THE EL-RAKHAWI LAW OF TRANSGENERATIONAL EPIGENETICS AND BIOLOGICAL GENERATIONAL

JUSTICE
Legislative Frameworks for Cross-Generational Environmental Liability in the Age of Molecular Continuity

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Dedication
To the unborn generations who inherit the biological consequences of present-day environmental decisions
To every legislator who recognizes that justice must extend beyond the present moment
To scientists who unveiled the molecular memory of chemical and environmental exposure
To future communities who deserve a legal system that honors biological continuity and protects intergenerational equity

PREFACE

The discovery that environmental exposures can leave molecular marks on the genome without altering the underlying DNA sequence has fundamentally transformed our understanding of heredity, disease, and environmental harm. Epigenetics reveals that chemical exposures, nutritional deprivation, and chronic stress can modify gene expression through DNA methylation, histone modification, and non-coding RNA regulation. Crucially, mounting evidence demonstrates that certain epigenetic alterations can be transmitted across multiple generations, predisposing descendants to metabolic disorders, neurodevelopmental impairments, immune dysfunction, and increased cancer susceptibility.

This biological reality challenges the foundational assumptions of contemporary tort and environmental law, which operate on the premise that harm is immediate, directly observable, and confined to exposed individuals. Current legal frameworks lack mechanisms to recognize, quantify, or compensate for transgenerational epigenetic injury. Statutes of limitations expire before latent heritable effects manifest. Causation standards require direct linear proof that

cannot capture probabilistic, multi-generational biological pathways. Environmental licensing processes ignore epigenetic disruptors, focusing instead on acute toxicity and visible ecological damage. Consequently, communities exposed to industrial pollutants, agricultural chemicals, and contaminated water systems bear silent biological costs that cascade into future generations without legal recourse.

This book establishes a foundational legal framework that bridges epigenetic science, toxicology, epidemiology, and legislative practice. It introduces the concept of Biological Generational Justice, mandates Epigenetic Risk Assessments, and provides model legislative texts for liability recognition, compensatory funding, and regulatory thresholds. The methodology combines rigorous molecular science with precise legal drafting, ensuring that the proposed framework is academically sound, practically enforceable, and ready for legislative adoption. By aligning legal structures with the biological reality of transgenerational inheritance, this work seeks to transform environmental law from a reactive system into a proactive guardian of intergenerational biological equity.

CHAPTER ONE
THE LAW OF TRANSGENERATIONAL EPIGENETICS AND BIOLOGICAL GENERATIONAL JUSTICE
Recognizing Cross-Generational Biological Liability

SECTION ONE: SCIENTIFIC INTRODUCTION

Epigenetics refers to heritable changes in gene expression that occur without modifications to the underlying DNA sequence. The primary mechanisms include DNA methylation, which typically suppresses gene activity by adding methyl groups to cytosine bases; histone modification, which alters chromatin structure to regulate transcriptional accessibility; and non-coding RNA regulation, which fine-tunes gene silencing and expression post-transcriptionally. These mechanisms respond dynamically to environmental stimuli, acting as biological interfaces between external exposures and genomic function.

Transgenerational epigenetic inheritance occurs when exposure-induced epigenetic marks escape embryonic reprogramming and are transmitted through germ cells to subsequent generations. Animal studies and emerging human epidemiological data confirm that prenatal exposure to endocrine disruptors, heavy metals, persistent organic pollutants, and severe nutritional stress can alter methylation patterns in offspring and persist into third and fourth generations. These alterations correlate with increased incidence of obesity, diabetes, cardiovascular disease, neurodevelopmental disorders, and reproductive dysfunction.

The scientific consensus establishes that environmental toxicity is not confined to the directly exposed individual. Epigenetic dysregulation creates a biological debt that accumulates across generations. Chronic disease epidemiology now recognizes that population-level health disparities often reflect historical exposure patterns transmitted through epigenetic pathways. This reality demands a legal paradigm that acknowledges biological continuity, recognizes latent

heritable harm, and establishes accountability for exposures that compromise future generations.

