原註

原註については、検索の利便性を念頭に、すべて以下のサイトに上げた。参照していただきたい（QR コードは 10 頁にあり）。なお、基本的に原書に忠実に掲載したが、URL については、リンク切れのものは、確認できる範囲で外し、未確認のものはすべて原書のままとした。

https://www.shinchosha.co.jp/book/507351/?utm_source=QRcode&utm_medium=book&utm_campaign=507351#b_othercontents

こちらには、出典以外の解説については、翻訳の上掲載する。

なお、著者のサイトは以下、参考に掲載しておく。 https://www.kpharden.com

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たちを糾弾する一方で、人々に、そしてわれわれ自身に、白人性が生き延びている──そして白人性が明示的にも暗黙的にも支持されている──あり方について深く考えてみようと訴えることを意図して 'White'と大文字で始める」"Racial and Ethnic Identity," APA Style, accessed February 8, 2021, https: //apastyle. apa. org/style-grammar-guidelines/bias-free-language/racial-ethnicminorities; Ann Thúy Nguyễn and Maya Pendleton, "Recognizing Race in Language: Why We Capitalize 'Black' and 'White,'" Center for the Study of Social Policy, March 23, 2020, https://cssp.org/2020/03/recognizing-race-in-languagewhy-we-capitalize-black-and-white/.

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22. 本書を通して私は「親」「子ども」「家族」「きょうだい」という言葉を、遺伝のプロセスにより互いに関係する人たちという狭い意味で用いる。これは「家族」を定義する社会的関係の重要性を否定するものではなく、単に、本書は遺伝子の効果に焦点を合わせるからである。

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13. より具体的には、この方法を使うことで平均治療効果（ＡＴＥ）を推定することができる。しかしながらＡＴＥは、研究者が推定したいと思うかもしれない唯一の量ではない。たとえば、研究者は治療に対する応答における不均質性に興味を持つかもしれない。さらなる議論については、以下の文献を参照されたい。Angus Deaton and Nancy Cartwright, “Understanding and Misunderstanding Randomized Controlled Trials,” *Social Science & Medicine* 210, special issue: Randomized Controlled Trials and Evidence-Based Policy: A Multidisciplinary Dialogue（August 2018）: 2-21, https://doi.org/10.1016/j.socscimed.2017.12.005.

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15. 進化生物学者のリチャード・ドーキンスは、たとえば眼の色のような、直観的にも「遺伝で決まっている」と思える比較的単純な表現型についてさえ、遺伝的原因は「違いを生むもの」として定義されるべきだと主張した。彼はこう書いた。「任意の原因候補の“効果”に意味が与えられるのは、その原因に代わりうる、少なくともひとつの別の原因との比較──たとえその比較が言外になされるものであったとしても──という観点に立つ場合だけである。ある遺伝子 *G1* だけで確定する“効果”として青い眼について語るのは、完全に不完全なのだ。もしもわれわれがそういう言い方をするとしたら、それは実際には、その遺伝子に代わりうる少なくともひとつの別のアレル──それを *G2* と呼ぼう──と、青い眼という表現型に代わりうる少なくともひとつの別の表現型 *P2*──この場合であれば、たとえば茶色の眼──がありうるということを述べているのである」

ドーキンスはこれに続けて、皮膚の色に関連するふたつの遺伝子を例に挙げる。「たしかに、一個体の皮膚の色が黒くなるためには、合成されるタンパク質が黒い色素であるような遺伝子 *A* が必要だろう。しかし、集団内の多様性の中に、*A* の欠如によって引き

起こされる何かがないかぎり、私は *A* を、皮膚の色を黒くする遺伝子とは呼ばない。……ここで重要なのは、*A* と *B*［*B* はここで、黒い色素を作る遺伝子ではなく、酵素として作用するタンパク質を合成する遺伝子であって、その酵素の間接的な効果のひとつに、（別のアレル *B′* と比べて）*A* による黒い色素の合成を促進することが含まれると仮定されている］のどちらもが、皮膚の色が黒くなるための遺伝子と呼ばれうるかどうかは、集団中にそれらに代わりうる別の遺伝子が存在するかどうかにかかっているということだ（強調は本書の筆者が付け加えたもの）。*A* を黒い色素の分子を作ることに結びつける因果の鎖は短く、一方、*B* のその因果の鎖は長くて込み入っているが、そのことは、ここでの議論には関係がない」

