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The efficiency of SLC6A4, COMT, MAOA , and other genes in Stress predispositions in psychogenetic process -To define stress in psychogenetic way

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ABSTRACT

One of the major aspect of formation of corporate human rights is employees, considering the socioeconomic aspects and profitable indicators based on employees contribution to the company, where as the corporate human rights as per constitution of india only favours employee rights in terms of productivity, but the mental health quotient is not been addressed or considered, the ratio and data related to employee stress, suicide attempts, and other related stress causing factors need some consideration at present 2025 context, from the data cited in article ALL THINGS TALENT- NAUKIRI TALENT CLOUD, stated that 60% of the employees faces stress irrespective of their age, being more specific about 70% of women at work places feel more stress than men, that indulge in questioning our corporate human rights provisions and evaluating the mental health care act in india.the stress is not a emotion that can be forgotten or erased, it has direct or indirect impact over genes, so what is the role of genes here , we shall state the genes are the identity of every human being that are heritable to stay as a evidence for generations, what if that Genes are destructed through stress in our day today life. Some of the factors that are negatively affected due to stress are the humans and their emotions through distress, so what happens in genes when stress is adhered by the human in workplace need a glance and details to enhance our mental health well-being at our work place and personal space.Some of the specific genes stress dispositions that we are going to consider here are SLC6A4,COMT,MAOA.We are going to analyse whether these genes has impact on stress dispositions in epigenetic genera for all workaholic people at different management levels.

KEYWORDS: neuro rights, neurocognition, Seethaframework, legal psychology, stress, health-laws, neurogenetics, corporate human rights

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INTRODUCTION

In general if we insists our statement saying money is mandatory in employee work settings, we should be familiar with stress as a mandatory quotient in every employees in the management , so there are policies adhered for employees cost to company, remuneration, employees skills training, employee insurance benefits etc. there are nor considerations or policies to address the employees stress, if there are work, people and communications happening, there are going to be stress, conflicts and perceptions that are going to occur in the company.so what are the negative aspect of stress, are they going to create a adverse environment in the company, or do they going to determine the employee mental health as priority, while considering the outlook of mental health care act in india 2017 and corporate human rights, lets deploy the emergence of neuro genetic rights and neural privacy every employee should adhere in the institution , lets determine the stress wave therapy by implementing Seetha framework in psychogenetic metrics that can enhance the health and well being of employees especially women's in workplace settings.

In general the equation to describe the human corporate rights in congruence with mental health care act can so-licitly with effect on determining the importance of missing indicator of addressing the stress in more genetic way, thus a ideal notation that could align the gap between the human corporate rights and mental health act is the equation of neuro rights and neuro cognitive resilience in psychogenetic way in short that can be addressed with providing the Seetha framework.

Human corporate rights+mental health act 2017——>neuro cognitive resilience act+psychogenetic

From the literature we could infer that human corporate rights has withheld the details of employees benefits and cost to company details alongside posh act to forbid the sexual harassment at workplace, where as the mental health act explains the acute mental illness foreseen by the human either major or minor, based on to address the mental illness like alzimers, mental disorders etc with regulations provided in the mental health care act 2017 , but this research has the vivid disclosure of how to enact the implementation of importance of preventing the stress in very early stage to avoid the acute mental illness, and fore gain the rights to access to mental health and mind well being of the employees directly or indirectly.

The misplaced details of stress as a indicator in determining the mental health of employees are still been not addressed in the congruent defined acts, that provoke the need for providing importance to employee well-being as a part of policies of Human Resource management.