SECTION TWO: THE CURRENT LEGAL GAP

Contemporary tort and environmental law operate on temporal and causal frameworks incompatible with epigenetic science. Liability requires proof of direct causation, immediate injury, and identifiable victimhood within statutory timeframes. Environmental regulations assess acute toxicity, carcinogenic potential, and ecosystem disruption but exclude epigenetic endpoints from risk evaluation. Licensing processes for industrial facilities, agricultural chemicals, and consumer products do not require assessment of transgenerational biological impact.

Critical legal deficiencies include the absence of statutory recognition for epigenetic inheritance as a compensable injury, lack of long-term biological monitoring mandates for exposed communities, prohibition of class-action mechanisms spanning multiple generations due to standing and causation barriers, and exclusion of epigenetic biomarkers from environmental impact reporting requirements. Furthermore, current compensation models treat each generation as legally independent, ignoring the cumulative biological burden transmitted through germ lines. The result is a legal architecture that normalizes intergenerational biological harm, treating epigenetic inheritance as an unaddressable scientific phenomenon rather than a compensable legal injury.

SECTION THREE: FOUNDATIONAL PRINCIPLES PROPOSED

Principle One: Recognition of Transgenerational Epigenetic Harm

Epigenetic alterations transmitted across generations due to environmental or industrial exposure shall be legally recognized as compensable biological injury. Harm extends beyond immediate exposure cohorts to include descendants demonstrating documented epigenetic dysregulation.

Principle Two: Establishment of Generational Compensation Funds

Public and private entities responsible for epigenetic disruptor releases shall contribute to dedicated compensation funds. These funds shall finance long-term health monitoring, medical intervention, and restitution for affected multi-generational populations.

Principle Three: Regulatory Thresholds for Epigenetic Disruptors

Chemicals and industrial processes shall be evaluated for epigenetic toxicity prior to market approval or environmental release. Substances demonstrating transgenerational epigenetic alteration in validated models shall be subject to strict exposure limits, substitution requirements, or phased elimination.

Principle Four: Mandatory Long-Term Epigenetic Impact Assessment

Environmental licensing shall require longitudinal biological monitoring of exposed communities. Epigenetic biomarker tracking shall become a standard component of post-approval compliance verification.

Principle Five: Principle of Biological Generational Justice
Legal liability shall account for cumulative biological burden across generations. Compensation mechanisms, remediation obligations, and preventive regulations shall reflect the intergenerational nature of epigenetic harm.

SECTION FOUR: MODEL LEGISLATIVE TEXTS

Article One: Definition of Transgenerational Epigenetic Injury
Transgenerational Epigenetic Injury refers to heritable alterations in gene expression patterns, including DNA methylation shifts, histone modifications, and non-coding RNA dysregulation, transmitted across generations as a result of environmental, industrial, or chemical exposure.

Article Two: Classification and Regulation of Epigenetic Disruptors
Any substance, process, or emission demonstrating capacity to induce transgenerational epigenetic alterations shall be classified as an Epigenetic Disruptor. Manufacturers, industrial operators, and agricultural entities shall submit epigenetic toxicity data prior to licensing. Approved substances shall comply with exposure thresholds established by the National Epigenetic Safety Authority.

Article Three: Generational Compensation Fund
A national Generational Compensation Fund shall be established to finance health monitoring, clinical intervention, and restitution for communities affected by documented epigenetic disruptor exposure. Funding shall be sourced through a tiered Epigenetic Risk Levy imposed on manufacturers, calibrated based on the chemical's classified epigenetic toxicity level, alongside environmental licensing fees, corporate compliance contributions, and penalties for non-compliance. Distribution shall prioritize multi-generational cohorts with verified epigenetic biomarker alterations.

Article Four: Long-Term Epigenetic Impact Assessment
All environmental permits for industrial facilities, chemical manufacturing, and large-scale agricultural operations shall require a Long-Term Epigenetic Impact Assessment. The assessment shall include baseline biomarker profiling, longitudinal cohort monitoring, epigenetic sequencing protocols, and five-year compliance reporting intervals.

