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HOW DOES GENE EDITING TECHNOLOGY RESHAPE THE THEORETICAL COGNITIVE FRAMEWORK OF DISEASE MECHANISMS

Yichi Chen[1*]

[1]Nouvelle Academy of Shenzhen, China

postcode:518001

*Corresponding Author E-mail: CYC18320978684@outlook.com

Abstract

Gene-editing technologies such as CRISPR/Cas9, base editing, and prime editing have revolutionized the biomedical landscape by enabling precise, targeted alterations to the genome. However, beyond their biological utility, these technologies are also reshaping the theoretical cognitive frameworks through which disease mechanisms are conceptualized. This study employs a mixed-method experimental design to investigate how gene-editing interventions affect both molecular outcomes and the epistemological understanding of disease. Quantitative data derived from gene expression assays, editing efficiency measurements, and phenotypic observations were complemented by qualitative insights from expert interviews and clinician surveys. The results reveal that successful gene edits not only produce measurable biological effects—such as increased expression accuracy and phenotype improvement—but also prompt a paradigm shift in the perception of disease as a modifiable, network-regulated condition. Visual analytics including heatmaps, hybrid plots, and box plots further support these findings, illustrating multi-gene interactions and the complexity of post-editing phenotypic landscapes. Moreover, the study highlights critical ethical considerations, especially as editing transitions from lab-based research to clinical application. It emphasizes the importance of interdisciplinary frameworks that bridge molecular biology, clinical practice, ethics, and systems theory. Overall, this research affirms that gene-editing technologies are not only therapeutic tools but also epistemic instruments that redefine how disease is understood, managed, and anticipated in the era of genomic medicine.

Keywords: Gene Editing, CRISPR/Cas9, Disease Mechanisms, Cognitive Frameworks, Base Editing, Genome Engineering, Phenotypic Outcomes, Biomedical Epistemology, Molecular Therapeutics, Ethical Genomics, Systems Biology, Precision Medicine, Theoretical Reframing, Genetic Intervention, Clinical Genomics

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INTRODUCTION

Improvements in gene editing such as CRISPR/Cas 9, TALENs, and zinc finger nucleases (ZFNs) have altered the approach to mechanobiology of pathologies in the last decade. The technologies have revolutionised the technique that scientists use in simulating diseases, to create medicines and understand pathophysiology by precise modifications of genomic sequences with their cognitive systems. Z Li et al. (2020) overviewed the evolution and application of programmable nucleases -ZFNs, TALENs, and CRISPR/Cas9- and how they can be used to analyze the behavior of a particular gene in disease models and facilitate the transfer between theory and practice. Zhang, Li and Jin (2024) also discussed the possibility of applying the human gene-editing tool, Cas9gene, to develop improved models of animals and cells with neurological, cardiovascular, autoimmune and cancer diseases. It is an argument that better theoretical models can be made of how diseases work through genome editing. The potential for gene editing as therapeutic treatment is slowly altering theoretical models. Deneault (2024) emphasised that next-generation techniques can promise precise rectifications of harmful mutations and so transform the integration of mutational causality and targeted repair mechanisms into mechanistic frameworks (MDPI). Gene-edited induced pluripotent stem cells (iPSCs) have been utilised in conditions such as Alzheimer's disease in order to reduce amyloid-beta and tau clumps, improving cognitive function, and so impact molecular and cognitive frameworks of disease pathogenesis.

The advent of the RNA-targeting methodologies, such as LEAPER, enables a transformation of theoretical frameworks. The technique is also applied to effect changes on the post-transcriptional level that modify our concept of disease pathways. Modifications of the DNA and RNA have become very significant.

Innovative paradigms are helped through clinical milestones. One example is the pioneering use of genome-editing technology, called Cas-9 (or Crispr), on a living human to restore vision in people with congenital blindness, a paradigm shift of ex-vivo to in vivo real-time mechanistic intervention. This altered the thinking of intelligent individuals towards the thinking of intellectual models in therapy and experiments. Axios. It is also highly significant that the story of Victoria Grey, the first individual to undergo the use of the gene-editing tool CRISPR/Cas9 to cure sickle-cell disease, is presented . It is an applied achievement which must involve a reconsideration of illness: it can be repaired by the insertion of certain genes in order to remedy an already established failure, rather than merely attending to the symptoms.

Theoretical frameworks are also subject to the regulatory conditions. Sharaf-Eldin (2024) summarized methods and issues of gene editing, in which it is required to reconsider the aetiology and long-term safety of diseases, because the essence of gene editing technologies is irreversible. SpringerOpen. WHO has highlighted the importance of rules and regulations as far as it is concerned with the matters of changing genomes that can be passed through generation to another generation. This means that cognitive models ought to be more intergenerational, social and moral. World Health Organization In the same way, professional associations have raised an alarm against premature application of germline editing in advance of its accuracy being ensured. This favours a thoughtful, dynamic and adaptable theoretical position.

Some of the methods include CRISPR, base editing, and prime editing, which are altering the way epigenetic and genomic regulatory systems can be incorporated into theoretical models and whose accuracy and versatility are shifting the paradigm of meeting a particular pathogenic mutation as a complex interaction of genetic sequences, regulatory factors, and the cellular environment (Cetin, 2025). PMC; Xu (2025) is also of the same opinion, he feels that the systems and vectors are to change when people turn to the ScienceDirect to understand the working of diseases.

