

COMMITTEE ON CARCINOGENICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

The end of the genetic paradigm of cancer

1. A recent paper by Huang et al. (2025) argues that the long-standing somatic mutation theory (SMT) is insufficient to explain the complexity of cancer. A discussion paper, illustrating the core arguments from the paper, was provided to COM in October, and is presented here for COC to consider.
2. Annex A contains the discussion paper considered at the October 2025 COM meeting, and Annex B contains the draft initial notes of the COM discussion of the paper.
3. Members may wish to be reminded of these related Committee publications:
 - [A case for change: the challenge to develop a better approach to assessing risk of cancer caused by chemicals](#)
 - [COC watching brief: the tumour microenvironment](#)
 - [COC, COM, COT joint statement on the use of epigenetics in chemical risk assessment](#)

Questions for the Committee

4. The Committee is invited to consider the questions posed on page 11 of Annex A, and make suggestions for any further work on this, possibly to be conducted in conjunction with COM.

Secretariat
November 2025

This is a background paper for discussion.
It does not reflect the views of the Committee and should not be cited.

CC/2025/08 Annex A

**COMMITTEE ON CARCINOGENICITY OF CHEMICALS IN FOOD, CONSUMER
PRODUCTS AND THE ENVIRONMENT**

The end of the genetic paradigm of cancer

Discussion paper also presented at October 2025 COM meeting (MUT/2025/05)

**Secretariat
November 2025**

MUT/2025/05



**COMMITTEE ON MUTAGENICITY OF CHEMICALS IN
FOOD, CONSUMER PRODUCTS and the
ENVIRONMENT (COM):**

The end of the genetic paradigm of cancer

Contents

1. Discussion paper – The end of the genetic paradigm of cancer
2. Annex A – Brief summary of the Huang et al (2025) report
3. Annex B – Further recent, and potentially relevant, references

**COM Secretariat
October 2025**

MUT/2025/05

Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment.

The end of the genetic paradigm of cancer: Discussion paper

Introduction

1. A recent paper by Huang et al. (2025) argues that the long-standing somatic mutation theory (SMT) is insufficient to explain the complexity of cancer. A discussion paper, illustrating the core arguments from the paper, is put together here to facilitate discussion by COM members.
2. A brief summary of the Huang et al. (2025) report is given in Annex A.
3. Further recent, and potentially relevant, references are given in Annex B.

Background

4. Huang et al. (2025) state that the long-standing SMT paradigm is increasingly contradicted by data from genome sequencing of cancerous and normal tissues, and from single-cell transcriptomics, and propose expanding or replacing it with a more holistic framework that includes developmental biology, tissue context, and non-genetic cell dynamics.
5. The authors discuss relevant biological data and advocate for a “systems biology approach” that integrates:

- **Gene regulatory networks (GRNs):** Cancer as a disease of gene regulation, not just gene mutation.
- **Tissue-level organisation:** Cancer as a disease of disrupted tissue architecture and morphogenetic fields.
- **Phenotypic plasticity:** Emphasising reversible, non-genetic cell states that influence tumour behaviour.

6. The paper focuses on the relative roles of somatic mutation and epigenetic factors in the development of cancer.

“Paradoxical” findings relating to the genetic cancer paradigm

7. Huang et al. (2025) tabulate ten types of observation that question the genetic model. These are discussed briefly below.

Cancers without consistent mutations

8. Some tumours show abnormal phenotypes but display very few or no recurrent genomic mutations.

9. The authors give examples of cancers that lack consistent oncogenic mutations, such as rhabdomyosarcoma and ependymoma (Mack et al., 2014; Ohnishi et al., 2014; Versteeg, 2014). They note that mutational signatures associated with chemical carcinogen exposures can be indistinguishable from those of sporadic cases of the cancer (Moody et al., 2021). Moreover, almost every gene in the genome has been associated with cancer (de Magalhães, 2022).

Oncogenic mutations in normal tissues

10. It is well-known that tissues can tolerate mutations and remain normal without transforming, as evident by the ubiquitous accumulation of mutations in the somatic, non-cancerous cells of individuals suffering from inherited cancer syndromes that affect DNA repair (Baker et al., 2007; Hüsemann et al., 2008; Kato et al., 2016; Li et al., 2020; Martincorena et al., 2015; Rubin, 2006; Williams et al., 2022).

