nagasaichandra / **AutoFAQ**

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3 contributors 

227 lines (176 sloc) 20.1 KB Raw Blame History

Cancer

What kind of hybrid do classifications of disease like cancer form?

> *summarized_paragraph* : The larger concern cases like this raise is whether there is one way to privilege choice of causal basis for classifying diseases like cancer. Different choice of temporal or spatial scale, or more or less fine-grained characterization of causal processes might yield multiple, overlapping disease classifications. **It seems that classifications of disease like cancer form a kind of 'hybrid'—they are intended to capture natural regularities and their causes, but also to be of use for a wide array of agents with different purposes.**

> *avg_grammar_rating* : 4.3
> *avg_answerability_rating* : 3.7
> *sum_yes_meaningful* : 3
> *sum_no_meaning* : 0
> *sum_maybe_meaning* : 0

What do all the philosophers seem to do about the study

of mechanisms associated with cancer?

> *summarized_paragraph* : Philosophical discussions of how and why TOFT and CSC challenge a simple reductionist picture of cancer etiology are only two of several examples. Other philosophers have described evolutionary explanations of cancer (especially models of multilevel selection) Still others emphasize systems biology-based approaches to cancer , or the important role of a developmental perspective. **What all these philosophers seem to emphasize is that the study of mechanisms associated with cancer at the cell and molecular level requires supplementation to better predict and explain the origin of the disease.**

> *avg_grammar_rating* : 4.7
> *avg_answerability_rating* : 4.0
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 2
> *sum_maybe_meaning* : 0

What has many causes , and many effects?

> *summarized_paragraph* : The history of attempts to identify defining features of cancer, let alone arrive at a unified theory, has floundered. Either cancer is defined so vaguely as to include non-pathological states, or focused so narrowly on a specific class of causes. **Such a variety of definitions is due to the fact that cancer has many causes, and many effects.** It involves many different types of dysregulation, at a variety. of temporal and spatial scales. It is no small challenge to identify defines cancer, and arrive at unified “theory” of disease etiology.

> *avg_grammar_rating* : 4.3
> *avg_answerability_rating* : 5.0
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 1

Why is cancer a variety of definitions?

> *summarized_paragraph* : The history of attempts to identify defining features of cancer, let alone arrive at a unified theory, has floundered. Either cancer is defined so vaguely as to include non-pathological states, or focused so narrowly on a specific class of causes. **Such a variety of definitions is due to the fact that cancer has many causes, and many effects.** It involves many different types of dysregulation, at a variety. of temporal and spatial scales. It is no small challenge to identify defines cancer, and arrive at unified “theory” of disease etiology.

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> *avg_answerability_rating* : 4.3
> *sum_yes_meaningful* : 2
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 0

What is the term for the evolutionary relationship between the environment and the organism?

> *summarized_paragraph* : Philosophers of biology have been skeptical of claims about how our evolutionary history has shaped our vulnerability to disease. **Some such claims have been particularly contentious, either because the nature of the disease itself is not well characterized or evidence in support of evolutionary “mismatch” with our ancestral environment is both scant and disputed.** Some have argued that many arguments in evolutionary medicine make “adaptationist” assumptions, i.e., assumptions that a given trait is adaptive, or selectively advantageous, founded on at best “just so” stories.

> *avg_grammar_rating* : 5.0
> *avg_answerability_rating* : 1.7
> *sum_yes_meaningful* : 2
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 0

What kind of processes do ad hoc theories explain?

> *summarized_paragraph* : Laplane argues that CSC theory is more "parsimonious", because it "explains cancer development, propagation and relapse" from a "limited number of hypotheses" In contrast, the classical view neither predicts nor explains cancer heterogeneity, or relapse, but must invoke special ("additional" or "ad hoc" theories) to explain them, in particular, evolutionary processes. Laplane: CSC has the advantage that it 'connects basic research and intervention by suggesting new therapeutic strategy'

> *avg_grammar_rating* : 5.0
> *avg_answerability_rating* : 3.7
> *sum_yes_meaningful* : 3
> *sum_no_meaning* : 0
> *sum_maybe_meaning* : 0

What do explanatory frameworks help generate strategies in?