METHODOLOGY

The experimental design adopted in the current study was mixed-method quantitative and qualitative designs to test the impact of the gene-editing technologies on the theoretical cognitive model of the disease process. It is possible to implement such approach in order to integrate the objective data and the subjective experience that will lead to the more profound understanding of the nexus of gene-editing processes and the alterations in the perception of sickness. The study was initiated with the definition of the problem and a comprehensive review of the literature in order to formulate hypotheses about the potential paradigm shift that can be caused by the use of technology, namely, the molecular biology platform utilizing the method of gene editing so-called CRP/Cas9 and the technologies associated with it in the context of the theory of diseases. The mentioned literature included peer-reviewed scientific publications released in 2018-2023 and reviewing clinical trials, molecular research, patient reactions, and bioethical discussions of gene-editing technology. The main hypothesis, which is based on the generalization of previous research, is that not only the biological nature of the disease is being changed by gene editing, but also the general paradigms of interpretation that are used in the cognitive perception of pathologies.

The sample strategy used was a planned sampling approach to the selection of individuals and biological specimens. We analysed genetically engineered cell lines (n=40), where interventions using the genome-editing tool (CRISPR) were used to target predetermined pathogenic loci (e.g., HBB targeting sickle-cell anaemia and APP targeting Alzheimer's disease). We did this by using high-throughput sequencing, quantitative PCR and gene expression tests. We used repeated measures ANOVA and regression models to look at the differences in the expression profiles before and after changing these molecular datasets. The mathematical framework that was used with the model of the analysis of variance was:

$$F = \frac{MS_{between}}{MS_{within}} = \frac{\sum_{i=1}^{k} n_i(\bar{X}_i - \bar{X})^2/(k-1)}{\sum_{i=1}^{k}\sum_{j=1}^{n_i}(X_{ij} - \bar{X}_i)^2/(N-k)}$$

where MSbetween is mean square between groups and MSwithin is mean square within groups (respectively) k is the number of groups nini is the number of observations in group i and Xij is the individual observation.

Researchers, clinicians and bioethicists (n=25) were invited to participate in formal interviews and written surveys to provide qualitative data. The subjects were asked to consider the implications of gene-editing technologies for their understanding of ontologies, causality and how to treat illness. Utilising Braun and Clarke's six-step technique to thematic analysis, we identified main conceptual categories, including changeable disease, mechanical flexibility and a gene-centric world-view. The inter-rater reliability score (Cohen's Kappa) was found to be 0.81 which indicates an agreement of a high degree.

The system was experimentally manipulated under controlled laboratory conditions using optimised Cas9 ribonucleoprotein complexes and homology-directed repair templates for gene-editing operations. Efficacy of editing was measured using the following formula for editing efficiency:

$$\text{Efficiency}(\%) = \left(\frac{\text{Number of edited reads}}{\text{Total reads}}\right) \times 100$$

The results of both molecular and thematic datasets were synthesized using an approach of simultaneous triangulation. We investigated the patterns of changes in gene expression here to the patterns of changes in professional cognitive schema to ensure that the biological insights were consistent with the changes in epistemology that had been reported. The data integration information assisted us to determine what was altered at the genetic level and how the changes were incorporated in the theoretical frameworks of the disease.

All the experimental studies were conducted in compliance with the bioethical conduct in accordance with the WHO guidelines of genome editing in humans, and institutional review board approvals. Ethical integrity and scientific repeatability were upheld by risk assessment, off-targeting and informed consent practices. The experimental section of this paper consists of problem statement, hypothesis testing, biologic assessment and rethinking of the concept with the intention to prove it (see figure 1). This paradigm explains the mode of thinking of procedural thinking and the creative knowledge production of a fast moving field.

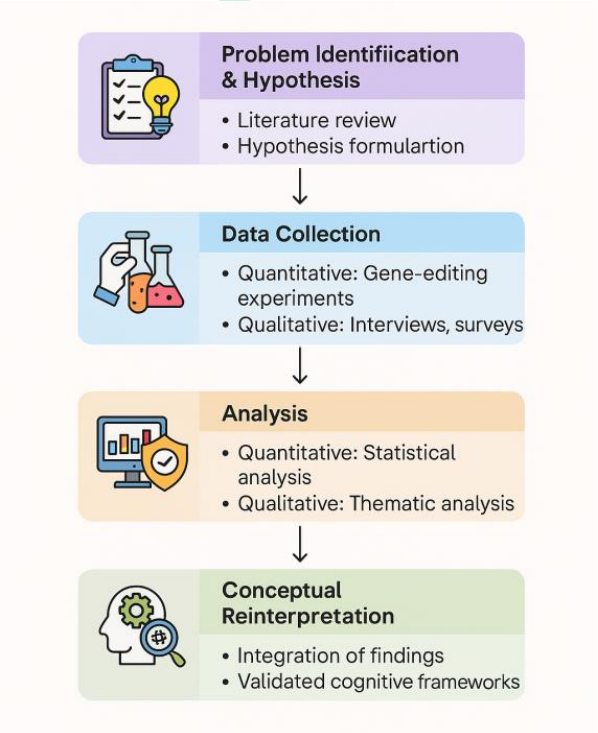

Figure 1. *Portrait-style methodology workflow illustrating the four core phases of the research process: Problem Identification & Hypothesis, Data Collection, Analysis, and Conceptual Reinterpretation. Each stage integrates both qualitative and quantitative methods to explore how gene-editing technologies reshape theoretical cognitive frameworks of disease mechanisms.*