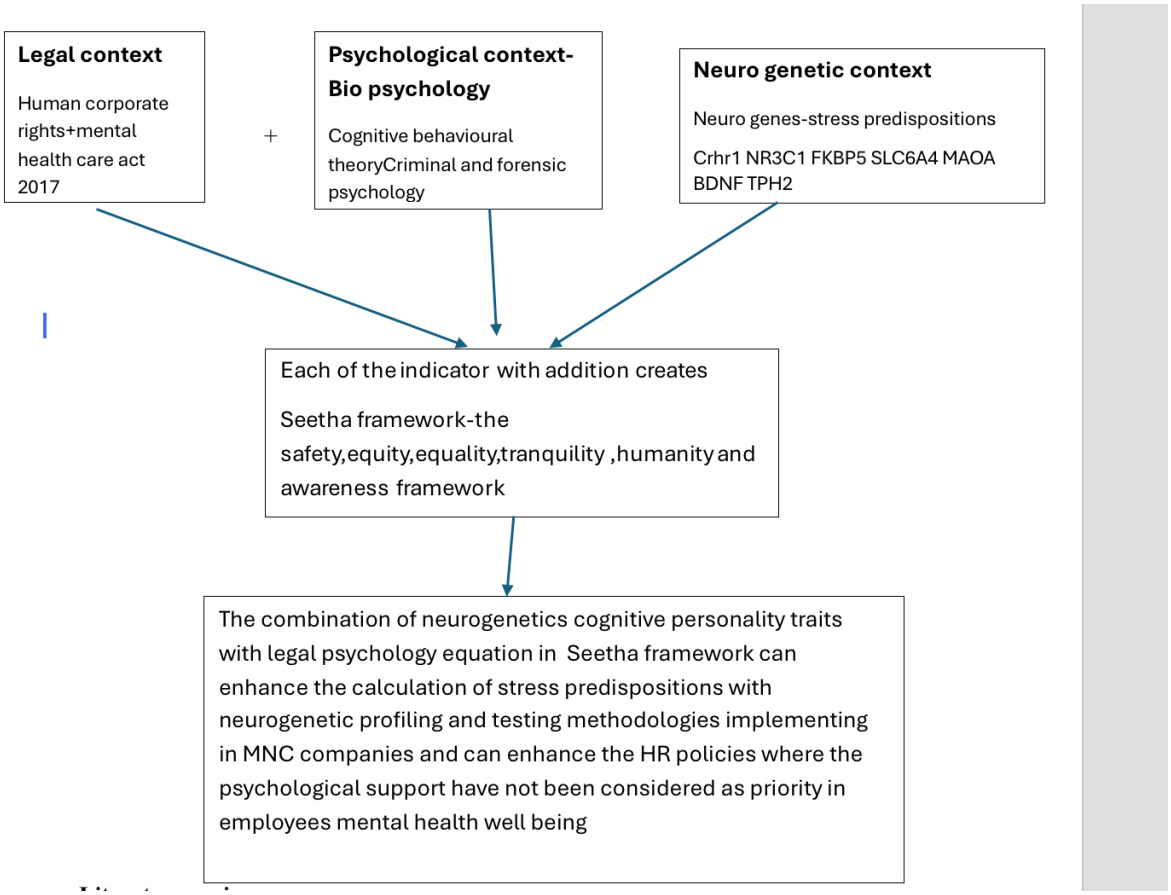
LITERATURE REVIEW

Role of stress genes is stress disposition in epigenetic methylation

DNA and its composition has a vital aspect or role in determining the stress variance in humans in all their life, the general suspect of how human beings could tent to react to things in stressful situations which has direct and indirect effects on health, to concentrate on women stress genomics that have modulation due to environmental factors, the hipbones, genes methylation and prolonged stress causing environment can enhance the effect in how the brain genes can adapt to the stressful situations. This the pathway to Hearfullness emotions and mindful mapping has difference indications that need details description of how stress can be handled and how it can be reduced and what are the negative impacts of stress genes and how it effects the decision making ability and hereditary functions can be disturbed.

The conceptual Seetha frame work that can ideate the mindfulness and Hearfullness is described belowHuman corporate right

Literature review



From the literature we could determine the dramatic ideal shift of mental instability with employees when it comes to grounds of equal rights without discrimination, but in general the emotional quotient in the legal psychology could even provide a variance of how a human have to use his cognitive abilities to overcome the self bias and fear to enhance his or her capabilities, thus to ensure the role of employee productivity, lets consider now the inner well being of employees, from the literature there are many theories such as Herbert motivational theories, Maslow hierarchy's, the theories that are related to leaderships etc, but we to define that there are proper concentration provided for mental health well being of the employees.the basic stigma here is there is only acute mental health illness present but not admissible relation with stress management and conflicts management.to relate bio psychology in general with the equation of legal psychology and neuropsycho genetics framework we shall adhere the role of the negative impact of stress over human neural genes other wise termed as epigenomics. The influence of environmental factors that has direct or indirect stress predispositions causing the human to suffer from psychological illness if prolonged for longer times. Thus to interpret in more psychological terms, the relation of stress and conflicts in human mental health that enhances the behaviour of humans and health in the negative manner.Thus to determine the standard framework and equation , we should be able to know what are the negative impact of stress over genes, and we're there any legal aid to address those issues for the employees in the MNC company.so in this criteria of stress dispositions.There are no proper definitions of Law in the Indian mental health care act to define the neurocognition to address the temporary psychological illness that need to be addressed with the psychological counselling, thus the deficiency of psychology in the legal aspects in mental health care act still needs a inclusion of psychological perspectives and even the HR policies with neurogenetics profiling need to be addressed.thus psychology and neurogenetics inclusions in HR policies and laws related to psychological support still need enactment.

METHODOLOGY TO DETERMINE STRESS PREDISPOSITION BY NEUROGENETIC TESTING

Stress-Related Gene Functions (for context)

Gene	Description / Function in Stress Response
CRHR1 / CRHR2	Encode corticotropin-releasing hormone receptors that regulate the hypothalamic-pituitary-adrenal (HPA) axis and cortisol release under stress.
NR3C1	Glucocorticoid receptor gene; mediates cortisol signaling, influencing resilience and stress reactivity.
FKBP5	Regulates glucocorticoid receptor sensitivity; linked to stress adaptation and PTSD vulnerability.
SLC6A4 (5-HTTLPR)	Serotonin transporter gene; influences mood regulation and susceptibility to anxiety/depression under stress.
MAOA	Breaks down neurotransmitters (serotonin, dopamine); variants influence emotional control and aggression.
BDNF	Supports neuroplasticity and neuron survival; reduced activity linked to chronic stress and mood disorders.
COMT	Degrades dopamine; variations affect stress-related cognition and executive function.
TPH2	Tryptophan hydroxylase 2; key enzyme in serotonin synthesis, linked to emotional regulation.

Small outlook of epigenomics in neuro genetics , stress predispositions, methylation of stress genes
Genes are in general can be explained with the aspects of neuroscience , so any relational understanding of neuro activities are related to central nervous system , a scientific way to analyse how humans behave,produce thoughts emotions and control important body functions etc.,mental illness are concentrated in relation with neuroscience and the prospects towards the variance of how the stress can enhance human way of thinking and behaviour.its an multidisciplinary approach of how the emotions variance has the impact over human brain functions in both aspects of neurogenetics and brain neuroscience. We all knew the importance of the nervous system, where the human tend to talk, walk, eat, respond, react all being controlled with nervous system directly or indirectly, the specific definition of human behaviour has been determined by the response signals that we give to brain through neuro transmitter and the way the brain respond to the ;parts of the body through neuro transmitters.