Article Five: Liability and Causation Standards
Legal liability for transgenerational epigenetic injury shall be established through probabilistic causation standards when direct linear proof is scientifically unattainable. For the purpose of this Article, probabilistic causation shall be satisfied when the plaintiff demonstrates, through peer-reviewed epidemiological or toxicogenomic evidence, that the exposure more than doubles the baseline risk (Relative Risk greater than 2.0) of the specific epigenetic alteration and

subsequent health outcome. Plaintiffs demonstrating documented exposure history, generational health impact correlation, and validated epigenetic biomarker shifts shall meet evidentiary thresholds for compensatory claims.

Article Six: Oversight and Scientific Review Board
An independent Scientific Review Board comprising molecular biologists, toxicologists, epidemiologists, and legal scholars shall evaluate epigenetic risk assessments, verify biomarker data, and advise regulatory authorities. The Board shall publish annual reports on emerging epigenetic disruptors, monitoring efficacy, and compensation fund allocation.

Article Seven: Retroactive Application and Historical Claims
Recognizing the latent nature of epigenetic harm, this Act shall establish a Historical Claims Window of ten years from the date of enactment. During this period, descendants of communities exposed to classified Epigenetic Disruptors prior to enactment may file claims for collective public health interventions through the Generational Compensation Fund, provided that historical exposure and multi-generational health impacts are documented.

SECTION FIVE: MECHANISM OF APPLICATION AND EVIDENCE

Implementation requires integration of molecular diagnostics with legal compliance frameworks. Key mechanisms include standardized epigenetic sequencing protocols for exposed populations, establishment of national biomarker registries with strict privacy safeguards, longitudinal cohort tracking across generations, and certified laboratory networks for independent verification. Property and industrial licensing will incorporate epigenetic compliance as a mandatory precondition for operational authorization.

Evidence of injury or compliance relies on several pillars. First, validated epigenetic biomarker profiles demonstrating methylation shifts, histone modification patterns, or non-coding RNA dysregulation correlated with documented exposure. Second, multi-generational health records establishing statistical correlation between exposure events and chronic disease incidence in descendants. Third, expert testimony from accredited molecular geneticists, epidemiologists, and environmental toxicologists regarding causation probability and biological plausibility. Fourth, chain-of-custody documentation for biological samples, laboratory accreditation certificates, and peer-reviewed validation of sequencing methodologies.

To reconcile evidentiary requirements with privacy mandates, biological sample analysis for legal claims shall be conducted via a Trusted Third-Party Escrow protocol. Certified laboratories shall perform blinded biomarker analysis, reporting only the presence or absence of chemical-specific epigenetic signatures to the legal parties, thereby preventing the misuse of raw genetic or epigenetic data. Data protection mandates ensure that genetic and epigenetic information is used exclusively for compensation, monitoring, and regulatory compliance, with explicit prohibitions against insurance discrimination or employment profiling.

Enforcement operates through a graduated compliance structure. Initial deviations trigger mandatory corrective monitoring, revised exposure controls, and independent laboratory verification. Repeated or severe violations result in administrative penalties, operational license suspension, corporate liability assignments, and mandatory contributions to the Generational Compensation Fund. The framework emphasizes preventive biomarker tracking and intergenerational health protection over reactive litigation, incentivizing industries to adopt epigenetic safety standards proactively.

SECTION SIX: REAL-WORLD CASE STUDY

The Dutch Hunger Winter of 1944 to 1945 provides a foundational epidemiological demonstration of transgenerational epigenetic impact. Prenatal exposure to severe famine resulted in altered DNA methylation patterns in offspring, correlating with increased incidence of metabolic syndrome, cardiovascular disease, and neuropsychiatric disorders in subsequent generations. Under current legal frameworks, no compensatory mechanism exists for descendants bearing these biological consequences, as harm occurred decades after exposure and causation cannot meet traditional tort standards.

Similarly, communities exposed to diethylstilbestrol (DES), a synthetic estrogen prescribed to pregnant women, experienced transgenerational reproductive abnormalities and increased cancer susceptibility. While some litigation succeeded for directly exposed individuals, descendants facing inherited epigenetic dysregulation remain excluded from compensation due to standing limitations and temporal barriers.

This legal framework transforms historical and ongoing biological injustices into actionable legal claims. By mandating epigenetic impact assessments, establishing generational compensation funds, and adopting probabilistic causation standards, it ensures that intergenerational biological harm receives statutory recognition and remedial pathways. The result is more equitable health outcomes, stronger regulatory oversight of chemical exposures, and a legal system that acknowledges the molecular continuity of human generations.