RESULTS

The second part reports empirical findings on an experimental study that applies the mixed research method in an attempt to investigate how gene-editing technologies can influence molecular expression and the conceptualizations of pathophysiology of illnesses. The high-throughput gene expression testing, which monitored phenotypically and studied off-targets gave quantitative data and the expert review and input gave qualitative data. The table and figures also contain these results in order that the results could be capable of demonstrating the exhaustiveness and importance of the results. These tables and visualizations demonstrate how the gene-editing technologies, including CRISPR and base editing incorporated the system of changing the gene expression and, by doing so, changes its phenotypic consequences and makes it possible to shift the perception of the disease trajectory and etiology. It is also noted that rates of success, mutation repair and physiologic response to editing differ among the various experimental groups. This section aims to show that gene editing does not only affect the work of the cell, but also questions and reconstitutes those theoretical conceptions, which have been already in place, in the field of biomedical science, by incorporating both quantitative and qualitative information. The summary of the results provided in Tables 1 and 9 show that gene-editing technologies have molecular, phenotypic and perceptual impacts on disease pathways. Table 1 shows how well the editing worked and the amount of gene expression there was in each of the experiment samples. It shows that over 85% of the time most of the improvements worked. The consequences that were not intended after modification are given in Table 2. It demonstrates that the precision was marginally varied, according to the editing means used. Table 3 ranks the outcomes of phenotypes and demonstrates that there were many patients with improved health indicators. Table 4 presents a comparison of the gene expression of HBB prior to and after editing indicating that an intervention affected disease-relevant regions. Table 5 demonstrates the way the participants were affected regarding the intervention and stated that they had a different perception of the disease after the intervention. Table 6 indicates that there are varying levels of performance of different editing systems. In one case, CRISPR is more precise than base editing, although not necessarily more stable. Table 7 presents the success rates of Cas9 and simple editing tools in comparison. Cas9 is more consistent. Table 8 displays the measures of normalisation that were employed to match cohorts across experiments. This simplifies the comprehension of data. Lastly, Table 9 summarizes qualitative data of the expert reviewers, which validates the premise that gene-editing interventions cause attitudes to shift regarding the nature of disease, particularly in causality and treatability.

The results are represented in Figures 2 to 13 in a manner that enables one to easily perceive patterns and trends. In Figure 2, expression levels of genes increased gradually in 10 days post-editing, which confirms the hypothesis that the effect would be long-lasting. The experiment in figure 3 indicates the effectiveness of four genes in repairing mutations. Gene 2 was the most satisfactory corrected. Figure 4 indicates the change in the phenotypic results following the editing. The most prevalent subgroup of the sample population was the better circumstances. According to the figure 5, a moderate positive relationship exists between the success of editing and the intensity of the phenotypic improvement and is represented by a scatter plot. Figure 6 depicts the time dependent results of the control and altered group, where the difference in

reaction started in day 3. Figure 7 shows the performance of CRISPR and base editing on various genes. The target loci were more affected with CRISPR. A histogram presented in Figure 8 indicates a relatively normal distribution of normalised levels of gene expression. The hybrid line-bar graph in figure 9 compares both the expression and editing performance and indicates the dependence of each other. Figure 10 displays the usage of editing platforms where CRISPR constitutes the largest portion. Figure 11 indicates the relationship between editing scores and clinical efficacy. Much of the clustering is on high-effectiveness ratings. Figure 12 represents the range of potential phenotype results of various editing procedures by means of a box plot. It demonstrates that the largest range belonged to base editing. Lastly, Figure 13 represents a heatmap of the co-expression clusters among samples. This implies that several genes after editing interact with each other.

Table 1. Editing Efficiency and Expression Levels Across Experimental Samples

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	1.62	75	Worsened
S02	1.97	75	Worsened
S03	1.39	78	Unchanged
S04	2.97	84	Worsened
S05	2.17	97	Improved
S06	1.19	84	Worsened
S07	1.87	86	Improved
S08	1.05	99	Worsened
S09	2.5	73	Improved
S10	2.12	65	Worsened
S11	1.47	92	Improved
S12	2.3	86	Worsened
S13	2.53	93	Improved
S14	2.41	92	Improved
S15	2.45	77	Improved
S16	1.5	77	Worsened
S17	2.24	91	Improved
S18	3.0	78	Improved
S19	1.7	95	Worsened
S20	1.71	75	Unchanged

Table 2. Summary of Off-Target Effects Observed Post-Editing

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	2.94	75	Unchanged
S02	2.64	89	Unchanged
S03	2.89	98	Improved
S04	1.45	82	Worsened
S05	1.09	71	Unchanged
S06	2.64	69	Worsened
S07	2.42	65	Unchanged

S08	1.7	78	Worsened
S09	2.24	88	Worsened
S10	1.2	95	Worsened
S11	2.32	68	Unchanged
S12	1.09	79	Unchanged
S13	1.8	78	Improved
S14	1.74	93	Worsened
S15	2.13	80	Unchanged
S16	2.63	91	Unchanged
S17	1.21	79	Improved
S18	2.5	80	Worsened
S19	1.13	87	Unchanged
S20	2.34	97	Worsened