Thus to ideate the general neurogenetic importance in the psychological aspects, the role of psychology in regulating the neurogenetics can be determined with how the epigenomics could ideally enhance the gene variability in both positive as well as negative manner, hence the general proposition of neurogenetics profiling can be explained with formulas such as Neurogenetic profiling=the stress genes evaluation+stress DNA methylation +Epigenomics (phenotype) To study about our genes that includes the structure of DNA , composition of the structure of DNA, that is chromosome.the variation of genes considering both internal and external factors includes genes variability and transcription along with translation.inorder to understand the neurogenetic profiling we should first be familiar with what is gene variability, epigenomics, neurogenes transcription and translation etc.

Components of DNA and Study of neurogenetics
We shall determine the variations of genes only if the gene is undergoing the negative translation in terms of the process under epigenomics, in order to determine the concentration of stress a normal human can adhere we should be able to understand why this is creating the negative impact in human health and wellness etc.the general size of the chromosome is 1.4 micro meters in diameter which are tightly regulated structures, 300 manometer fibre is the composition of 30 nano Meer fibre which are tightly packed to create a chromosome etc.and it compose of nucleoside witch is 10 nano meter.which has beads and strings structures called as nucleosome with the protons attached to it such as H2A,H2B,H3 and H4 etc.When these nucleosome are further analysed the structure of the DNA can be ideated that is 137 pairs of DNA wrapped around finally creates the double helix DNA..one of the major aspect we study in gene tics is coding anatomy.which includes the in-tons and exons which are being regulated with enhancers, silencers, promotors and locus control regions.

GENE TRANSCRIPTION

The gene transcription has been highly regulated with the epigenomics that is the environmental factors that are related with change in stress modulations due to environmental factors.take a example of bacteria if it gets exposed to lot of heats and suddenly the gene expression caused due to the impacts of heat shocks are relatively the damage caused in cellular level where the so called proteins heat shocks will be used to withstand that heat or fight against that heat, if there are going to be no heat shock then the the gene variations are silent and there wont be need to use energy to defend anything, thus the study of stress caused due to epigenomics will be explained n criteria with gene regulations where the genes are regulated in both manner in terms of rest and gene variability.

The role of gene description will enhance the study of stress in more detailed manner.to determine the stress predispositions, the state of gene expression can either be positive or negative based on the standard value of determination.the stress factors are generally been influenced by the external environment that can be otherwise termed as epigenomics, in this research the role of mental reactions can be either understood by the brain related functions through networks,circuits, proteins etc. the role of epigenomics in stress management plays a crucial role between genomics,transcriptomics,and epigenomics.It is important for us to study the interactions of the genes, When we talk about 21st-century neurogenetics,it is important to understand that we cannot talk about genetics alone.We really have to talk about the interplay between genomics, transcriptomics, and epigenomics.This is

because with the exception of a few monogenetic diseases such as sickle cell anaemia or Huntington's disease, a brain disorder, it is rather rare that we have one gene that causes one phenotype and one disease.It is rather that we have the interaction of genes that give rise to a certain phenotype on the transcriptome level, and that this is all regulated by epigenomics. It is important to understand that there is an intricate relationship between these three fields. There's a dynamic interplay, and altogether, this brings up a multiscale approach that bridges basic and clinical research. This fact that genomics gives rise to transcriptomics, which is regulated by epigenomics, is particularly important for neurodevelopment and also for brain function in health and disease.Then what is really neurogenetics?Neurogenetics is really the interplay between three different pillars. On the top, we have genetics,which can be brought about by point mutations or single-nucleotide polymorphisms or SNPs. On the bottom left, we have the environment,which is everything we do, essentially,and every environment that the cell is exposed to.This can be our nutrition, our lifestyle, pathogens we're exposed to, and also toxins we're exposed to.Finally, the third pillar are the epigenetic modifications in terms of post-translational histone modifications, in terms of DNA methylation,and in terms of non-coding RNAs.And it is really the epigenetics that bring the phenotype into being.So the epigenetics is really the interaction of genes with their environment, which bring the phenotype into being, as was proposed already in 1940 by the developmental biologist Conrad Waddington. What we're going to cover today is really the structure of the DNA for one. When we talk about the DNA,we also mean the chromosome.We also mean gene anatomy.We will hear about the concept that is called the locus control region.Furthermore,we're going to see steps and triggering mechanisms of transcription.We're going also to see how transcription can be tightly regulated, its mechanisms and its purpose.