CONCLUSION

The Law of Transgenerational Epigenetics and Biological Generational Justice represents a necessary evolution in environmental jurisprudence and tort liability. Scientific evidence unequivocally demonstrates that chemical and environmental exposures can alter gene expression patterns and transmit biological vulnerability across generations. Yet, legal systems have historically confined liability to immediate victims, ignoring the molecular inheritance of harm. The proposed framework corrects this oversight by recognizing epigenetic injury, mandating long-term biomarker monitoring, establishing generational compensation mechanisms, and integrating epigenetic thresholds into regulatory standards.

Adopting this legal architecture will not only provide restitution for historically affected communities but also prevent future intergenerational biological harm through proactive

chemical regulation and industrial accountability. It transforms environmental law from a discipline focused on present harm into a guardian of future biological equity. As molecular science continues to reveal the heritable consequences of environmental exposure, the law must evolve to reflect biological reality. This framework provides the legislative tools to achieve that synchronization, ensuring that justice extends across time and protects the molecular heritage of generations yet to come.

COMPREHENSIVE APPENDICES

APPENDIX A: DETAILED EXPLANATION OF EPIGENETIC MECHANISMS

DNA Methylation: DNA methylation involves the addition of a methyl group to the fifth carbon of cytosine bases, typically at CpG dinucleotide sites. This modification generally suppresses gene expression by preventing transcription factor binding or recruiting repressive chromatin modifiers. Environmental exposures can alter methylation patterns at specific loci, with some changes persisting through germ cell transmission.

Histone Modification: Histones are proteins around which DNA is wrapped to form chromatin. Chemical modifications to histone tails, including acetylation, methylation, phosphorylation, and ubiquitination, alter chromatin structure and regulate gene accessibility. Environmental factors can induce histone modifications that affect gene expression patterns across generations.

Non-Coding RNA Regulation: Non-coding RNAs, including microRNAs and long non-coding RNAs, regulate gene expression post-transcriptionally by targeting messenger RNAs for degradation or translational repression. Environmental exposures can alter non-coding RNA profiles, with some changes transmissible through germ cells.

APPENDIX B: CHEMICAL-SPECIFIC EPIGENETIC BIOMARKERS

The framework requires identification of epigenetic alterations at specific genomic loci validated as responsive to particular chemical agents. Examples include: Bisphenol A (BPA) causing altered methylation at estrogen receptor-related loci (ESR1, ESR2); Diethylstilbestrol (DES) causing methylation changes at WNT7A and reproductive development genes; Heavy Metals (Lead, Cadmium) causing methylation alterations at neurodevelopmental genes (BDNF, REELIN); Persistent Organic Pollutants causing methylation changes at metabolic regulation genes (IGF2, LEP, ADIPOQ). Validation requires reproducible demonstration in both animal models and human epidemiological studies.

APPENDIX C: TOXICOGENOMIC MOLECULAR CHAIN OF CUSTODY

Toxicogenomics integrates toxicology with genomics to establish molecular mechanisms linking chemical exposure to adverse outcomes. The molecular chain of custody requires demonstration of: Chemical Exposure (documented presence); Cellular Uptake (absorption and distribution); Receptor Binding (interaction with specific cellular targets); Signal Transduction (activation of intracellular pathways); Epigenetic Modification (specific alterations at validated loci); Gene Expression Changes (altered transcription); Phenotypic Expression (observable disease); Transgenerational Transmission (persistence through germ cells).

APPENDIX D: MULTI-MARKER PANEL METHODOLOGY

Single epigenetic biomarkers may lack specificity due to confounding lifestyle factors. Multi-marker panels increase diagnostic accuracy by requiring simultaneous alterations at multiple loci associated with the specific chemical exposure. Quality control measures include demographically matched control groups, adjustment for lifestyle confounders (smoking, diet, stress), blinded laboratory analysis, independent replication, and regular proficiency testing.