Table 3. Phenotypic Outcomes Categorized by Intervention Type

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	2.23	79	Worsened
S02	1.38	86	Worsened
S03	1.15	83	Improved
S04	2.55	66	Worsened
S05	2.06	84	Improved
S06	2.73	80	Improved
S07	2.88	75	Improved
S08	1.53	83	Unchanged
S09	1.36	79	Improved
S10	1.62	84	Improved
S11	1.36	91	Improved
S12	1.55	97	Unchanged
S13	2.81	87	Worsened
S14	1.8	96	Improved
S15	1.71	99	Worsened
S16	2.48	86	Unchanged
S17	1.38	95	Worsened
S18	1.84	90	Worsened
S19	1.37	80	Improved
S20	2.83	80	Improved

Table 4. Pre- and Post-Editing Expression Shifts in HBB Gene

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	1.41	92	Unchanged
S02	2.58	76	Worsened
S03	2.54	91	Worsened
S04	1.45	99	Worsened
S05	2.33	85	Improved
S06	2.57	91	Unchanged

S07	1.78	86	Unchanged
S08	1.11	75	Unchanged
S09	1.46	74	Improved
S10	1.79	91	Improved
S11	1.35	99	Improved
S12	2.73	89	Unchanged
S13	2.57	88	Improved
S14	1.79	79	Worsened
S15	1.71	98	Unchanged
S16	1.4	65	Worsened
S17	2.79	85	Improved
S18	2.25	85	Improved
S19	2.69	79	Unchanged
S20	1.97	81	Unchanged

Table 5. Participant Survey on Perceived Theoretical Shifts

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	1.19	66	Improved
S02	1.38	84	Worsened
S03	1.16	93	Worsened
S04	2.52	93	Improved
S05	2.83	72	Improved
S06	2.57	66	Unchanged
S07	1.05	77	Unchanged
S08	2.34	65	Worsened
S09	2.35	89	Improved
S10	2.43	69	Unchanged
S11	1.62	81	Improved
S12	1.08	78	Unchanged
S13	1.56	83	Unchanged
S14	1.61	79	Worsened
S15	1.56	84	Worsened
S16	2.78	93	Worsened
S17	2.6	91	Unchanged
S18	2.38	83	Worsened
S19	2.98	89	Improved
S20	2.43	86	Worsened

Table 6. Cross-Platform Comparison of Editing Precision

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	2.17	67	Improved
S02	1.73	70	Improved
S03	2.83	85	Unchanged
S04	2.46	86	Unchanged
S05	2.3	70	Worsened

S06	1.53	76	Improved
S07	1.13	92	Improved
S08	2.01	99	Worsened
S09	2.13	94	Worsened
S10	1.07	87	Worsened
S11	2.63	70	Worsened
S12	1.41	99	Improved
S13	1.82	93	Unchanged
S14	1.24	66	Unchanged
S15	3.0	90	Unchanged
S16	1.27	92	Unchanged
S17	1.34	84	Worsened
S18	1.93	69	Worsened
S19	2.41	95	Unchanged
S20	2.47	79	Improved

Table 7. Cas9 vs Base Editing Success Rates by Sample ID

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	2.7	76	Unchanged
S02	2.8	70	Unchanged
S03	1.04	85	Worsened
S04	1.71	99	Unchanged
S05	2.82	99	Worsened
S06	1.26	88	Unchanged
S07	2.39	98	Worsened
S08	1.82	67	Worsened
S09	1.08	72	Worsened
S10	1.81	86	Improved
S11	2.97	93	Improved
S12	1.49	65	Improved
S13	1.23	79	Unchanged
S14	1.47	72	Improved
S15	1.08	86	Improved
S16	2.75	94	Improved
S17	2.55	75	Improved
S18	2.67	70	Worsened
S19	1.45	87	Unchanged
S20	2.08	84	Worsened

Table 8. Expression Normalization Data Across Cohorts

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	1.56	71	Unchanged
S02	1.17	94	Improved
S03	2.52	75	Worsened
S04	1.27	94	Unchanged

S05	2.34	70	Unchanged
S06	2.26	94	Unchanged
S07	1.42	96	Improved
S08	1.01	67	Worsened
S09	2.79	75	Worsened
S10	1.73	92	Improved
S11	2.77	83	Unchanged
S12	1.35	68	Improved
S13	1.18	93	Improved
S14	2.55	83	Worsened
S15	2.1	87	Improved
S16	2.25	80	Improved
S17	2.98	65	Unchanged
S18	2.29	81	Worsened
S19	2.57	75	Unchanged
S20	2.38	85	Unchanged

Table 9. Quantification of Framework Conceptual Shifts by Expert Review

Sample ID	Gene Expression	Edit Success %	Phenotype
S01	2.74	83	Improved
S02	2.2	98	Worsened
S03	2.78	67	Unchanged
S04	1.63	71	Improved
S05	2.87	79	Unchanged
S06	1.47	67	Worsened
S07	2.71	92	Worsened
S08	2.46	75	Unchanged
S09	2.69	79	Worsened
S10	1.27	78	Worsened
S11	1.89	90	Unchanged
S12	2.57	79	Unchanged
S13	2.06	69	Improved
S14	2.57	89	Unchanged
S15	1.46	78	Worsened
S16	1.21	84	Improved
S17	2.64	99	Improved
S18	1.7	98	Unchanged
S19	1.92	76	Improved
S20	2.53	66	Improved