Finally, we're going to end with a brief introduction of what is translation,which is the final step that brings a gene from its genetic form into a protein form, and which is then the effector state.What are the building blocks of a gene?The building blocks of a gene are really the chromosome. On the right side here, we can see that in its replicated form,we can see a very characteristic structure that is 1.4 micrometres in diameter, which is composed of two sister chromatids. But if we zoom into this structure a little bit further,we can see that it is actually a compilation of different tightly regulated structures.For instance,we have the 300-nanometre fibre here that is composed by a scaffold and looped domains. And if we are to zoom into this 300-nanometre fibre even further, we can see by electron microscopy up here that there's a very characteristic 30-nanometer fibre. When we zoom into this 30-nanometer fibre,we can see another fibre which is essentially compiled upon each other to form this 30-nanometer fibre.And this is the 10-nanometer fibre here that is called beads on a string. What composes this beads on a string structure is a structure that is called the nucleosome, which is essentially an octamer of histones. And we have two times each of the following proteins, H2A, H2B, H3, and H4.Together they form this nucleosome structure, which we can see here in electron microscopy,around which the DNA, and more precisely,137 base pairs of DNA are wrapped around.Then on the far left side, we have, of course, the naked DNAthat is composed of a double helixand our four bases,which are adenine, thymine,guanine, and cytosine that are paired with one another by Watson-Crick base pairing. If we leave the chromosomal scale and zoom in into a gene's anatomy, then we can appreciate that there are certain characteristic elements that are specific for each gene. For instance,we have what is called a coding sequence that is composed of exons which are going to remain in the final product of the gene, as we will see later, and of introns which are essentially going to be cut or spliced out, as we will see later.Then, of course,we have a certain numbers regulatory sequences,such as enhancers that promote the transcription of a gene,such as silencers that shut off the transcription of a gene.Then we have elements that are called promoters,which are important fortranscription factors to bind and find the gene that they have to transcribe.And all of this is controlled by a region The objectives of the research Epigenetic codes are often also referred to as an epigenetic landscape, a term that was coined by the developmental biologist Konrad Waddington in 1957. What Waddington depicted up here is a cell that just like a ball that we would roll down a hill, can take differentt ajectories. You can take a trajectory to the left or you can take a trajectory to the right depending on how hilly the landscape is. It has to go through and what Waddington supposed was to be the case is that the hills are defined by epigenetic modifications. Waddington was a developmental biologist, so the way he pictured this was that on top of the hill, we would have a stem cell that is totipotent, such as a zygote. As the stem cell starts to roll down the hill, it loses its totipotency and will become pluripotent such as an embryonic stem cell, or even an induced pluripotent stem cell. As it rose further down the hill, as it becomes more and more differentiated, it will switch from a pluripotent to a multipotent state and ultimately to a fully differentiated or unipotent state, such as is the case for neurones, astrocytes, microglia or oligodendrocytes.

RESEARCH OBJECTIVE

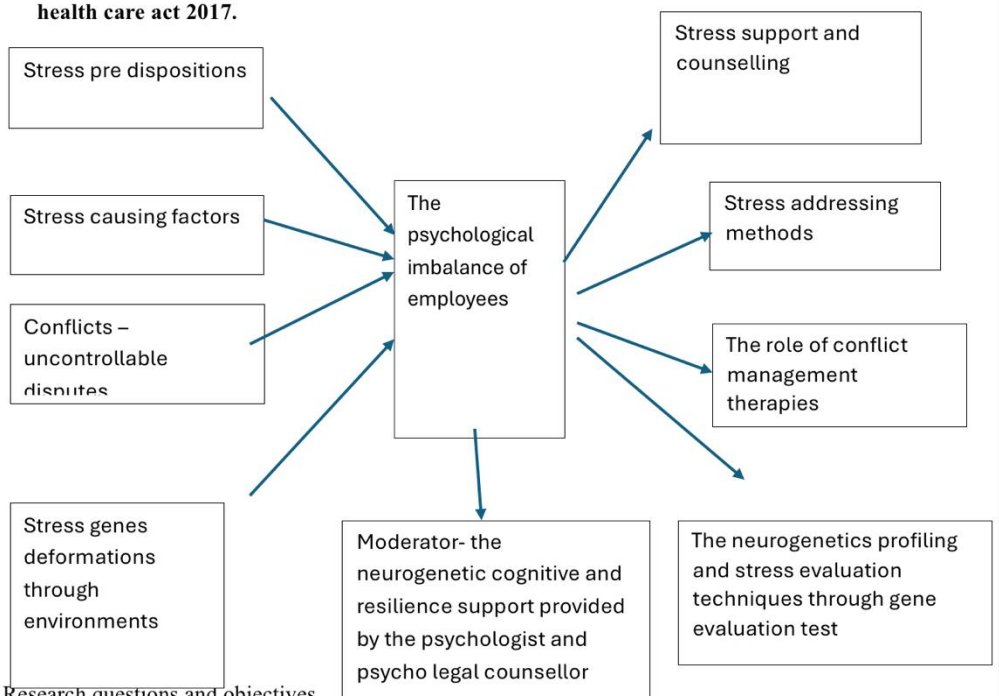
To enact the access to metal health in terms of neurogenetic cognitive resilience in constitution of india by 2026-2030
To address the HR policies with inclusion of neurogenetic profiling in the recruitment and policy related to mental health care
To idealise alternate dispute resolution committee in All MNC companies in chennai
To appoint a chief legal psycho officer in Companies to address the legal and health related aspect including the safety and ethics of the companies as per Indian culture and traditions
To in initiate the psycho legal congruence related HR policies in terms of health and wellness of the employees in respective companies
To calculate the neurogenetic deformations of the individual from being how the stress full genes can create a impact in health manner in comparison with normal stress less DNA terms

Research questions and objectives
The evaluation of stress genes in congruence to normal genes, and its variation to determine the stress values
The role of epigenomics in inducing the deformation in stress genes
The calculation of stress predispositions to evaluate the health related risk towards it
The role of gene methylation to enhance the stress response in gene interactions

Defining the stress related genes to idealise the types of stress and its causes of health risks in employees
Making employees aware of their psycho genes to evaluate their mental health well being
To determine the levels of stress variance based on management positions of the employees

RESEARCH OBJECTIVES

Figure 1: the conceptual framework of Access to cognitive and resilience act in mental health care act 2017.