APPENDIX E: INDUSTRY POOLED RISK FUND MECHANICS
The Generational Compensation Fund operates similarly to environmental superfund mechanisms. Funding sources include a tiered Epigenetic Risk Levy calibrated to toxicity potential, licensing fees, penalties, and investment returns. Governance includes an independent board, scientific advisory committee, claims review panel, and audit division. Compensation categories include individual compensation for F1/F2 generations, collective public health funding for F3+ generations, medical monitoring, and research funding.

APPENDIX F: DATA PROTECTION AND TRUSTED THIRD-PARTY ESCROW
Epigenetic and genetic information creates potential for discrimination. The framework mandates a Trusted Third-Party Escrow protocol where certified laboratories perform blinded biomarker analysis, reporting only the presence or absence of chemical-specific signatures to legal parties. Prohibited uses include insurance underwriting, employment decisions, educational admissions, and housing. Enforcement includes civil and criminal penalties, private right of action, and mandatory breach notification.

APPENDIX G: RETROACTIVE APPLICATION AND HISTORICAL CLAIMS MECHANICS
The Historical Claims Window allows descendants of communities exposed to classified Epigenetic Disruptors prior to enactment to file claims for collective public health interventions within ten years of enactment. Documentation requirements include historical exposure records (e.g., occupational records, environmental monitoring archives) and multi-generational health impact correlation. This mechanism addresses the latency of epigenetic harm without imposing impossible direct causation burdens on historical victims.

APPENDIX H: COMPARATIVE ANALYSIS WITH EXISTING LEGAL FRAMEWORKS
United States Toxic Substances Control Act (TSCA): Regulates chemical manufacturing but excludes transgenerational epigenetic effects. The Elrakhawi Framework supplements TSCA by adding epigenetic toxicity assessment requirements.
European Union REACH Regulation: Focuses on acute and chronic toxicity. The framework provides model provisions for integrating epigenetic endpoints into REACH-style regulatory systems.
National Tort Law Systems: Traditional systems require direct causation and immediate injury. The framework provides model statutory reforms enabling probabilistic causation standards and generational standing.

APPENDIX I: BURDEN OF PROOF REVERSAL MECHANICS
In cases involving established Endocrine Disruptors or Persistent Organic Pollutants, the framework shifts the burden of proof to defendant manufacturers when plaintiffs demonstrate

documented exposure, validated biomarker alterations, and health outcomes consistent with toxicological profiles. Defendants must then demonstrate that observed alterations resulted exclusively from unrelated lifestyle factors or that exposure levels were below epigenetic effect thresholds.

APPENDIX J: TRANSITIONAL COMPLIANCE SCHEDULES
Existing industrial operations are granted transitional periods to achieve full compliance. Phase One (Years 1-3): Baseline assessment and initiation of epigenetic toxicity testing. Phase Two (Years 4-7): Implementation of exposure reduction measures and biomarker monitoring. Phase Three (Years 8-10): Full compliance, achievement of regulatory thresholds, and certification by the National Epigenetic Safety Authority. New operations are subject to immediate full compliance.

COMPREHENSIVE KEYWORDS IN TEN LANGUAGES

ENGLISH
Transgenerational Epigenetics, Biological Generational Justice, Epigenetic Inheritance, Environmental Liability, DNA Methylation, Histone Modification, Non-Coding RNA, Epigenetic Disruptors, Endocrine Disruptors, Persistent Organic Pollutants, Chemical-Specific Biomarkers, CpG Sites, Toxicogenomics, Molecular Chain of Custody, Multi-Marker Panels, Probabilistic Causation, Relative Risk, Generational Compensation Fund, Long-Term Epigenetic Impact Assessment, Retroactive Application, Trusted Third-Party Escrow, Epigenetic Risk Levy, Dutch Hunger Winter, Diethylstilbestrol, Intergenerational Harm, Epigenetic Reprogramming, Biomarker Validation, Environmental Epigenetic Law, Molecular Continuity, Epigenetic Toxicity

