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Exploiting tumor evolution and immune interactions to optimize cancer therapies

Treatment resistance limits lasting cancer therapy responses through tumor evolution driven by cellular heterogeneity. Therapeutic pressure selectively enhances resistant clone fitness while eliminating sensitive populations. We track evolutionary trajectories using genomic profiling and functional analyses to identify diverse resistance mechanisms in oncogene-dependent lung cancer, including bypass pathway activation. This knowledge guides rational combination therapy design. Additionally, we identified non-genomic adaptations in drug-tolerant persister cells that alter immune interactions and cell death pathways, revealing targetable vulnerabilities. Furthermore, these persister cells can be eliminated through exploiting induced sensitivities. Understanding these dynamic evolutionary and immunological landscapes thus helps to optimize therapeutic strategies and limit resistance development.

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Institute for Biological Physics, Zülpicher Str. 77a

Seminar Room S0.02

Hosted by Michael Lässig