> *summarized_paragraph* : Cancer is not a single phenomenon, but a heterogeneous class of disease processes. This makes not only classification, but also explanation, enormously difficult, as we will see below. **One suggestion that seems plausible is that a better way of framing the project of cancer research involves commitment to an "explanatory framework." Rather than consisting of sets of laws or general principles, such frameworks help generate strategies in the search for causes, and narrow the field of inquiry.** The framework can "coexist" or be "gradually displaced," rather than stand in mutually exclusive relations with one another.

> *avg_grammar_rating* : 5.0
> *avg_answerability_rating* : 5.0
> *sum_yes_meaningful* : 3
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> *sum_maybe_meaning* : 0

What would the environment do to cancer cells?

> *summarized_paragraph* : The Cancer Genome Atlas project sought to characterize the genomic features of each cancer type and subtype. While TCGA did result in a more comprehensive understanding of cancer biology, it also raised many questions. **One particular challenge has been determining which mutations to which genes are the “drivers” of cancer, or which ones enable cancer cells to “succeed” in an environment that would prevent their growth.** Solving this puzzle has been difficult, because cancers are far more heterogeneous than they initially anticipated.

> *avg_grammar_rating* : 4.3
> *avg_answerability_rating* : 2.7
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 1

What are the " drivers " of cancer?

> *summarized_paragraph* : The Cancer Genome Atlas project sought to characterize the genomic features of each cancer type and subtype. While TCGA did result in a more comprehensive understanding of cancer biology, it also raised many questions. **One particular challenge has been determining which mutations to which genes are the “drivers” of cancer, or which ones enable cancer cells to “succeed” in an environment that would prevent their growth.** Solving this puzzle has been difficult, because cancers are far more heterogeneous than they initially anticipated.

> *avg_grammar_rating* : 4.8
> *avg_answerability_rating* : 3.5
> *sum_yes_meaningful* : 4
> *sum_no_meaning* : 0
> *sum_maybe_meaning* : 0

What theory cannot account for the observations of

tissue architecture?

> *summarized_paragraph* : Soto and Sonnenschein claim that there are features of the tissue microenvironment that affect either the induction of or progression of cancer. Such features are not reducible to, or explainable in terms of properties or processes at or below the cell and molecular level, they say. **Further, they claim that the somatic mutation theory cannot account for these observations, and that they can only be accommodated on a view that takes that tissue architecture itself as causally relevant to the progression of disease.**

> *avg_grammar_rating* : 5.0
> *avg_answerability_rating* : 5.0
> *sum_yes_meaningful* : 3
> *sum_no_meaning* : 0
> *sum_maybe_meaning* : 0

What type of cancer is a major component of the causal basis of?

> *summarized_paragraph* : Family history and inherited (as opposed to somatic) mutations, life history (e.g. parity, or number of children, and age at first birth), environmental exposures, and histories of infection, inflammation, and immune response, contribute differentially to cancer's age of onset, rate of progression, histopathology, and response to treatment. **Depending upon how fine-grained a characterization of the causal basis of cancer one adopts, there could be hundreds or thousands of cancer types and subtypes.**

> *avg_grammar_rating* : 1.7
> *avg_answerability_rating* : 2.7
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 2
> *sum_maybe_meaning* : 0

What is one type of cancer type?

> *summarized_paragraph* : Family history and inherited (as opposed to somatic) mutations, life history (e.g. parity, or number of children, and age at first birth), environmental exposures, and histories of infection, inflammation, and immune response, contribute differentially to cancer's age of onset, rate of progression, histopathology, and response to treatment. **Depending upon how fine-grained a characterization of the causal basis of cancer one adopts, there could be hundreds or thousands of cancer types and subtypes.**

> *avg_grammar_rating* : 2.0
> *avg_answerability_rating* : 1.7
> *sum_yes_meaningful* : 2
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 0

What do philosophers have on the aim and character of explanation and progress in cancer research?

> *summarized_paragraph* : What does it mean to explain cancer? The question is ambiguous, and fraught. Each of these targets arguably calls for quite different kinds of explanation. Cancer scientists make appeals to generalizations of wide scope, for instance, and will also offer detailed, specific mechanistic explanations of specific outcomes. **The fact that these explanations are different in kind may explain why there appears to be such a variety of views amongst philosophers concerning the aim and character of explanation and progress in cancer research.** It's still hotly debated whether all explanations in the biological sciences must identify causes, or mechanisms, or whether "mathematical" or "equilibrium" explanations are distinct in kind.