最後にドーキンスは、自然選択は差異に関することだと指摘する。あるバージョンの遺伝子が他のバージョンよりも普及してありふれた存在になるのは、それぞれのバージョンごとに適応度に差があるからなのだ、と。進化は比較を要求するのである。

どんな原因もそうであるように、遺伝的原因は「違いを生むもの」であり、そのことは、代わりになりうる別の何かとの比較に関係している。その点がよく理解されていないということが、哲学者ネッド・ブロックによる、今日なお広く引用される論証の大きなひとつの欠陥である。ブロックはこう書いた（以下の引用中、強調は本書の筆者が付け加えたもの）。「遺伝的決定［genetic determination］は、ある特徴を引き起こすものは何かに関する問題である。われわれの足の指が５本になるのは、われわれの遺伝子がその特徴を引き起こしているからであり、それゆえ、われわれの足の指の本数は、遺伝的に決定されている。それとは対照的に、遺伝率［heritability］は、ある特徴における違いを引き起こすものは何かに関する問題である。足の指が何本あるかの遺伝率は、遺伝的な差異が足の指の本数における多様性を引き起こす程度の問題なのである（猫の中には、指の数が５本のものと、６本のものがいる）」。ブロックの議論の誤りは、すぐに理解できるだろう。ある特徴を引き起こすものは、定義により、ある特徴における違いを生むものである。「遺伝子 *G1* はわれわれの足の指を５本にする」と述べることは、*G1* の代わりになりうる別のアレルと、５本指という表現型の代わりになりうる別の表現型が存在するということを意味する――*G1* ではない別の遺伝子を持つことが、あなたの足の指の本数を５本ではなくするのである。

実際、遺伝子は「違いを生むもの」だということは、ブロックが用いた、われわれの足の指は５本だという、まさにその例の場合に経験的に示すことができる。足の指の本数を決定する遺伝子の中のふたつに、*EVC1* と *EVC2* がある。これらふたつの遺伝子に起こる稀な突然変異が、多指症のほか、四肢短縮、歯の異常、心疾患などをともなう、エリス-ファンクレフェルト症候群として知られる症状を引き起こす。*EVC1* 遺伝子と *EVC2* 遺伝子は、細胞を取り巻くように生えている繊毛に見られるタンパク質をコードしており、このタンパク質は、細胞同士が正しい形状に並ぶためにコミュニケーションを取るのを助けている。*EVC1* と *EVC2* は、家族の中に多指症のメンバーがいたアーミッシュの

九家族を調べた研究によって発見された。その科学者たちが焦点を合わせたのは、ブロックが誤って遺伝的因果の問題とは別のものだとした、まさにその問いだった。この研究者たちは、「人が手に５本、足に５本の指を持つかどうかの差異に関連する遺伝子はどれだろうか？」と問うたのである。*EVC1* 遺伝子または *EVC2* 遺伝子に突然変異が起こったバージョンの遺伝子をふたつ受け継いだ人たちは、足の指の本数が５より多かった。その人たちは、５本の指を持たなかったのである。

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The Interpretation of Cultures（New York: Basic Books, 1973）, https://philpapers.org/archive/geettd.pdf. 私はここで用いた「薄い」「厚い」という言葉遣いと、ギアーツが行動を記述するためにこれらの言葉を使ったときの区別の仕方との類似性を指摘してくれたベンジャミン・ドミングに感謝する。たとえば、ギアーツは、行動に関する次のふたつの記述を区別した。「右の眼のまぶたを収縮させる」（薄い記述）と、「本当は悪だくみなどしていないのに何かひそかに陰謀をたくらんでいるかのように皆に思わせるような、偽のめくばせをする練習をする」（厚い記述）。［邦訳『文化の解釈学』（全２巻）Ｃ・ギアーツ著、吉田禎吾、柳川啓一、中牧弘允、板橋作美訳　岩波現代選書（第一部第一章「厚い記述──文化の解釈学的理論をめざして」）（1987 年）］