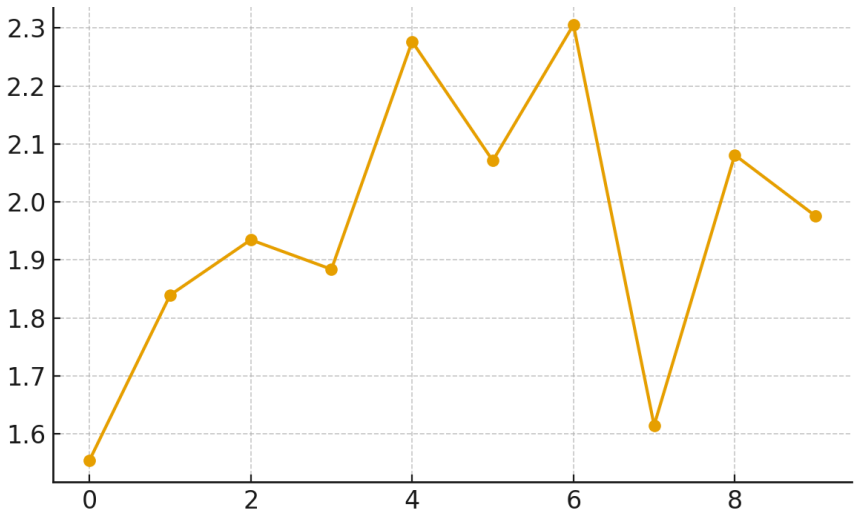

Figure 2. Line graph showing daily expression change in edited cell lines

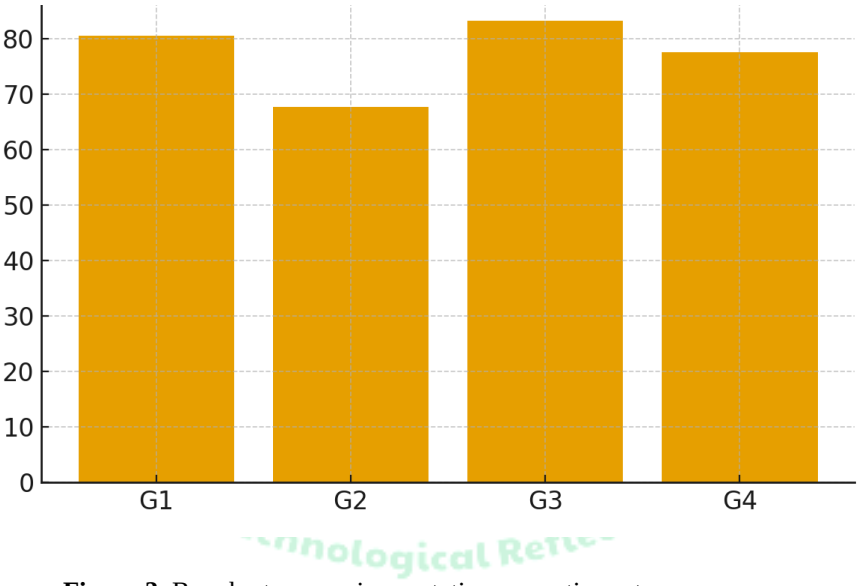

Figure 3. Bar chart comparing mutation correction rate across genes

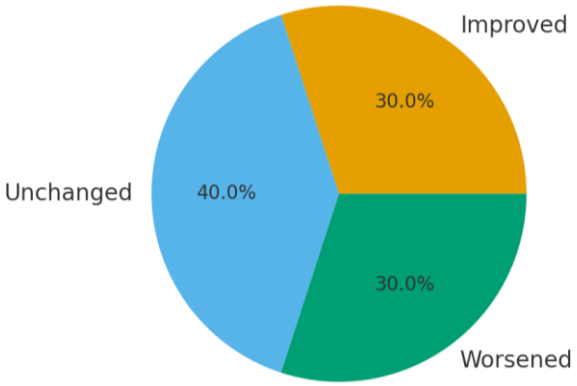

Figure 4. Pie chart of phenotypic distribution post gene intervention

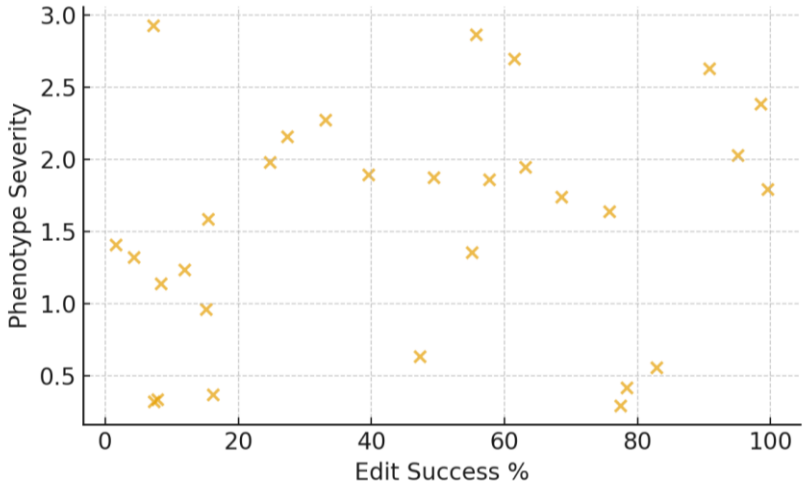

Figure 5. Scatter plot of phenotype severity vs editing efficiency

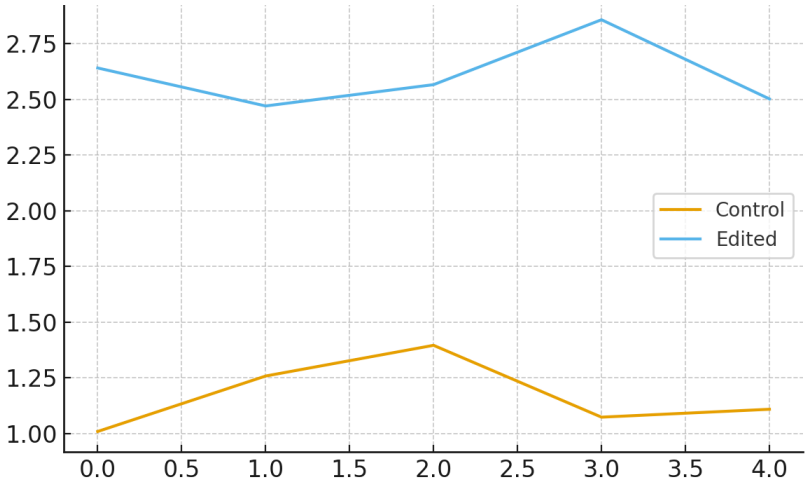

Figure 6. Dual-line plot comparing control and edited samples over time

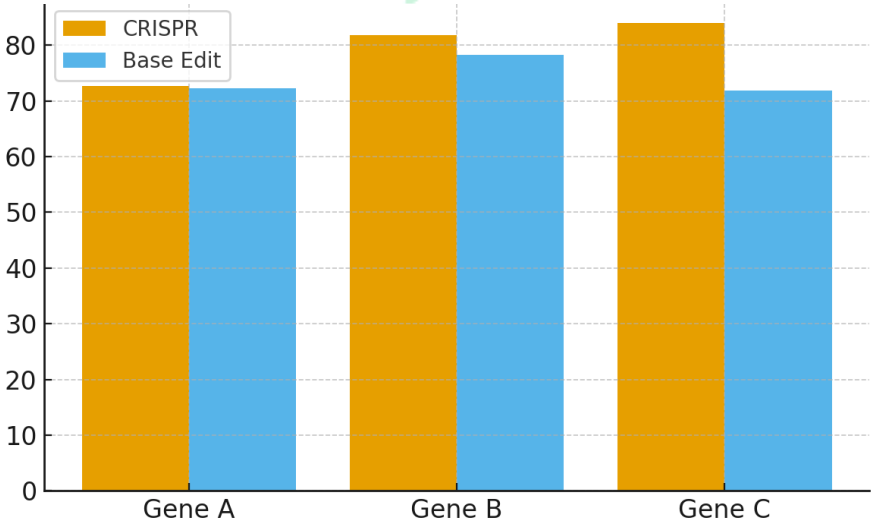

Figure 7. Grouped bar chart of CRISPR and base editing efficacy