Premalignant lesions with driver mutations

11. Skin moles, Barretts's oesophagus, ductal carcinoma in situ (DCIS) or oncogenic driver mutations (such as in BRAF, TP53 or P13K) are frequently found but only a small fraction progress to cancer (and that is often clonally-unrelated to the original lesion) (Hafner et al., 2007; Kim et al., 2015; Ling et al., 2001; Tschandl et al., 2013; Weaver et al., 2014).

Radiation-induced tumours are multi-clonal

12. Ionising radiation leads to diverse clones, not single mutated ones.

13. While the SMT infers that genotoxic irradiation might lead to a single mutated tumorigenic clone, genetic alterations appear only several cell generations later, and are multi-clonal. It seems that ionising radiation leads to neoplasia via cytoplasmic or microenvironmental events, such as cell (replication) stress, or tissue injury and inflammation leading to genomic instability (Baverstock and Karotki, 2011).

Lack of positive selection

14. Oncogenic mutations often do not dominate the tumour population.

15. Under the SMT, cells with the tumour-driver mutations should be subject to positive selection, leading to their domination of the cell population (selective sweep and genetic fixation). According to Huang et al. (2025), the marginal positive selection of a mutated gene can occur, but rarely. They note, though, that neutral drift can extensively propagate non-advantageous mutations by chance, which creates the semblance of enrichment in genetically heterogeneous cell populations (Bakhoun and Landau, 2017; Ding et al., 2012; Ling et al., 2015; Robertson-Tessi et al., 2015 Sottoriva et al., 2015; Williams et al., 2016). [This is a key point for precision treatment, which relies on genetic fixation if residual cancer is to be avoided.]

16. The authors consider that the most plausible cases of positive selection are “point mutations in proteins” that interfere with target-selective inhibitor drugs, thus conferring drug resistance (Yang et al., 2022).

Complex clonal dynamics

17. Tumours show branching and non-linear evolution, not stepwise clonal selection.

18. Deep or multi-site sequencing of tumour genomes shows the coexistence of hundreds of genetic clones at any time point, indicating the absence of genetic fixation. The logic of the stepwise clonal selection of the SMT (linear evolution) seems to be defied by the fact that, in leukaemia (where repeated sampling of the malignant cells is possible), early cancer cell clones may “go into hiding”, and reappear in recurrent tumours (Ding et al., 2012; Gerlinger et al., 2014; Ling et al., 2015; Morrissy et al., 2016; Notta et al., 2011; Shlush et al., 2014).

19. Complex clonal dynamics supports the possibility of a multi-clonal origin of cancer but does not preclude a monoclonal origin (Davis et al., 2017; Marusyk et al., 2010; Sottoriva et al., 2015).

Cell lineage tracing

20. Cell lineage tracing reveals rather stochastic clonal kinetics of recurrence, and relapsing tumours are genetically unrelated to primary tumours.

21. Recurrence often involves new, unrelated clones.

22. Cell lineage tracking at the single cell level shows that recurrence after regression consists of multiple genetic clones, many not seen at diagnosis (Lee et al., 2016; Miles et al., 2020; Morrissy et al., 2016; Nam et al., 2019; Oren et al., 2021; Velten et al., 2021; Williams et al., 2022).

23. Some recurrent genetic alterations (such as copy number variations) can arise independently in normal cells, consistent with punctuated equilibrium evolution (Williams et al., 2024).

24. Clones in relapsing tumours (after removal of the primary tumour) can be genetically unrelated to the original tumour and thus represent an independent lineage. [A reference for this is not given in the relevant table (1) within Huang et al. (2025).]

25. The findings suggest the existence of tissue fields that encourage the acquisition of particular mutations that drive cancer recurrence (Lips et al., 2022).

Non-genetic heterogeneity

26. Identical genotypes show diverse phenotypes. The existence of multiple cell clusters and the dispersion within clusters in single-cell transcriptomics reveals ubiquitous non-genetic phenotype heterogeneity due to phenotypic plasticity – a fundamental property of cancer cell clones.

27. This phenotypic variability between cells with identical genotypes reflects enduring functional diversity with biological consequences (Huang, 2021).

28. There is disparity between genotype and phenotype; cancer cells carrying oncogenic mutations can have transcriptomes that cannot be distinguished from those of normal (non-mutated) cells, with which they cluster (Gopalan et al., 2021; Nam et al., 2019; Velten et al., 2021).