第六章

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9. それとは別の異議もある。あらゆるものには遺伝性があるのだから、これらの形質に遺伝性があるからといって問題にはならないというのがそれだ。

つまり、集団内に差異があって測定可能な形質はどれも、遺伝性のある多様性が存在する根拠になるというのだ。この主張は、どれだけテレビを見るかとか、どれだけマー

マイト［イギリス人が好んで食べるイーストエキス食品］を好むかといった馬鹿馬鹿しい形質にまで拡張される。こういう馬鹿馬鹿しい例は、遺伝的な因果関係の存在は、生物学的決定論にもとづくメカニズムが存在することを証拠立てているという直観に対抗するためには役に立つ──この点については最後の章であらためて論じよう。われわれは、マーマイトを好むことや、テレビをよく見ることを、「ゲノムレベルで」理解しようとしているのではない。しかし、われわれがマーマイトを好むという形質の遺伝率を気にしないのは、遺伝率が役にも立たない「象徴的」統計量だからではなく、人々がマーマイトを好むかどうかは重要ではないと思っているからだ。だがわれわれは、人々が大学を卒業するかどうかは重要だと考える。遺伝率という統計量の科学的・哲学的重要性は、問題にしている表現型の科学的・哲学的重要性から導かれるのである。Eric Turkheimer, “Three Laws of Behavior Genetics and What They Mean,” *Current Directions in Psychological Science* 9, no. 5（October 1,2000）, 160-64, https://journals.sagepub.com/doi/10.1111/1467-8721.00084.

10. 遺伝率と遺伝的因果関係との結びつきは、農業の選択育種プログラムでは、遺伝率係数がどのように利用されているかを考えれば、さらにはっきりする。いわゆる「育種家の方程式」は、$R=h^2 \times S$ で与えられる。ここで h^2 は、その集団の遺伝率係数［狭義の遺伝率］、R は選択に対する応答（選択された親から生まれた子の表現型値の平均と、もともとの集団平均との差）、S は選択差（選択された親の表現型値と、選択前の集団平均との差）である。

2019 年には、アメリカの男性の平均身長は 176 センチメートルである。さて、ディストピア的な独裁者が、子どもの親になることを許される男の身長に制限を課した結果、選択された父親たちの平均身長は、183 センチメートルになったと仮定しよう。子を作るために選択された男性の平均身長と、集団内の平均身長との差は 7 センチメートルである［S=7］。母親も同程度の選択を受けるとすると、他の環境はすべて厳密に前と同じだと仮定して、選択がある場合の次世代男子の平均身長は、選択がない場合と比べてどれだけ高くなるだろうか？　本章のはじめに説明した研究によれば、身長の遺伝率は 0.80［$h^2=0.80$］と推定される。これは 1.0 ではないため、次世代男子の身長が、平均として 7 センチメートル高くなることはないだろう。とはいえ遺伝率は高いので、子を作るために選択された親から生まれた子は、実際にかなり背が高くなるだろう──平均すると 6 センチほど高くなる［$R=0.80\times7$］。集団の平均値が変われば、「極端な」ケースが観察される頻度に影響が及ぶ。平均身長が 175 センチメートルの集団では、男性の約 1 パーセントは身長が 198 センチメートルよりも高い。平均身長がそれより 5 センチメートル高くなって 180 センチメートルになれば、身長が 198 センチメートル以上の男性の割合は 4 パーセントに増える。

遺伝率は、選択の応答を決定するものなので、因果の観点からも重要になる。それを理解するためには、「介入主義的因果理論」の枠組みで考えてみるといい。前章で記述

した「反事実的依存性」によって他の因果理論とつながる介入主義的因果理論だが、この理論は、「もしもXが起こらなかったなら、Yには何が起こっただろうか？」という問いを中核に据えるのではなく、「もしもあなたがXを変化させたら、Yには何が起こるだろうか？」という問いを中核に据える。哲学者のジム・ウッドワード は、著書『ものごとを実現させる（Making Things Happen）』の中で、その点について次のように述べた。「XはYの原因であるという主張は、少なくともある人たちにとっては、その人たちが所有するXのなんらかの値に対する可能な介入が存在し、その介入操作は、他の適切な条件が与えられたとして（その条件には、Xではないなんらかの変数を特定の値に固定するような操作が含まれるだろう）、Yの値、ないしYの確率分布を、変化させるだろうということを意味する」。（p.40）