Research questions and objectives

Does The environment has significant impact of stress genes in human genome
Does the stress gene evaluation has significant impact in psychological balance of humans
Does neuro genomics testing has significance influence in enhancing the determination of stress related diseases in earlier stage of disease detection

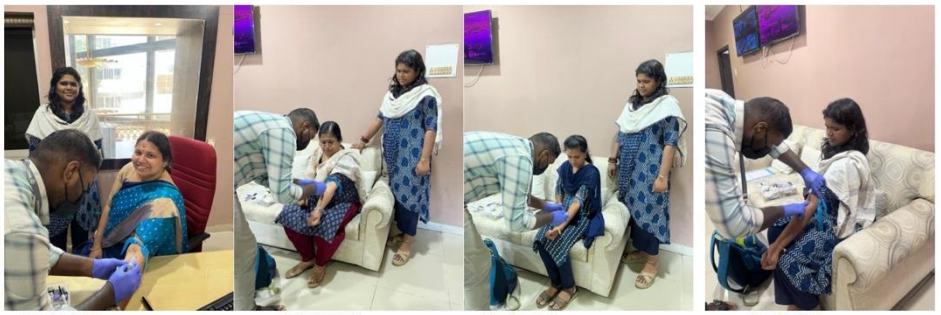
Does neurogenetic profiling could enhance the leadership skills of employees Role of stress predispositions has significance impact in employee health support in organisations Can neurogenetic policies could improve the effectiveness of health of employees health benefits in the organisations Can genetic evaluation has significance impact to categories the stress predispositions as high level risk employees, mid level risk employees and lose level risk of stress in employees.

RESEARCH METHODOLOGY

The research methodology adopted here is clinical testing, in this testing the blood sample of 4 participants has been collected, the general aspect to determine the value of stress predispositions in humans brain has been determined by analysing the genome evaluation concentrating on stress genes such as neurocognition to address internal mind disputes

Neuro genes-MAOA-L (CRIMINAL DEFENCE), DRD4/DRD2(ADDICTION), COMT(STRESS)
Neuro law and its impact on jurisprudence and health psychology in human cognition process in analysing things with strategic emotional handling perspective Neural privacy and ethics The unstructured interview has been carried out in order to determine the ideology of how these gene related to stress can be evaluated to underpin n the causation of stress in employees The interviews has been carried out with neurogenetic doctors and health related advocates to ensure the ideas are been explained in terms to stress predispositions genes that could be prevented in stress causation in employees in very early stage by providing the preventative measures and ethical and legal support, a solid evidence can be collected to support the research to enhance the laws related to stress management for employee The photographic details of test sampling collected

The blood sample for neurogenetic testing was done with four participants, the demographic details of the participants and explained below



CLIENT 1 CLIENT2 CLIENT3 CLIENT4

CLIENT	AGE	DESIGNATION LEVEL	DEPARTMENT TYPE
1	48 YEARS	HIGH LEVEL	SERVICE
2	51 YEARS	MID LEVEL	SERVICE
3	40 YEATS	LOW LEVEL	SERVICE
4	32 YEARS	RESEARCHER	SERVICE

The above table describes the demographic profile of the clients and their designation level, noe the blood test was collected by the MED-GENOME private limited Bangalore to analyse the whole genome sequencing concentrating on stress genes. The role of stress calculation and their predispositions data will be obtained from this blood sampling results and more concentrating on stress related genes.This test was carried out on date 24 th September and the result was obtained on 11th November through mail.

STESS GENE EVALUATION REPORT AND STRESS GENE CALCULATION ANALYSIS

Summary of test report

Here's a tabulated summary of all four MedGenome clinical DNA reports, focusing on stress-related genes and their corresponding findings/descriptions from each file:

Sample/Report ID	Genes Analyzed (Stress-related)	Findings / Variants Detected	Interpretation / Description
Client 2 TRN5480392 / 9415294 / 1458644	CRHR1, CRHR2, NR3C1, FKBP5, SLC6A4 (5-HTTLPR), MAOA, BDNF, COMT, TPH2	- No pathogenic or likely pathogenic variants detected in stress-related genes.- HTRA1 (c.1120G>T, p.Gly374Ter) – heterozygous nonsense variant, clinically correlate.- NR3C1 (c.146C>T, p.Ala49Val) – heterozygous variant with high minor allele frequency (benign).	No clinically relevant variants in the stress-response genes. Genes were 100% covered. Incidental variants not directly linked to stress.
Client 1 TRN5481282 / 9415283 / 1458742	CRHR1, CRHR2, NR3C1, FKBP5, SLC6A4, MAOA, BDNF, COMT, TPH2	No pathogenic or likely pathogenic variants detected.	All target genes fully covered; no mutations associated with HPA axis or serotonin pathway. Recommended genetic counselling for context-based review.
Client 3 TRN5481690 / 9415234 / 1458900	CRHR1, CRHR2, NR3C1, FKBP5, SLC6A4, MAOA, BDNF, COMT, TPH2	No pathogenic or likely pathogenic variants detected.	No significant genetic variation correlated with stress or emotional regulation pathways. Counselling advised for interpretation.
Client 3 TRN5483461 / 9415201 / 1458899	CRHR1, CRHR2, NR3C1, FKBP5, SLC6A4, MAOA, BDNF, COMT, TPH2	No pathogenic or likely pathogenic variants detected.	No mutations identified in key stress-response genes (HPA axis and serotonin system). No CNVs or SNVs relevant to phenotype.