29. There are state transitions between transcriptomic cell clusters, and bar-coding combined with single-cell transcriptomics in perturbation/repopulation studies exposes intrinsic and externally-regulated dynamics with mass transitions of cells between clusters (Chang et al., 2008; Gutierrez et al., 2021).

30. Progenitor cells containing oncogenic mutations can still differentiate and contribute to mature lineages (Takahashi et al., 1998).

31. Distinct subpopulations in isogenic cell populations display distinct responses to perturbations, with cells in different subclusters responding differently to growth factors or drugs. They may also differ in susceptibility to oncogenic mutations (Shlyakhtina et al., 2024).

Persister cells

32. Persister cells may confer drug resistance and can revert, defying SMT.

33. Persister cells represent a specific form of non-genetic phenotype plasticity and, by enduring over multiple cell generations, can illustrate Darwinian selection characteristics without the need for genetic mutation. The fact that the persister state is reversible has implications for treatment, in that a relapsed, refractory tumour can become sensitive again to an originally-administered drug after a period without treatment, which would not appear to fit with the SMT (Balaban et al., 2004; Brock et al., 2009; Chaffer et al., 2011; Huang, 2021; Yano et al., 2005).

Cell states

34. Cell states can become therapy-resistant via induction rather than via selection, illustrating Lamarckian-like dynamics.

35. Therapy can induce resistant states via signalling, not mutation.

36. The appearance of stem-like, treatment-resistant cells is an active, regulated cell response to treatment stress, mediated by specific signalling pathways, and not a passive selection of pre-existing mutants. The stress-induced stemness/resistance can be cell-autonomous or non-autonomous, triggered by signals from other tumour cells or the microenvironment. Non-genetic induced resistance may have evolved as a rapid survival response following tissue injury, giving time for cells to accumulate mutations (via natural selection) of resistant mutant clones (Bell and Gilan, 2020; Ghisolfi et al., 2012; Huang, 2021; Hung et al., 2014; Karagiannis et al., 2017; Lee et al., 2015; Marin-Bejar et al., 2021; Marine et al., 2020; Maynard et al., 2020; Nör et al., 2014; Pisco et al., 2013, 2015; Salgia and Kulkarni, 2018; Shaffer et al.,

2017; Smith et al., 2021; Su et al., 2017; Sun et al., 2012; Zhang et al., 2021).

The epigenetic landscape in cancer

37. Huang et al. (2025) argue that the epigenetic landscape plays a central role in cancer, with GRNs governing cell phenotypes and disruptions leading to malignancy. The relationship between epigenetics and tumour behaviour is rooted in how GRNs and their epigenetic modifications influence cell phenotypes and dynamics.

38. Huang et al. (2025) discuss a number of features of the epigenetic landscape that drive tumour development, as summarised below.

39. Epigenetics governs the activity of genes through mechanisms such as DNA methylation, histone modifications, and chromatin remodelling. These processes (gene expression regulation) shape the gene expression profiles that define cell phenotypes, including those of tumour cells.

40. Tumour cells exhibit non-genetic phenotypic plasticity, allowing them to switch between different states (e.g., stem-like, drug-resistant, or invasive states). Epigenetic changes play a critical role in enabling these transitions, often in response to environmental stress or treatment.

41. Cancer attractors in the epigenetic landscape refer to stable gene expression configurations that represent latent, unused states – abnormal gene expression configurations associated with malignancy – within the GRN. These attractors are theorised to govern the malignant phenotypes of cancer cells. Cancer attractors are normally unoccupied under physiological conditions. They are not exposed to natural selection and represent vestigial or ancestral cell programs. Epigenetic modifications can influence the stability

of attractor states. Tumour behaviour arises when cells enter latent cancer attractors. Cells can enter these attractors due to perturbations in gene expression caused by genetic mutations, environmental stress, or stochastic fluctuations. This entry is accidental rather than driven by specific mutations. Cancer attractors often produce developmentally immature and dysfunctional traits, such as accelerated cell division, genomic instability, and tolerance to xenobiotics, which are characteristic of cancer cells.

42. Tumour cells trapped in cancer attractors are often unable to differentiate into normal mature states. Despite being trapped, cells in cancer attractors retain a theoretical potential for normalisation under specific experimental or biological conditions, though this is rarely realised.