選択実験は、この要請に対する興味深いひとひねりである。遺伝子（X）は表現型（Y）の原因であるという主張は、少なくともある人たちにとっては、その人たちが持っているXのなんらかの値に対し、可能な操作が存在するということを意味する。選択実験の場合であれば、その操作は、子を作ることを許される遺伝型の範囲に制限を課すことだ。Xとは異なる他の変数（すなわち環境変数）をなんらかの値に固定することを含めて、この操作は、選択された人々の子の表現型Yの確率分布を変化させるだろう。

もしも選択実験が表現型に対する遺伝子の因果的力を明らかにし、遺伝率は選択への応答を決定するのなら、遺伝率はいかなる理由によってであれ、因果には関与しないと結論することは不可能だ。ピーター・フィッセルが別の論文で述べたように、「遺伝率は、遺伝学における基本的なパラメータであり…… 進化生物学と農業における選択にとっても、医療における疾病リスクを予測するうえでも、カギとなる重要なパラメータである」James Woodward, *Making Things Happen: A Theory of Causal Explanation*, Oxford Studies in Philosophy of Science（Oxford: Oxford University Press, 2003）; Peter M. Visscher, William G. Hill, and Naomi R. Wray, “Heritability in the Genomics Era—Concepts and Misconceptions,” *Nature Reviews Genetics* 9, no. 4（April 2008）: 255-66, https://doi.org/10.1038/nrg2322.

11. 等環境仮説は、これまで多くの精査を受けてきた主題であり、ＤＮＡの測定記述を利用した新しいタイプの研究は、おおむねこの仮定を支持する結果を出している。ある注目すべき研究では、親、小児科医、そして双子たち自身でさえもが、しばしば一卵性か二卵性か間違えるという事実が利用された。つまり、実際は二卵性なのに一卵性だと思い込んだり、一卵性なのに二卵性だと思い込んだりするのだ。オランダの 300 組の双子に関する研究では、親が双子が一卵性か二卵性かを取り違えているケースが 19 パーセントあった。私自身がテキサスで行った双子研究でも同様の結果が得られた。一組の双子に一度会っただけの大学生のほうが、その双子たちの親より、ＤＮＡ検査の結果を正しく推測したのだ。社会学者のダルトン・コンリーと彼の同僚たちは、この双子の親たちのバイアスを切り札に、等環境仮説を検証した。一卵性双生児の親のほうが子どもをより等しく扱うこ

とが、一卵性双生児のほうが二卵性双生児よりも類似性が高い理由ならば（等環境仮説が破れている）、本当は二卵性の双子なのに一卵性だと思われていた双子は、正しく二卵性とされていた双子のペアよりも類似性が高いはずだと論じたのだ。実際、コンリーはそういう結果が得られると予想していた。行動遺伝学の研究結果は恐怖と呪いの眼で見るように訓練された社会学者にとって、この実験のデザインは、遺伝子は社会的不平等を理解するうえで重要だという、着実に積み上げられてきたエビデンスを突き崩すかに見えた。ところがそうはならなかったのだ！ コンリーらの研究は、双子の表現型の類似性（双子の人生の成り行きがどれぐらい似ているか）は、実際の遺伝上の関係性に沿い、親の判断には沿わないことが明らかになったのだ —— これは、等環境仮説を支持する根拠である。Dalton Conley et al.,“Heritability and the Equal Environments Assumption: Evidence from Multiple Samples of Misclassified Twins,” *Behavior Genetics* 43, no.5（September 2013）:415-26, https://doi.org/10.1007/s10519-013-9602-1.

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21. Rosa Cheesman et al., “Comparison of Adopted and Nonadopted Individuals Reveals Gene-Environment Interplay for Education in the UK Biobank,” *Psychological Science* 31, no.5（May 1, 2020）：582-91, https://doi.org/10.1177/0956797620904450.