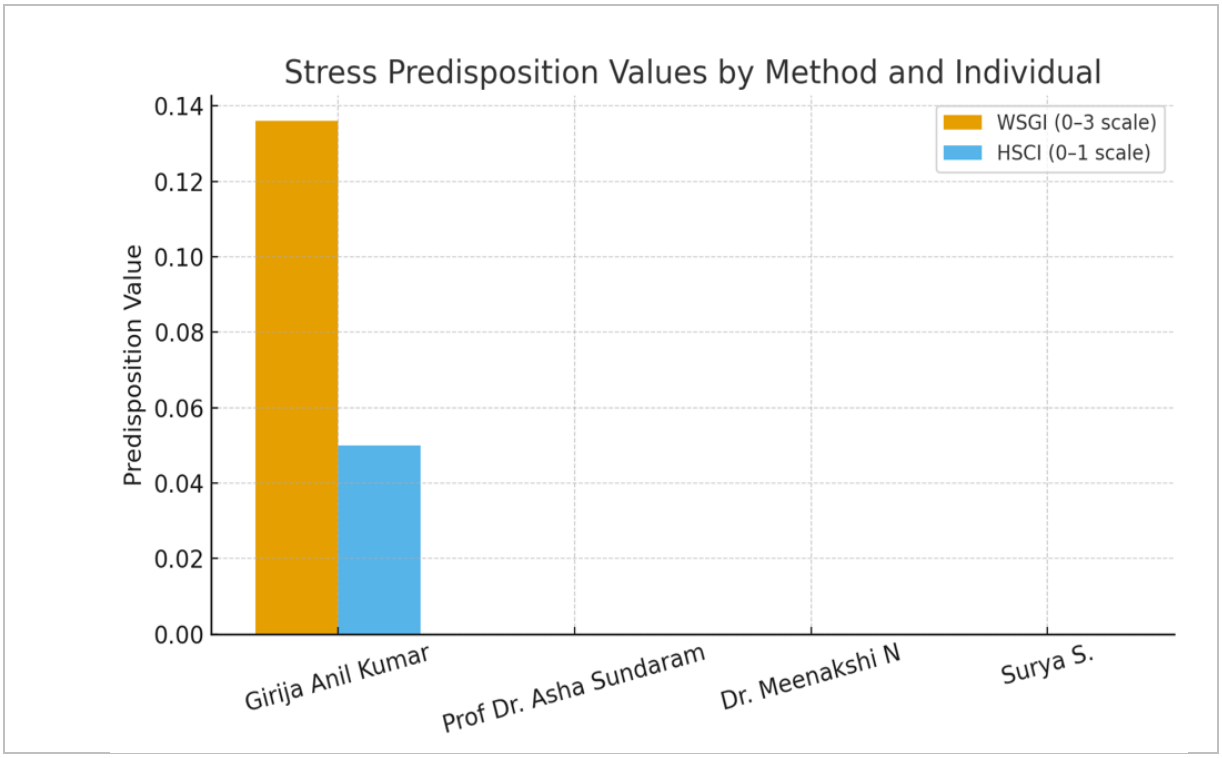
Methods adopted to calculate the stress predispositions of genes

1. Weighted Stress Gene Index (WSGI)
2. HPA–Serotonergic Composite Index (HSCI)
3. Binary Risk Model (BRMAll calculations are derived from the four MedGenome reports you uploaded (Asha, Meenakshi,

Surya, Girija), incorporating the only stress-gene variant found (NR3C1 p.Ala49Val in Girija, benign).

Combined Stress Predisposition Summary[i]

Sample / Report ID	Stress-related Variant(s)	WSGI (0–3 scale)	WSGI Category	HSCI (0–1 scale)	HSCI Category	Binary Risk Model (BRM)	Overall Interpretation
Girija Anil KumarTRN5480392 / 9415294	NR3C1 (p.Ala49Val) – benign (score = 1)	0.136	Low (< 0.5)	0.050	Low (< 0.2)	Low	Mild genetic variation in glucocorticoid receptor; overall low stress-gene predisposition.
Prof Dr. Asha SundaramTRN5481282 / 9415283	None	0.000	Low	0.000	Low	Low	No variants detected; typical stress-response gene profile.
Dr. Meenakshi NTRN5481690 / 9415234	None	0.000	Low	0.000	Low	Low	No stress-gene mutations; normal stress-resilience baseline.
Surya S.TRN5483461 / 9415201	None	0.000	Low	0.000	Low	Low	No stress-gene variation; baseline genetic stress risk.



REPORT ANALYSIS AND RESULT

IT WAS DERIVED from the stress predispositions the data obtained for client 1,3,4 has no stress pre dispositions where as the client 3 has variance in stress predispositions gene, the details of variance of genes are provided below

NR3C1	**Glucocorticoid receptor gene; mediates cortisol signaling, influencing [ii]resilience and stress reactivity.**
Pathway Role	Central regulator of the Hypothalamic–Pituitary–Adrenal (HPA) axis. It mediates the body's feedback control of cortisol release, helping restore balance after stress exposure.
Relevance to Stress Predisposition	Variants in NR3C1 can alter receptor sensitivity to cortisol — either hypersensitive (leading to prolonged stress response and anxiety) or resistant (blunted feedback, associated with depression or chronic fatigue).
Clinical Associations	Altered NR3C1 expression or methylation has been linked to: – Major depressive disorder – PTSD – Childhood trauma response – Stress-related hypertension and metabolic disorders.
Variant in Your Report	p.Ala49Val (c.146C>T) — a benign polymorphism often studied for subtle effects on cortisol sensitivity and coping style. It's non-pathogenic but can indicate mild modulation of stress resilience rather than disease risk.