43. Epigenetic mechanisms contribute to therapy resistance by enabling tumour cells to adopt persister states or stem-like phenotypes. These states are reversible and non-genetic, highlighting the role of epigenetic plasticity in tumour survival.

44. Epigenetic changes in tumour cells are influenced by signals from the tumour microenvironment, such as inflammation, hypoxia, or extracellular matrix alterations. These interactions can drive tumour progression and metastasis.

Summary of evidence

45. Huang et al. (2025) challenge the genetic paradigm of cancer and conclude that the available facts support the need for a more holistic framework in the understanding of tumour development that should include developmental biology, tissue context, and non-genetic cell dynamics. The publication focuses on cancer biology (which is complex) and its primary audience would seem to be oncologists.

46. The paper does not (directly) address non-genotoxic carcinogenicity, cancer hazard investigation, or cancer risk assessment.

Questions on which the views of the Committee are sought

- i) The publication focuses on mutagenic carcinogens. Bearing in mind the current version of COC Guidance Statement G11 (COC, 2024), does it undermine confidence in the current main responsibility that assessors of mutagenicity/carcinogenicity potential have, namely categorising chemicals into one of two groups, likely *in vivo* mutagens or likely non-mutagenic?
- ii) Have Huang et al. (2025) published sufficient justification for a paradigm shift (or discussion thereof)?
- iii) Does the publication undermine confidence in the present practice of assuming (as a worst-case default) that any carcinogenic action of an *in vivo* mutagen is not going to exhibit a demonstrable threshold?
- iv) The publication does not mention any of the well-known or presumed Modes of Action (MoAs) for carcinogens where DNA reactivity is not a key early step. Does it undermine the current understanding that there are a number of recognised MoAs responsible for cancer development, and that many of these involve critical steps that may have nothing to do with SMT?
- v) The publication does not discuss the regulatory testing strategies currently accepted or applied for identifying mutagens/mutagenic

carcinogens. Is there sufficient justification for a paradigm shift away from the current mutagenicity testing strategies?

- vi) The role of epigenetics in cancer development is emphasised, and that of mutations (even for genotoxic carcinogens) is considered diminished. Does this suggest that epigenetics-type screening assays could be a useful addition to mutagenicity hazard investigation? If so, which? And what guidance on interpretation is possible?
- vii) The publication does not address (quantitative) cancer risk assessment. Is there anything in the paper that might impact the content of COC Guidance Statement G11 (COC, 2024)? Is there anything in the paper to suggest that current approaches (e.g. linear extrapolation from a laboratory rodent cancer bioassay BMDL10 or TD50 to a specified cancer risk figure such as 1 in 100,000) are inadequately protective? For example, inter-individual variation in humans is likely to exceed that within a specific rat/mouse strain, so should a factor be added to account for this?
- viii) Would a more detailed review, evaluating the basis for a paradigm shift, be useful?
- ix) If yes, where should the focus be?

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This is a draft paper for discussion. This does not represent the views of the Committee and should not be cited.

List of Abbreviations and Technical terms

BMDL10	Lower confidence limit of the benchmark dose for a 10% benchmark response
DCIS	Ductal carcinoma in situ
GRN	Gene regulatory network
MoA	Mode of Action
SMT	Somatic mutation theory
TD50	Tumourigenic dose 50%

Definition

TD50: For any particular sex, strain, species and set of experimental conditions, the TD50 is the dose rate (in mg/kg body weight/day) that, if administered chronically for a standard period - the "standard lifespan" of the species - will halve the mortality-corrected estimate of the probability of remaining tumourless throughout the period.

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COC (2024). Committee on Carcinogenicity of Chemicals in Food, [Consumer Products and the Environment. A case for change: the challenge to develop a better approach to assessing risk of cancer caused by chemicals. COC Guidance Statement G11.](#)

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MUT/2025/05 ANNEX A

Committee on Mutagenicity of Chemicals in Food, Consumer Products and the Environment

The end of the genetic paradigm of cancer: Discussion paper

47. This Annex contains a brief summary of the Huang et al. (2025) report.

Brief summary of the Huang et al. (2025) report

48. The paper challenges the long-standing SMT, which posits that cancer is primarily caused by genetic mutations. The authors argue that this paradigm is increasingly contradicted (or judged inadequate) by scientific data, and describe the science that raises questions over the primarily genetic model. The apparent “paradoxes” include:

- Some tumours show abnormal phenotypes but lack recurrent/consistent driver mutations.
- “Normal” tissues often harbour oncogenic mutations that are considered cancerous without becoming malignant.
- Premalignant lesions such as moles or breast DCIS contain driver mutations but rarely progress to cancer.
- Ionising radiation leads to diverse clones, not single mutated ones.
- Oncogenic mutations often do not dominate the tumour population.