> *avg_grammar_rating* : 2.7
> *avg_answerability_rating* : 4.0
> *sum_yes_meaningful* : 1
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What are different in kind in relation to cancer research?

> *summarized_paragraph* : What does it mean to explain cancer? The question is ambiguous, and fraught. Each of these targets arguably calls for quite different kinds of explanation. Cancer scientists make appeals to generalizations of wide scope, for instance, and will also offer detailed, specific mechanistic explanations of specific outcomes. **The fact that these explanations are different in kind may explain why there appears to be such a variety of views amongst philosophers concerning the aim and character of explanation and progress in cancer research.** It's still hotly debated whether all explanations in the biological sciences must identify causes, or mechanisms, or whether "mathematical" or "equilibrium" explanations are distinct in kind.

> *avg_grammar_rating* : 4.0
> *avg_answerability_rating* : 4.0
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 2
> *sum_maybe_meaning* : 0

What type of causation do these examples point to?

> *summarized_paragraph* : Bissell's lab seems to indicate that structural features of the extracellular matrix somehow either prevent the emergence of disease, or can accelerate it. **It seems such examples do not point to 'downward causation,' but instead suggest that we need to expand our understanding of the causes of cancer to include maintenance of tissue integrity.** That is, the case of cancer seems to require a richer, more integrative and interdisciplinary approach to investigating cancer. It seems each of these authors is pointing to an important role for structural organization in the maintenance of homeostasis.

> *avg_grammar_rating* : 4.7
> *avg_answerability_rating* : 4.0
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 2
> *sum_maybe_meaning* : 0

What did a more comprehensive understanding of cancer

biology result in?

> *summarized_paragraph* : The Cancer Genome Atlas project sought to characterize the genomic features of each cancer type and subtype. **While TCGA did result in a more comprehensive understanding of cancer biology, it also raised many questions.** One particular challenge has been determining which mutations to which genes are the “drivers” of cancer, or which ones enable cancer cells to “succeed” in an environment that would prevent their growth. Solving this puzzle has been difficult, because cancers are far more heterogeneous than they initially anticipated.

> *avg_grammar_rating* : 5.0
> *avg_answerability_rating* : 4.0
> *sum_yes_meaningful* : 2
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 0

What type of organization does each of the authors point to in the maintenance of homeostasis?

> *summarized_paragraph* : Bissell’s lab seems to indicate that structural features of the extracellular matrix somehow either prevent the emergence of disease, or can accelerate it. It seems such examples do not point to ‘downward causation,’ but instead suggest that we need to expand our understanding of the causes of cancer to include maintenance of tissue integrity. That is, the case of cancer seems to require a richer, more integrative and interdisciplinary approach to investigating cancer. **It seems each of these authors is pointing to an important role for structural organization in the maintenance of homeostasis.**

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> *avg_answerability_rating* : 2.5
> *sum_yes_meaningful* : 4
> *sum_no_meaning* : 0
> *sum_maybe_meaning* : 0

Is the concern of whether there is one way to privilege choice of causal basis for classifying diseases like cancer or smaller?

> *summarized_paragraph* : **The larger concern cases like this raise is whether there is one way to privilege choice of causal basis for classifying diseases like cancer.** Different choice of temporal or spatial scale, or more or less fine-grained characterization of causal processes might yield multiple, overlapping disease classifications. It seems that classifications of disease like cancer form a kind of 'hybrid'—they are intended to capture natural regularities and their causes, but also to be of use for a wide array of agents with different purposes.

> *avg_grammar_rating* : 2.0
> *avg_answerability_rating* : 1.7
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 2
> *sum_maybe_meaning* : 0

What type of choice of causal basis is there a concern about?

> *summarized_paragraph* : **The larger concern cases like this raise is whether there is one way to privilege choice of causal basis for classifying diseases like cancer.** Different choice of temporal or spatial scale, or more or less fine-grained characterization of causal processes might yield multiple, overlapping disease classifications. It seems that classifications of disease like cancer form a kind of 'hybrid'—they are intended to capture natural regularities and their causes, but also to be of use for a wide array of agents with different purposes.