From the analysis done with neurogenetic profiling testing, we could define or find that client 2 is having some pathogenic variance in the stress genes NR3C1. Thus to address this prolonged minimal stress variation it will be advised for client 2 to undergo psychologist counselling and guidance to overcome this stress mutations. Which not treated now may cause chronic stress and PTSD in future.

HYPOTHESIS GENERATION AND DISCUSSIONS

Hypothesis 1: Does The environment has no significant impact of stress genes in human genome
Hypothesis 1A: Does The environment has significant impact of stress genes in human genome
Preposition the significant stress factors related with genes has direct and indirect influence towards the environment can be analysed using the neurogenetic testing concentrating only on the associated genes related to stress. The related stress genetic testing report shall give us the ideology of how much stress an employee will be undergoing from his past till present
Discussions: this genome testing shall explain the basic functional of the stress genes and its impact with values
Outcome:the neuro genetic testing could be a preventative measure which will be taken by the company to ensure the employees mental health as this neurogenetic testing concentrates only on stress genes If all these factors are considered properly with proper support we can easily overcome the psychological imbalance faced by humans
Hypothesis 2: Does the stress gene evaluation has no significant impact in psychological balance of humans
Hypothesis2a: Does the stress gene evaluation has significant impact in psychological balance of human
Hypothesis 3 :Does neuro genomics testing has no significance influence in enhancing the determination of stress related diseases in earlier stage of disease detection
Hypothesis 3a: Does neuro genomics testing has significance influence in enhancing the determination of stress related diseases

in earlier stage of disease detection

Name[iii]	Weighted Stress Gene Index (WSGI) (0–3 scale)	HPA–Serotonergic Composite Index (HSCI) (0–1 scale)	Overall Stress Factor (Relative)
Girija Anil Kumar	0.136	0.050	◈ Slightly elevated due to benign NR3C1 variant
Prof. Dr. Asha Sundaram	0.000	0.000	◍ Very Low (baseline normal)
Dr. Meenakshi N	0.000	0.000	◍ Very Low (baseline normal)
Surya S.	0.000	0.000	◍ Very Low (baseline normal)

Prepositions: the role of pathology in testing the stress related genes from blood sample will have significant impact in determining the stress factors that could be explained with weighted stress gene index and HPA composite index and relative overall stress factors.

Discussions: form the report obtained attached in annexure 1 we could infer the value of each employee and their relative stress values after computing with the stress value calculation formulas in terms of neurogenetic profiling Outcome:this ideal tabulation could make us self aware of how our environmental factors has impact over our neurogenes related to stress , and a concerned stress gene variance employee can be advised to take a necessary medications or counselling to undergo to overcome future stress related diseases.

Hypothesis 4: Does neurogenetic profiling could enhance the leadership skills of employees
Hypothesis 4A:Does neurogenetic profiling could enhance the leadership skills of employees
Hypothesis 6:: Can neurogenetic policies could improve the effectiveness of health of employees health benefits in the organisations
Hypothesis 6a:Can neurogenetic policies could improve the effectiveness of health of employees health benefits in the organisations
Preposition: the relationship with neurogenetic testing and leadership styles have been relation with each other based on the variance with stress genes predespositional factors that are identified through neurogenetic testing, AND THERE IS NO CORRESPONDING POLICIES IN HRM TO ADDRESS THE NEUROGENETIC PROFILING AND TO ADHERE THE BENEFITS OF IT
Discussions: in general with the out put that we have obtained from the report we could say that client 2 has variance in one of the stress genes which could indicate the stress coping capability should be less for the client 2 and corresponding stress variable of other clients doesn't possess any stress variance which could state that based on their further psychometric test a leader personality type can be identified

Outcome: the workload pattern and responsibility pattern for clients 1,3,4 can be designed based upon the neurogenetic testing report obtained and with stress variance client 2 the work load and responsibility allocation can be allocated less as the more workload may induce more stress for the client 2. The basic neurogenetic profiling legal amendments n the corporate settings needs the legislation to address the mental health preventative care to overcome shortcoming in their psychological imbalance as till now there are no support or legal aspect been addressing this issues in company settings

Hypothesis 5: Role of stress predispositions has NO significance impact in employee health support in organisations
Hypothesis 5A: Role of stress predispositions has significance impact in employee health support in organisations
Hypothesis 7:Can genetic evaluation has NO significance impact to categories the stress predispositions as high level risk employees, mid level risk employees and lose level risk of stress in employees
Hypothesis 7A: Can genetic evaluation has significance impact to categories the stress predispositions as high level risk employees, mid level risk employees and lose level risk of stress in employees.

Preposition: From the result obtained and calculated stress predespositions of the 4 clients, it has been defined that the stress predisposition has direct influence of employees mental health being and we are rejecting the null hypothesis and there is significance influence of stress genes caused due to environmental factors and this need proper considerations of health support b y adoption of neurogenetic testing.

