequences to individuals and to society.

The National Academies has a useful web site concerning obtaining a visa (<http://national-academies.org/visas>) for international participants. As part of new security procedures, many applications are being sent to the State Department in Washington where they are reviewed, with assistance from other agencies. Because of the number of visas being processed and the need to be thorough with the reviews, this can take as much as 3 to 4 months or more. Therefore, we advise scientists intending to come to the United States to apply for their visa as early as possible.

Sincerely,

Suzy Brown
Senior Director of Conferences
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How Cancer Cells Undergoing a Treatment Course Evolve to Survive Multiple Mass Extinctions

The vast diversity of genetic and phenotypic characteristics observed in human cancers, according to Hanahan (2026), can be understood in the context of how cancers arise via multistep pathways of tumorigenesis that lead to the formation of primary tumors, which subsequently evolve and progress due to selective advantage in the face of multifaceted barriers to continuing proliferative expansion, resulting in metastasis and adaptive resistance.

Here we elucidate the adversarial viewpoint: a press-pulse framework borrowed from paleobiology (Arens & West, 2008) for modeling therapeutic intervention targeted at the primary tumor, whose microenvironment undergoes terraforming and subsequently becomes unfit for cancer cells proliferation, leading to multiple mass extinctions over the course of cancer treatment, finally resulting in remission as the residual cancer cells turn quiescent.

The population dynamics driven by tumor growth vs. dose response is captured in our proposed model, as the cancer cells population follows a Gompertzian growth curve after metabolic reprogramming but shrinks in response to drug treatment. To shrink a tumor over multiple treatment cycles, dose responses are modeled as the outcome of catastrophic events decimating cancer cells as intravenous antibody-drug conjugate (Madhusoodanan, 2024) diffuses across blood vessel walls and penetrates the tumor, driven by concentration gradient according to Fick's laws.

Tumoral evolution within our framework offers mechanistic insights into the structure of a shifting fitness landscape characterized by the selective pressure of dose responses on residual regrowth. We illustrate how residual subpopulations of cancer cells evolve to survive multiple mass extinctions originated from a prescribed course of antibody-drug conjugate targeting the primary tumor, potentially laying the seeds for future metastasis or adaptive resistance in the unfortunate event of a relapse. We propose to construct a simulation testbed to further explore this key insight of population dynamics.

References:

1. Hanahan, D. (2026). Hallmarks of cancer—Then and now, and beyond. *Cell*, 189.
2. Arens, N. C., & West, I. D. (2008). Press-pulse: a general theory of mass extinction? *Paleobiology*, 34(4), 456–471.
3. Madhusoodanan, J. (2024, March 1). These cancers were beyond treatment—but might not be anymore. *Scientific American*.