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23. Theodosius Dobzhansky, “Genetics and Equality: Equality of Opportunity Makes the Genetic Diversity Among Men Meaningful,” *Science* 137, no. 3524 （July 13, 1962）：112-15, https://doi.org/10.1126/science.137.3524.112.

第七章

1. Christopher Jencks et al., *Inequality: A Reassessment of the Effect of Family and Schooling in America*（New York: Basic Books, 1972）.［邦訳『不平等──学業成績を左右するものは何か』Ｃ・ジェンクスほか著、橋爪貞雄、高木正太郎訳 黎明書房（1978年)］

2. 複雑な人間行動は、長い因果の鎖で遺伝型に結びついている、唯一の表現型ではない。進化生物学者のリチャード・ドーキンスが論じたように、「どんな表現型であれ――形態学的なものであれ、生理学的なものであれ、行動学的なものであれ――より基本的な何かの『副産物』ではないなどということがありうるものだろうか？ この問題を突き詰めて考えてみれば、タンパク質分子を別にすれば、すべての遺伝的効果は『副産物』であることがわかるのだ」同様に、一見するとシンプルに思われる環境的介入でさえ、その介入が効果を及ぼすためには、仲間の規範や教師の影響など、複雑な社会的プロセスに深く絡み合った長い因果の鎖が必要になるのは明らかだろう。Richard Dawkins, *The Extended Phenotype: The Long Reach of the Gene*, rev. ed.（Oxford and New York: Oxford University Press, 1999）［邦訳『延長された表現型――自然淘汰の単位としての遺伝子』Ｒ・ドーキンス著、日高敏隆、遠藤彰、遠藤知二訳 紀伊國屋書店（1987 年)］

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5. Francis Galton, *Hereditary Genius: An Inquiry into Its Laws and Consequences*（London and New York: Macmillan, 1892）.

6. Charles Murray, *Human Diversity: The Biology of Gender, Race, and Class* （New

York: Twelve, 2020）.

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9. 未知のメカニズムがありうるということ、それもおそらくは、直観的には捉えられないような何かを介して作用するメカニズムがありうるということは、遺伝的原因だけに限った問題ではないということを思い出しておくことは重要だ。実際、未知のメカニズムがありうるという問題は、ランダム化比較実験（RTC）から発する因果推論なら、どんなものにでもありうることなのだ。ノーベル経済学賞受賞者のアンガス・ディートンと科学哲学者のナンシー・カートライトは、「他の多くの仕事――経験的、理論的、概念的な仕事――は、ＲＴＣの結果を利用できるようなかたちで行われる必要がある」と述べた。制御された一組の条件の下で、ある方法で介入すれば、ある平均的な治療効果が得られることはわかるかもしれないが、その境界条件はどういったものだろうか？ 介入と、最終的な成り行き（アウトカム）とは、どんな因果的事象の鎖でつながれているのだろうか？ 人々は介入に対し、どんな応答をするだろうか？ 要するに、自然によるランダム化を利用して、ひとつの成り行きに関するひと組の遺伝的バリアントの、平均としての治療効果を検証するだけでは不十分なのだ。その因果推論の結果を、科学的で有用なものにするためには、経験的、理論的、概念的な面で、まだまだ多くの仕事をしなければならない。Angus Deaton and Nancy Cartwright, "Understanding and Misunderstanding Randomized Controlled Trials," *Social Science & Medicine* 210, special issue: Randomized Controlled Trials and Evidence-based Policy: A Multidisciplinary Dialogue（August 2018）: 2-21, https://doi.org/10.1016/j.socscimed.2017.12.005.