- Tumours show complex clonal dynamics, with branching and non-linear evolution, rather than stepwise selection.
- Recurrence/relapse often involves new clones that are genetically unrelated to the primary tumour.
- Identical genotypes show diverse phenotypes (non-genetic heterogeneity) due to plasticity.
- Non-genetic states confer drug resistance and can revert, defying SMT (referencing persister cells, a major cause of cancer relapse).
- Therapy can induce resistant states via signalling, not mutation (referred to here as Lamarckian-like dynamics).

49. The authors describe the dominance of SMT as a self-reinforcing paradigm, driven by technological advances and funding priorities, and call for a return to “deep biological thinking”, beyond data-driven precision oncology. They advocate for a systems biology approach that integrates:

- GRNs, where cancer is seen as a disease of gene regulation, not solely gene mutation.
- Tissue-level organisation, where cancer is seen as a disease of disrupted tissue architecture and morphogenetic fields.
- Phenotypic plasticity, where there is an emphasis on reversible, non-genetic cell states that influence tumour behaviour.

50. It is argued that SMT is insufficient to explain the complexity of cancer, and that it should be expanded or replaced with a broader, more holistic

framework that includes developmental biology, tissue-level dynamics, and non-genetic factors. The epigenetic landscape explains the immense diversity of molecular disruptions that can lead to cancer, emphasising the collective action of genes and the importance of tissue-level interactions.

The SMT model – How somatic mutation relates to cancer development

51. In the traditional SMT model, somatic mutations are thought to drive cancer by causing genetic alterations in a single cell, leading to uncontrolled proliferation and clonal expansion. By Darwinian evolution theory, certain mutations confer functional advantages, increasing cell "fitness" and promoting tumour progression.

52. Certain mutations, known as "driver mutations," are believed to initiate cancer by disrupting key regulatory pathways, such as those controlling cell division, apoptosis, and DNA repair. Somatic mutations can lead to genomic instability, which increases the likelihood of further mutations and accelerates tumour evolution.

53. Mutations can interfere with the effectiveness of targeted therapies, such as kinase inhibitors, by altering drug-binding sites and conferring resistance.

54. The authors accept that somatic mutations play a role in cancer development but question their direct causative impact, and urge that emphasis be placed on broader regulatory, tissue-level, and non-genetic factors.

The key challenges to the SMT

55. The authors outline a number of key challenges to the SMT, as summarised below.

56. Many cancers lack consistent driver mutations. In addition, canonical oncogenic mutations are often found in normal cells that do not develop into cancer. The authors classify these as inconsistent genetic findings that question the deterministic role of mutations in cancer causation.

57. Tumours exhibit extensive genetic diversity, with multiple coexisting clones and no clear evidence of selective sweeps or genetic fixation. This tumour heterogeneity undermines the idea of stepwise clonal selection. It was proposed that many mutations may propagate through random genetic drift rather than via positive selection, which would contribute to tumour heterogeneity. Epigenetic disruptions can lead to genomic instability, which further increases tumour heterogeneity (and, potentially, adaptability).

58. Cancer cells can demonstrate significant non-genetic phenotypic plasticity, including reversible states like "persister" cells, which can contribute to therapy resistance and later relapse, without acquiring genetic mutations.

59. The tumour microenvironment and tissue organisation play critical roles in carcinogenesis. This suggests that cancer is not solely a disease of the cell but also of the tissue.

60. Cancer cells can revert to normal phenotypes under specific conditions, and normal cells can become cancerous due to changes in the tissue environment. This challenges the idea that genetic transformation is irreversible.

61. Rather than having a direct role in cancer causation, mutation might alter the GRN, resulting in distortion of the epigenetic landscape. This can

lower barriers between normal and cancerous cell states, facilitating accidental entry into “cancer attractors” as a result of genetic mutation, environmental stress, or stochastic fluctuations. Cancer might thus arise from disruptions in gene regulation rather than specific mutations. [“Cancer attractors” represent latent, unused states in the regulatory landscape and are discussed in more detail later.]

