- **Full Name:** Charles Swanton
- **Current Position & Affiliation:** Principal Investigator, Francis Crick Institute
- **Country:** United Kingdom

- **Educational Background:**

1995	BSc – University College London, UK
1998	MBPhD – University College London and Imperial Cancer Research Fund (which became Cancer Research UK in 2002), London, UK
1999 2007 2008	MBBS – University College London Medical Schools, UK PG Cert Oncology CCT, PMETB Medical Oncology

- **Professional Experience:**

Charles Swanton has made seminal contributions to our understanding of cancer evolution and how it can be applied to cancer therapy. He demonstrated the existence of subclonally related cell populations in renal tumours [confirming Peter Nowell's 1976 hypothesis], in parallel with work by Navin and Greaves. He subsequently established the UK-wide TRACERx programme to characterise lung cancer evolution during the clinical therapeutic trajectory. Swanton has also generated major insights into how genome instability facilitates cancer evolution, and in ongoing work has established a cardinal role for inflammation in lung cancer initiation, particularly in never-smokers.

- **Professional Organizations:**

- 2023-present Deputy Clinical Director, Francis Crick Institute, London, UK
- 2017-present Cancer Research UK Chief Clinician
- 2016-present Royal Society Napier Professor of Cancer Medicine
- 2014-present Co-Director, CRUK UCL/Manchester Lung Cancer Centre of Excellence
- 2013-present Principal Investigator, Francis Crick Institute (formerly ICRF, CRUK)
- 2011-present Professor of Cancer Medicine, Chair in Personalised Medicine, University College London

- **Main Scientific Publications:**

1. Gerlinger, M., et al. Intratumour heterogeneity and branched evolution revealed by multiregion sequencing. N Engl J Med, 2012 366(10): p.883-92
2. Jamal-Hanjani, M., et al. Tracking Non-Small Cell Lung Cancer Evolution. N Engl J Med. 2017 Jun 1;376(22):2109-2121
3. Watkins, T., et al. Pervasive chromosomal instability and karyotype order in tumour evolution. Nature. 2020 Nov;587(7832):126-132
4. Lee, AJX., et al. Chromosomal instability confers intrinsic multi-drug resistance. Cancer Research. Mar 1;71(5):1858-70

5. Turajlic, S., et al. Deterministic evolutionary trajectories influence primary tumor growth: TRACERx Renal. Cell. 2018 Apr 11. pii: S0092-8674(18)30375-1.
6. Turajlic, S., Tracking cancer evolution reveals constrained routes to metastases: TRACERx Renal Cell. 2018 Apr 9. pii: S0092-8674(18)30389-1.
7. McGranahan, N., et al. Allele Specific HLA Loss and immune escape in lung cancer evolution. Cell 2017 Nov 30;171(6):1259-1271
8. Rosenthal, R., et al., Neoantigen directed immune escape in lung cancer evolution. Nature, 2019. Mar:567(7749):479-485
9. Dewhurst, SM., et al. Tolerance of whole genome doubling propagates chromosomal instability and accelerates cancer genome evolution. Cancer Discovery **(2014)** Feb;4(2):175-85
10. López S., et al. Interplay between whole-genome doubling and the accumulation of deleterious alterations in cancer evolution. Nature Genetics. 2020 Mar;52(3):283-293.
11. McClelland, SE., et al. Replication stress links cancer structural and numerical chromosomal instability. Nature Feb 28;494(7438):492-6
12. McGranahan, N., et al. Clonal status of actionable driver events and the timing of mutational processes in cancer evolution. Science Translational Medicine Apr 15;7(283): 283ra54.
13. Burrell, RA., et al. Replication stress links cancer structural and numerical chromosomal instability. Nature Feb 28;494(7438):492-6.
14. Murugaesu, N., et al. Tracking the genomic evolution of esophageal adenocarcinoma through neoadjuvant chemotherapy. Cancer Discovery Aug;5(8):821-31.
15. Rosenthal, R., et al. deconstructSigs: Delineating mutational processes in single tumours distinguishes DNA repair deficiencies and patterns of carcinoma evolution**.** Genome Biology 2016 Feb 22;17(1):31.
16. Kanu, N., et al. DNA Replication Stress mediated APOBEC3 family mutagenesis in breast cancer. Genome Biology (2016) Sep 15;17(1):185
17. Venkatesan, S., Induction of APOBEC3 exacerbates DNA replication stress and chromosomal instability in early breast and lung cancer evolution. Cancer Discov. 2021 Oct;11(10):2456-2473.
18. Birkbak, NJ., et al. Paradoxical Relationship between chromosomal instability and survival outcome in cancer. Cancer Research. May 15;71(10):3447-3452.
19. Sansregret, L., et al. APC/C Dysfunction limits excessive cancer chromosomal instability. Cancer Discov. 2017 Feb;7(2):218-233.
20. López-García, C., et al. BCL9L dysfunction permits caspase-2 dependent aneuploidy tolerance in colorectal cancer. Cancer Cell**.** 2017 Jan 9;31(1):79-93.
21. Frankell, AM., et al. The evolution of lung cancer and impact of subclonal selection in TRACERx. Nature 2023 Apr;616(7957):525-533
22. McGranahan, N., Clonal neoantigens elicit T cell immunoreactivity and sensitivity to immune checkpoint blockade. Science (2016) Mar 25;351(6280):1463-9.
23. Al Bakir, Maise., et al. The evolution of non-small cell lung cancer metastases in TRACERx 421. Nature. 2023 Apr;616(7957):525-533.
24. Abbosh, C., et al., Phylogenetic ctDNA analysis depicts early-stage lung cancer evolution. Nature, 2017. 554(7961); p.264.
25. Abbosh, C., et al. Tracking early lung cancer metastatic dissemination in TRACERx using ctDNA. Nature. 2023 Apr;616(7957):553-562.
26. Litchfield, K. et al., Meta-analysis of tumor-and T cell-intrinsic mechanisms of sensitization to checkpoint inhibition. Cell 2021 Feb 4;184(3):596-614.
27. Turajlic, S., et al. Insertion-and-deletion-derived tumour specific neoantigens and the immunogenic phenotype: a pan-cancer analysis. Lancet Oncology 2017 Aug;18(8):1009-1021.
28. Litchfield, K., et al. Escape from nonsense-mediated decay associates with anti-tumor immunogenicity. Nature Communications. 2020 Jul 30;11(1):3800.

29. Hill. W., Lung adenocarcinoma promotion by air pollutants. Nature. 2023 Apr;616(7955):159-167.