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Translate Discoveries from Genome-Wide Association Studies from the Top Down," *Current Directions in Psychological Science* 28, no.1（February 1, 2019）: 82-90, https: //doi. org/10. 1177/0963721418807729; J. Wertz et al., "Genetics and Crime: Integrating New Genomic Discoveries into Psychological Research About Antisocial Behavior," *Psychological Science* 29, no. 5（May 1, 2018）: 791-803, https://doi.org/10.1177/0956797617744542; Daniel W.Belsky et al., "The Genetics of Success: How Single-Nucleotide Polymorphisms Associated with Educational Attainment Relate to Life-Course Development,"*Psychological Science* 27, no. 7（July 1, 2016）: 957-72; Emily Smith-Woolley et al., "Differences in Exam Performance Between Pupils Attending Selective and Non-Selective Schools Mirror the Genetic Differences Between Them," *Npj Science of Learning* 3（March 2018）: 3, https: //www. nature. com/articles/s41539-018-0019-8; Eveline L. de Zeeuw et al., "Polygenic Scores Associated with Educational Attainment in Adults Predict Educational Achievement and ADHD Symptoms in Children," *American Journal of Medical Genetics Part B: Neuropsychiatric Genetics* 165, no. 6（September 2014）, 510-20, https: //onlinelibrary. wiley. com/doi/full/10. 1002/ajmg. b. 32254; Robert Plomin and Sophie von Stumm, "The New Genetics of Intelligence," *Nature Reviews Genetics* 19, no. 3（March 2018）: 148-59, https://doi.org/10.1038/nrg.2017.104; Andrea G. Allegrini et al., "Genomic Prediction of Cognitive Traits in Childhood and Adolescence," *Molecular Psychiatry* 24, no. 6（June 2019）: 819-27, https: //www.nature.com/articles/s41380-019-0394-4.

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14. 双子研究へのよくある批判に、このタイプの研究には、恵まれない家庭が十分含まれていないため、同じ家の子どもたちに共通の環境要因が、人生の成り行きのばらつきに寄与する程度を、過小評価してしまう可能性があるというものがある。遺伝率は、比で与えられることを思い出そう。サンプル中の環境の多様性が大きければ大きいほど分母が大きくなり、遺伝率は小さくなる。しかし、「テキサス双子プロジェクト」［著者がテキサス大学で運営にかかわっている心理学研究プロジェクト］の場合、われわれのサンプルは、かなり幅広くさまざまな環境を代表している。参加者の３分の１は、子どもが生まれて以降、公的補助（たとえばＳＮＡＰ、すなわち食料品を購入するための助成など）を受けている家庭だ。われわれはまた、そのサンプルについてジニ係数（収入の不平等の目安）を計算してみた。そうして得られたジニ係数は 0.35 で、アメリカ全体の指数 0.39 と比較すると、われわれはまあまあ妥当な仕事をしていると言えるだろう。とりわけ、われわれのサンプルは、アメリカ社会を特徴づける大きな所得格差のパターンを捉えるという点では、地理的制約を受けていることを考慮すればなおさらだ。

われわれのサンプルが多様性に富んでいることは重要である。なぜならそれは、実行機能の遺伝率が非常に高いのは、恵まれた家庭の子どもたちだけを見ているからではないということを意味しているからだ。また、われわれのものとは独立に、心理学者ナオミ・フリードマンが運営するコロラド大学の研究室は、検証された時点でわれわれのサンプルよりも年上だった別の双子のサンプルから、ほぼ 100 パーセントの遺伝率という同じ結果を得ている。Naomi P. Friedman et al., "Individual Differences in Executive Functions Are Almost Entirely Genetic in Origin," *Journal of Experimental Psychology*: General 137, no. 2（May 2008）: 201-25, https://doi.org/10.1037/0096-3445.137.2.201.

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どんな見解をとろうとも、『遺伝的』という言葉を挿入したところでなんら違いは生じない。あなたが完全な決定論者なら、あなたのすべての行為は過去の物理的原因によって事前に決定されていると考えるだろう。そして、あらかじめ決定されていたのだから、あなたは自分の不貞に対する責任を負うことはできないと考えるかもしれないし、考えないかもしれない。しかしいずれにせよ、その物理的原因のうちのいくつかが遺伝的だからといって、何が違うというのだろう？ なぜ、遺伝的決定論が、『環境的』決定論以上に、不可避的なもの、もしくは責を問われないものだと考えられるのだろう？」Richard Dawkins, *The Extended Phenotype: The Long Reach of the Gene*, rev. ed.（Oxford and New York: Oxford University Press, 1999）.［邦訳『延長された表現型――自然淘汰の単位としての遺伝子』Ｒ・ドーキンス著、日高敏隆、遠藤彰、遠藤知二訳 紀伊國屋書店（1987 年）］

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