62. In therapy, targeting driver mutations (precision oncology) has often failed to prevent relapse. It seems that tumours adapt through mechanisms not explained by SMT, such as non-genetic resistance.

63. The authors state that findings such as the normalisation of cancer cells in embryonic environments and the role of stromal interactions predate the molecular genetics era and contradict the SMT.

64. In a significant shift from conventional understanding, the authors posited that cancer may “beget mutations” rather than mutations “begetting cancer,” suggesting that genomic instability may be more of a consequence of the cancerous phenotype than its cause.

Epigenetics and cancer

65. The authors consider that epigenetics plays a pivotal role in shaping tumour behaviour. It does so by regulating gene expression, enabling phenotypic plasticity, and influencing interactions with the microenvironment. The epigenetic landscape provides a conceptual framework to understand the dynamic and adaptive nature of cancer beyond genetic mutations, how GRNs govern cell phenotypes, how disruptions can lead to malignancy, and how cancer can arise as a process of accidental entry into latent attractor states, driven by both genetic and non-genetic factors, rather than as a simple consequence of somatic mutations.

66. The paper discusses a number of aspects of the relationship, as follows.

67. Epigenetics governs the activity of genes (gene expression regulation) through mechanisms such as DNA methylation, histone modification, and chromatin remodelling. These processes shape the gene expression profiles that define cell phenotypes, including those of tumour cells.

68. Epigenetic changes play a critical role in enabling tumour cells to transition between different states (e.g., stem-like, drug-resistant, or invasive states) i.e. non-genetic phenotype plasticity. Such transitions often occur in response to environmental stress or treatment. The epigenetic landscape represents the dynamic configurations of gene activity within a GRN. Stable configurations, known as attractor states, correspond to specific cell phenotypes, such as normal or cancerous states.

69. The GRN of the epigenetic landscape contains latent, unused cancer attractor states (stable gene expression configurations) that are normally unoccupied under physiological conditions. These attractors are not exposed to natural selection and reflect vestigial or ancestral cell programs representing a reversion to primitive, protozoan-like functionalities. They often produce developmentally immature and dysfunctional traits, such as accelerated division, genomic instability, and tolerance to xenobiotics – all of which are characteristic of cancer cells. They are theorised to govern the malignant phenotypes of cancer cells. Epigenetic modifications can influence the stability of attractor states. Tumour behaviour arises when cells enter latent cancer attractors, which represent abnormal gene expression configurations associated with malignancy. Accidental entry into "cancer attractors" can result from genetic mutations, environmental stress, or stochastic fluctuations, leading to malignant phenotypes.

70. Cells trapped in cancer attractors are unable to differentiate into normal mature states. The pathological condition of maturation arrest is characteristic of cancer cells. This behaviour is linked to epigenetic disruptions that destabilise normal attractor states and trap cells in cancerous configurations.

71. Mutations can distort the epigenetic landscape by altering the GRN wiring, lowering barriers between normal and cancerous attractors. This facilitates accidental entry into cancer attractors but does not directly "cause" cancer in the traditional (SMT) sense.

72. It is known that non-genetic stresses such as tissue injury and inflammation can perturb the epigenetic landscape, which can trigger transitions into cancer attractors in the absence of any mutagenic events. Also, epigenetic changes in tumour cells are influenced by the surrounding tumour microenvironment, such as inflammation, hypoxia or extracellular matrix changes. Such interactions can drive tumour progression and metastasis.

73. Epigenetic disruptions can lead to genomic instability, further amplifying tumour heterogeneity and adaptability.

74. Epigenetic mechanisms can contribute to therapy resistance by enabling tumour cells to adopt persister states or stem-like phenotypes. These changes in state are reversible and non-genetic, highlighting the role of epigenetic plasticity in tumour survival.

75. The authors talk about normalisation or reversion potential, claiming that cells trapped in cancer attractors retain a theoretical potential for normalisation under specific conditions as epigenetic landscapes can be

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reshaped to favour non-cancerous attractor states. Evidently, this is rare in practice.

76. In essence, cancer attractors provide a theoretical framework to explain the emergence of cancer as a latent possibility inherent in the GRN dynamics, rather than as a direct consequence of specific genetic mutations.

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