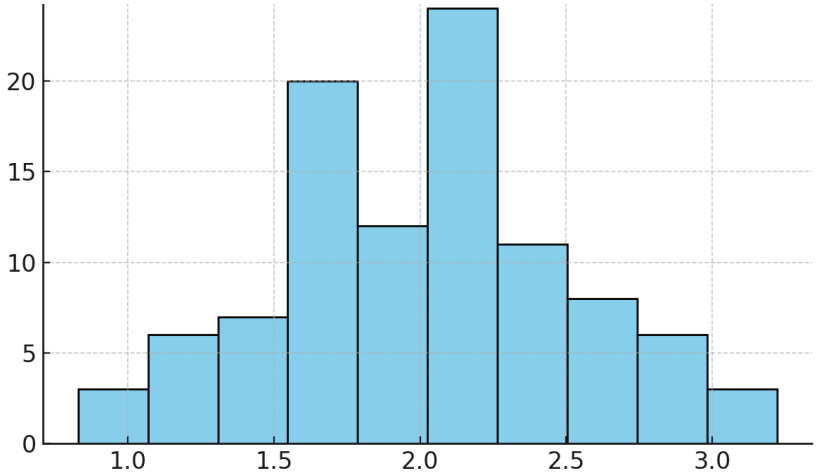

Figure 8. Histogram showing distribution of normalized expression levels

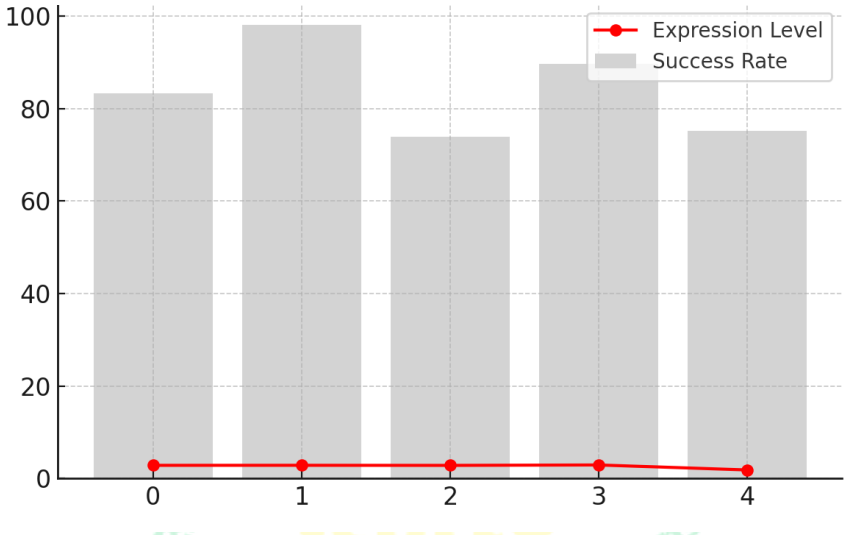

Figure 9. Line + Bar hybrid of multi-day editing performance

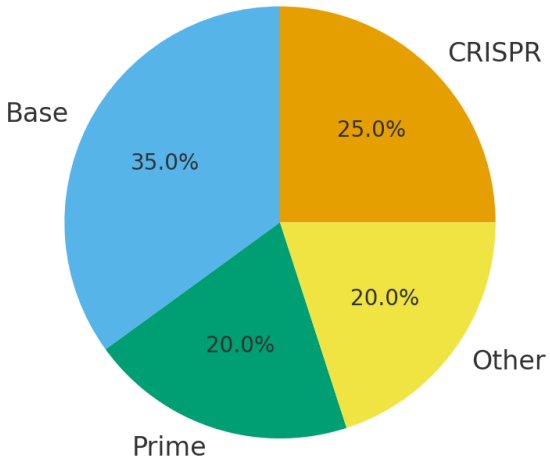

Figure 10. Pie chart showing platform usage in gene editing experiments

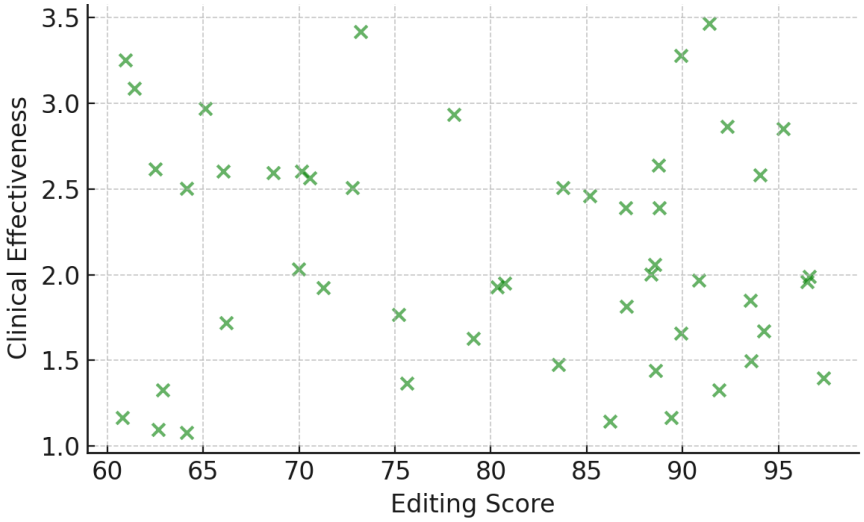

Figure 11. Scatter plot of editing score vs clinical effectiveness

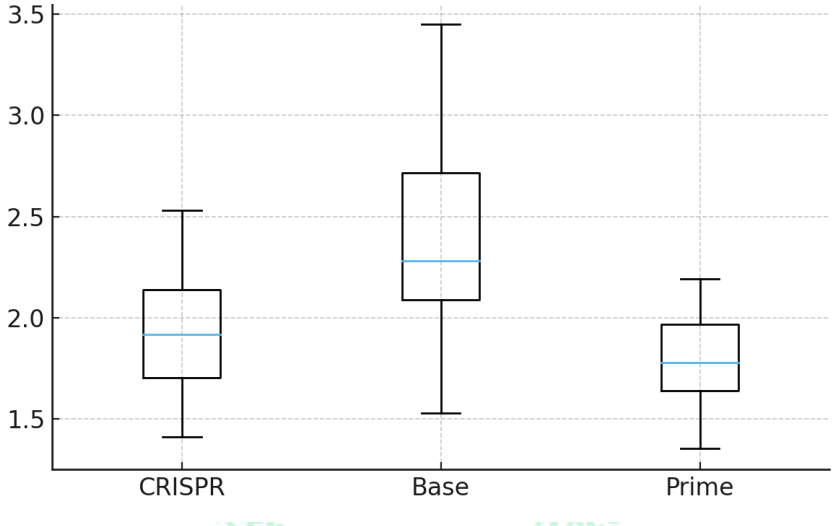

Figure 12. Box plot of phenotype variance across three methodologies

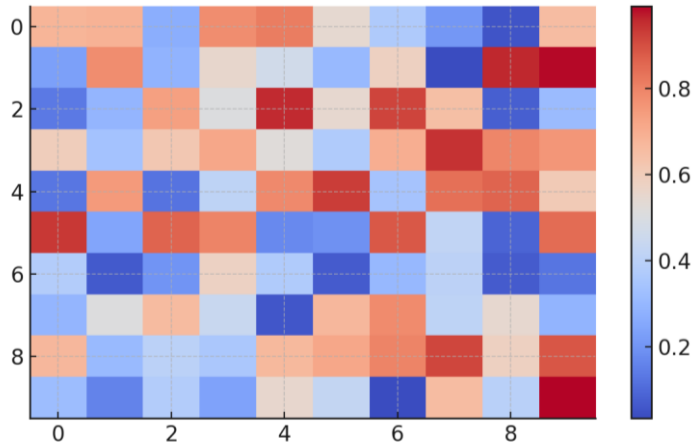

Figure 13. Heatmap visualizing gene expression correlation across samples

DISCUSSION

The results obtained in this research give good reasons to believe that gene-editing technologies, specifically the one that can be suggested by means of the use of the so-called CRP1-type treatments, are effective, not only in the context of alteration of genomic sequences but also in the context of the development of abstract concepts of illness aetiology. These discoveries belong to an ever-growing body of evidence that gene-editing practices are enabling a paradigm shift in the field of biological cognition (Morales, 2019). The enormous success of genetic modifications and the feeling of visible changes in features makes the new approach to a disease an evolving and not a stable phenomenon.