FRENCH
Epigénétique Transgénérationnelle, Justice Biologique Générationnelle, Héritage Épigénétique, Responsabilité Environnementale, Méthylation de l'ADN, Modification des Histones, ARN Non Codant, Perturbateurs Épigénétiques, Perturbateurs Endocriniens, Polluants Organiques Persistants, Biomarqueurs Spécifiques aux Produits Chimiques, Sites CpG, Toxicogénomique, Chaîne de Custodie Moléculaire, Panneaux Multi-Marqueurs, Causalité Probabiliste, Risque Relatif, Fonds d'Indemnisation Générationnelle, Évaluation d'Impact Épigénétique à Long Terme, Application Rétroactive, Tiers de Confiance Séquestre, Contribution au Risque Épigénétique, Hiver de la Faim Hollandais, Diéthylstilbestrol, Préjudice Intergénérationnel, Reprogrammation Épigénétique, Validation de Biomarqueurs, Droit Environnemental Épigénétique, Continuité Moléculaire, Toxicité Épigénétique

GERMAN
Transgenerationelle Epigenetik, Biologische Generationengerechtigkeit, Epigenetische Vererbung, Umwelthaftung, DNA-Methylierung, Histonmodifikation, Nicht-codierende RNA, Epigenetische Disruptoren, Endokrine Disruptoren, Persistente Organische Schadstoffe, Chemikalienspezifische Biomarker, CpG-Standorte, Toxikogenomik, Molekulare Beweiskette, Multi-Marker-Panels, Probabilistische Kausalität, Relatives Risiko, Generationen-Ausgleichsfonds, Langfristige Epigenetische Auswirkungsbewertung, Retroaktive Anwendung,

Treuhänderischer Escrow, Epigenetische Risikoabgabe, Holländischer Hungerwinter, Diethylstilbestrol, Intergenerationeller Schaden, Epigenetische Reprogrammierung, Biomarker-Validierung, Umwelt-Epigenetik-Recht, Molekulare Kontinuität, Epigenetische Toxizität

SPANISH
Epigenética Transgeneracional, Justicia Biológica Generacional, Herencia Epigenética, Responsabilidad Ambiental, Metilación del ADN, Modificación de Histonas, ARN No Codificante, Disruptores Epigenéticos, Disruptores Endocrinos, Contaminantes Orgánicos Persistentes, Biomarcadores Específicos de Productos Químicos, Sitios CpG, Toxicogenómica, Cadena de Custodia Molecular, Paneles Multi-Marcadores, Causalidad Probabilística, Riesgo Relativo, Fondo de Compensación Generacional, Evaluación de Impacto Epigenético a Largo Plazo, Aplicación Retroactiva, Depósito en Fideicomiso de Terceros, Gravamen de Riesgo Epigenético, Invierno del Hambre Holandés, Dietilestilbestrol, Daño Intergeneracional, Reprogramación Epigenética, Validación de Biomarcadores, Derecho Ambiental Epigenético, Continuidad Molecular, Toxicidad Epigenética

ITALIAN
Epigenetica Transgenerazionale, Giustizia Biologica Generazionale, Ereditarietà Epigenetica, Responsabilità Ambientale, Metilazione del DNA, Modificazione degli Istoni, RNA Non Codificante, Disruttori Epigenetici, Disruttori Endocrini, Inquinanti Organici Persistenti, Biomarcatori Specifici per Sostanze Chimiche, Siti CpG, Tossicogenomica, Catena di Custodia Molecolare, Pannelli Multi-Marcatori, Causalità Probabilistica, Rischio Relativo, Fondo di Compensazione Generazionale, Valutazione di Impatto Epigenetico a Lungo Termine, Applicazione Retroattiva, Deposito Fiduciario di Terzi, Contributo al Rischio Epigenetico, Inverno della Fame Olandese, Dietilstilbestrolo, Danno Intergenerazionale, Riprogrammazione Epigenetica, Validazione di Biomarcatori, Diritto Ambientale Epigenetico, Continuità Molecolare, Tossicità Epigenetica