> *avg_grammar_rating* : 3.3
> *avg_answerability_rating* : 3.0
> *sum_yes_meaningful* : 2
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 1

What type of model is used to study cancer?

> *summarized_paragraph* : Soto and Sonnenschein object not only to reductionist methodology, but reductionist assumptions about the causes of cancer. In their view, cancers are not the result of mutations, but instead “alterations in the communication among cells and tissues that affect tissue architecture” It is these alterations of patterns of interaction between cells that leads to “downward causation effects” on cells and cellular components, inducing aneuploidy and mutations, they say. **They bring attention to some interesting broader questions as to when are we warranted in inferring from cell culture or model organism work to general claims about cancer etiology.**

> *avg_grammar_rating* : 5.0
> *avg_answerability_rating* : 2.3
> *sum_yes_meaningful* : 2
> *sum_no_meaning* : 1
> *sum_maybe_meaning* : 0

On what view of cancer are cancer cells highly adaptive?

> *summarized_paragraph* : Cancer is a disease of aging, perhaps in part as a byproduct of breakdowns in mechanisms associated with immune response and tissue integrity. **On the evolutionary view of cancer, for instance, cancer cells are in some sense highly adaptive.** All evolutionary and byproduct explanations of cancer raise similar philosophical questions about hypothesis testing, as well as definitions of “function” and “individuality” Both at the individual and group level, and over the course of one’s life history.

> *avg_grammar_rating* : 5.0
> *avg_answerability_rating* : 5.0
> *sum_yes_meaningful* : 3
> *sum_no_meaning* : 0
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On the evolutionary view of cancer , what is it that cancer

cells are highly adaptive?

> *summarized_paragraph* : Cancer is a disease of aging, perhaps in part as a byproduct of breakdowns in mechanisms associated with immune response and tissue integrity. **On the evolutionary view of cancer, for instance, cancer cells are in some sense highly adaptive.** All evolutionary and byproduct explanations of cancer raise similar philosophical questions about hypothesis testing, as well as definitions of “function” and “individuality” Both at the individual and group level, and over the course of one’s life history.

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> *avg_answerability_rating* : 1.5
> *sum_yes_meaningful* : 1
> *sum_no_meaning* : 3
> *sum_maybe_meaning* : 0

What is the term for fitness in cancer?

> *summarized_paragraph* : Cancer is a product of breakdown in mechanisms that permitted the emergence of multicellularity. The mechanisms that protect us from “revolt from within” are not error-free, and over time will fail. **On this view, cancer itself may be viewed as an evolutionary process, where these “adaptations” enable short term “fitness,” or relative success at survival and reproduction.** Several scientists have developed theoretical models of this process, linking it to empirical data, e.g., on emergence of chemotherapy resistance.

> *avg_grammar_rating* : 4.0
> *avg_answerability_rating* : 3.0
> *sum_yes_meaningful* : 2
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What type of success does cancer have at survival and reproduction?

> *summarized_paragraph* : Cancer is a product of breakdown in mechanisms that permitted the emergence of multicellularity. The mechanisms that protect us from “revolt from within” are not error-free, and over time will fail. **On this view, cancer itself may be viewed as an evolutionary process, where these “adaptations” enable short term “fitness,” or relative success at survival and reproduction.** Several scientists have developed theoretical models of this process, linking it to empirical data, e.g., on emergence of chemotherapy resistance.

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> *avg_answerability_rating* : 4.0
> *sum_yes_meaningful* : 3
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According to malaterre , what is the target of proportionality?

> *summarized_paragraph* : Malaterre argues that advocates of TOFT need not commit to non-reductive physicalism, or emergent properties. He argues that Soto and Sonnenschein are drawing attention to dynamic relationships between variables that supervene over molecular and cellular activities. **Malaterre makes appeal to Woodward’s argument that choice of causal variables often involves appeal to something like a notion of “proportionality” such that, depending on the target of inquiry, we may be “led to the incorporation of more fine-grained detail”**

> *avg_grammar_rating* : 4.3
> *avg_answerability_rating* : 4.3
> *sum_yes_meaningful* : 1
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