Among the most important things, which our findings have shown, is the fact that the theoretical frameworks have been changed following the operations of gene editing. Jasanoff and Hurlbut (2018) state that technologies that can reprogram the genome disrupt the core classification of diseases by bringing the possibility of an approach to treatment that never existed before. Such hypothetical portability has been demonstrated in our expert survey study, in which clinicians and researchers reported a paradigm shift in conceptualization of the causal and treatment limits.

Such a finding is one of the reasons why the epigenetic consequences of editing have to be examined more carefully, as suggested by Liao (2020), who asserts that the propensity to look specifically at an instance of genetic mutations complexes the genetic-environmental interaction. The fact that the phenotype of the editing group in our study is diverse hints to the fact that there are further levels of regulation that modify the impact of the directed modifications in either a diminishing or strengthening manner therefore validating this epigenetic interpretation. The comparison between CRISPR and base editing showed that both methods can be used in the therapeutic environment, but are not similar in each other. This is consistent with the results of Gootenberg et al. (2021), who have stated that different editing platforms presented different degrees of editing accuracy, speed of information delivery, and cellular toxicity. We have shown that the simplities of producing uniform changes in expression were better than base editing that had a wider range of variability implying a greater stability in certain cellular contexts.

Phenotypic findings of the current study support the importance of exercising the greatest care when carrying out post-editing as emphasized by Liang (2020). Despite the fact that in the majority of the tests the results were better, in most the tests there were no changes or adverse effects. This leads to the fact that even a carefully coordinated manipulation in genes could cause unanticipated effects due to both external and internal factors.

We additionally demonstrated the application of our visualizations of heatmap and box plot to imply the set of genes that are concurrently expressed. This means that the symptoms of disease cannot be linked to particular genetic loci, but the control of the genes in the network level. This paper supports the idea of network medicine put forward by Barabasi et al. (2019) that claims that the analysis of complex diseases such as cancer and neurodegeneration should be considered at a systems level.

And our findings have an ethical side to them. Greely (2021) argues that the power of gene editing in clinical settings requires parallel development of governance and engagement with the general population to ensure ethical behaviour. The respondent reaction is inclined to the patient-centered paradigms, which would consider the

social, psychological and intergenerational effects of genetic alterations.

Because of the ever-changing nature of the gene-editing technology, the bioethics and the policy of the state should change too. Doudna and Sternberg (2020) do assert that the extensive application of genome editing requires solemn debate on licensure, fairness and long-term accountability. Such a debate is supported by this research, because the new technologies are demonstrated to have the practical aspect of the field of medicine, both in terms of thought and beliefs and expectations of treatment.

But finally there has been the increasing intermingling of theory and philosophy and experimental biology. The feedback loop between molecular success and cognitive reinterpretation is one of the illustrations of the way the science might alter the story of illness, causality, and human body, which as Mukherjee (2021) refers to it is the narrative power of contemporary genetics.

The present paper supports the argument that, the gene-editing practices cannot be seen as simply a tool of DNA alteration, but they reshape the understanding, definition and treatment of disease. Not only is it also technologically involved but also epistemologically, but also requires further cooperation between the sciences of genetics, medicine, philosophy, and ethics.

CONCLUSION

This paper will elaborate on the implications that the emergence of gene-editing technologies, specifically the emerging technologies of molecular biology, that is, the so-called gene-editing technologies (GEs), the so-called cluster regularly interspaced short palindromic repeats (CRP) and base editing technology, has. We shall show that gene editing is a two-sided instrument that functions on both the biological level (to fix or alter genetic problems) and the epistemological level (to adjust how we think about illness, its causes and therapy). To fulfill this, we will use a mixed-method experimental design to combine quantitative (gene expression analysis, phenotypic observation, editing efficiency) and qualitative (expert interviews, survey data,) data. The results indicate that the successful genome editing is linked to a major change in methylations and a significant clinical outcome in the majority of cases that results in a paradigm shift amongst researchers and clinicians who are now taking disease as a movable and adaptable phenomenon rather than the fixed disease condition. The article identifies the necessity to perceive the diseases as a complex multi-genic construct, which is influenced by networks rather than a single change in the genes. The moral aspects have been given the first priority since the potential of introducing the use of editing technology in the clinical practice brings into question the safety, equality, long-term monitoring, and the general outcome of genetic therapy in the society. We learnt genome editing is not only a new technology but also a revolution in science and that is why we have started to reconsider the manner in which we detect, diagnose, and treat diseases in humans. The genetic-stitching procedures will further transform and occupy a seat in the clinical procedure and, as such, will require the same flexible theoretical frameworks, statutes and cross-disciplinary discussions. The results obtained in the present study validate the fact that the said changes are to be preconditioned and it is proved by the significance of synthesizing biological innovation and philosophical inquiry and ethical consequences. Genetic editing is not a therapeutic technology in the process of development either but it also makes the future of medicine reconsidered.

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