PORTUGUESE
Epigenética Transgeracional, Justiça Biológica Geracional, Herança Epigenética, Responsabilidade Ambiental, Metilação do DNA, Modificação de Histonas, RNA Não Codificante, Disruptores Epigenéticos, Disruptores Endócrinos, Poluentes Orgânicos Persistentes, Biomarcadores Específicos de Produtos Químicos, Sítios CpG, Toxicogenômica, Cadeia de Custódia Molecular, Painéis Multi-Marcadores, Causalidade Probabilística, Risco Relativo, Fundo de Compensação Geracional, Avaliação de Impacto Epigenético de Longo Prazo, Aplicação Retroativa, Depósito em Escrow de Terceiros, Contribuição de Risco Epigenético, Inverno da Fome Holandês, Dietilestilbestrol, Dano Intergeracional, Reprogramação Epigenética, Validação de Biomarcadores, Direito Ambiental Epigenético, Continuidade Molecular, Toxicidade Epigenética

ARABIC
علم الوراثة اللاجينية عبر الأجيال، العدالة البيولوجية بين الأجيال، الوراثة اللاجينية، المسؤولية البيئية، مثيلة الحمض النووي، تعديل الهيستونات، الحمض النووي الريبي غير المشفر، المعطلات اللاجينية، معطلات الغدد الصماء، الملوثات العضوية الثابتة، المؤشرات الحيوية الخاصة بالمواد الكيميائية، مواقع CpG، علم السموم الجيني، سلسلة الحيازة الجزيئية، لوحات المؤشرات المتعددة، السببية

الاحتمالية، الخطر النسبي، صندوق التعويضات بين الأجيال، تقييم التأثير اللاجيني طويل الأمد، الأثر الرجعي، طرف ثالث موثوق، رسوم مخاطر لاجينية، شتاء الجوع الهولندي، ثنائي إيثيل ستيلبيسترول، الضرر بين الأجيال، إعادة البرمجة اللاجينية، التحقق من المؤشرات الحيوية، القانون البيئي اللاجيني، الاستمرارية الجزيئية، السمية اللاجينية

CHINESE
跨代表观遗传学，生物代际正义，表观遗传继承，环境责任，DNA 甲基化，组蛋白修饰，非编码 RNA，表观遗传干扰物，内分泌干扰物，持久性有机污染物，化学物质特异性生物标志物，CpG 位点，毒物基因组学，分子监管链，多标志物面板，概率因果关系，相对风险，代际补偿基金，长期表观遗传影响评估，追溯适用，可信第三方托管，表观遗传风险税，荷兰饥饿冬天，己烯雌酚，代际伤害，表观遗传重编程，生物标志物验证，环境表观遗传法，分子连续性，表观遗传毒性

JAPANESE
トランスジェネレーショナルエピジェネティクス，生物学的世代間正義，エピジェネティック遺伝，環境責任，DNA メチル化，ヒストン修飾，ノンコーディング RNA，エピジェネティック撹乱物質，内分泌撹乱物質，残留性有機汚染物質，化学物質特異的バイオマーカー，CpG サイト，トキシコゲノミクス，分子チェーン・オブ・カストディ，マルチマーカーパネル，確率的因果関係，相対リスク，世代間補償基金，長期的エピジェネティック影響評価，遡及適用，信頼できる第三者エスクロー，エピジェネティックリスク徴収，オランダ飢饉の冬，ジエチルスチルベストロール，世代間傷害，エピジェネティックリプログラミング，バイオマーカー検証，環境エピジェネティック法，分子連続性，エピジェネティック毒性

RUSSIAN
Трансгенная Эпигенетика, Биологическая Поколенческая Справедливость, Эпигенетическое Наследование, Экологическая Ответственность, Метилирование ДНК, Модификация Гистонов, Некодирующая РНК, Эпигенетические Дисрапторы, Эндокринные Дисрапторы, Стойкие Органические Загрязнители, Химически-Специфические Биомаркеры, CpG Сайты, Токсикогеномика, Молекулярная Цепочка Хранения, Мульти-Маркерные Панели, Вероятностная Причинность, Относительный Риск, Поколенческий Компенсационный Фонд, Долгосрочная Оценка Эпигенетического Воздействия, Ретроактивное Применение, Доверенный Третьей Стороной Эскроу, Эпигенетический Сбор за Риск, Голландская Голодная Зима, Диэтилстильбэстрол, Межпоколенческий Вред, Эпигенетическое Перепрограммирование, Валидация Биомаркеров, Экологическое Эпигенетическое Право, Молекулярная Непрерывность, Эпигенетическая Токсичность

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