Discussions: the relational aspect of determine the neurogenetic test obtaining the stress predespositions its been ideated to address the stress variance obtained for employees in corporate settings, thus to group them under the categories of high stress risk employees, middle stress link employees and lose stress link employees. Thus from the sample obtained client 2 can be placed under middle risk stress related employees and can be addressed with proper psychologist and neurogenetic doctors Outcome: preventative stage of psychological imbalance can be easily identified by the neurogenetic testing , thus the role of this stress predespositions testing can enhance the employee well-being by providing them the health insurance in terms of mental health

and necessary support to address the preventative care to mental health should be idealised in all employee settings to address the well being of employee mental health

FUTURE SCOPE OF RESEARCH AND CONCLUSION:

In general the role of law plays a major role in determining the human conduct in a county, when it comes to health and mind, mostly of the companies don't have policies to address the issues with health support and nor have awareness to know their brain stress related genes, thus in this context, this research concentrated more on the stress genes and their advantages to utilise the neurogenetic testing in more efficient and smartest manner and the application towards the test could variable contribute to address the mental health psychological imbalance of the employees in more efficient manner, thus the neuro cognitive resilience laws can be enacted to just address the mental health care access to every employee in the corporate settings thus this could ideally be described to address the stress predisposition in related stress genes and can variable contribute in deterring the employees prone to high risk of stress, medium risk of stress and low risk of stress related health issues.thus aiming to provide a stress free environment the management of the company should validate the neurogenetic profiling as one of the methods in recruiting the employees and to define the health benefits of each employees to adhere the provisions of genetic testing to address their stress which could ideally provide us the foundation to stress free company and necessary legal amendments should be made to address those issues.and would suggest that every company should adopt neurogenetic testing and profiling made mandatory.

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- [i] See ChatGPT (GPT-5), Comparative Analysis of Stress-Response Gene Variants and Stress Predisposition Indices from MedGenome Clinical DNA Reports (OpenAI, Nov. 12, 2025), https://chat.openai.com/.
- [ii] See ChatGPT (GPT-5), Comparative Analysis of Stress-Response Gene Variants and Stress Predisposition Indices from MedGenome Clinical DNA Reports (OpenAI, Nov. 12, 2025), https://chat.openai.com/.
- [iii] See ChatGPT (GPT-5), Comparative Analysis of Stress-Response Gene Variants and Stress Predisposition Indices from MedGenome Clinical DNA Reports (OpenAI, Nov. 12, 2025), https://chat.openai.com/.

ANNEXURE

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Total reads aligned (%)	99.99
Reads that passed alignment (%)	87.66
Data ≥ Q30 (%)	98.51

[$]The classification of the variants is done based on American College of Medical Genetics as described below [PMID:25741868] and strength based evidence(s). Details will be given upon request.

Variant	A change in a gene. This could be disease causing (pathogenic) or not disease causing (benign).
Pathogenic	A disease-causing variant in a gene which can explain the patient's symptoms has been detected. This usually means that a suspected disorder for which testing had been requested has been confirmed.
Likely Pathogenic	A variant which is very likely to contribute to the development of disease however, the scientific evidence is currently insufficient to prove this conclusively. Additional evidence is expected to confirm this assertion of pathogenicity.
Variant of Uncertain Significance	A variant has been detected, but it is difficult to classify it as either pathogenic (disease causing) or benign (non-disease causing) based on current available scientific evidence. Further testing of the patient or family members as recommended by your clinician may be needed. It is probable that their significance can be assessed only with time, subject to availability of scientific evidence.

[#]The transcript used for clinical reporting generally represents the canonical transcript (MANE Select), which is usually the longest coding transcript with strong/multiple supporting evidence. However, clinically relevant variants annotated in alternate complete coding transcripts could also be reported.

Variants annotated on incomplete and nonsense mediated decay transcripts will not be reported.

[#]The *in-silico* predictions are based on Variant Effect Predictor (v109), [SIFT version - 5.2.2; PolyPhen - 2.2.2; LRT version (November, 2009); CADD (v1.6); Splice AI; dbNSFPv4.2] and MutationTaster2 predictions are based on NCBI/Ensembl 66 build (GRCh38 genomic coordinates are converted to hg19 using UCSC LiftOver and mapped to MT2).

Diseases databases used for annotation includes ClinVar (updated on 20250227), OMIM (updated on 20052025), HGMD (v2024.4), LOVD (Nov-18), DECIPHER (population CNV) and SwissVar.

LIMITATIONS

- Genetic testing is an important part of the diagnostic process. However, genetic tests may not always give a definitive answer. In some cases, testing may not identify a genetic variant even though one exists. This may be due to limitations in current medical knowledge or testing technology. Accurate interpretation of test results may require knowing the true biological relationships in a family. Failing to accurately state the biological relationships in {my/my child's} family may result in incorrect interpretation of results, incorrect diagnoses, and/or inconclusive test results.
- Test results are interpreted in the context of clinical findings, family history and other laboratory data. Only variants in genes potentially related to the proband's medical condition are reported. Rare polymorphisms may lead to false negative or positive results. Misinterpretation of results may occur if the information provided is inaccurate or incomplete.
- Specific events like copy number variants, translocations, repeat expansions and chromosomal rearrangements may not be reliably detected with targeted sequencing. Variants in untranslated region, promoters and intronic variants are not assessed using this method.
- Genetic testing is highly accurate. Rarely, inaccurate results may occur for various reasons. These reasons include, but are not limited to: mislabeled samples, inaccurate reporting of clinical/medical information, rare technical errors

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MEDGENOME

DNA TEST REPORT - MEDGENOME LABS

Full Name / Ref No:	**GIRIJA ANIL KUMAR**	Order ID/Sample ID:	**1458644/9415294**
Gender:	Female	Sample Type:	Blood
Date of Birth / Age:	51 years	Date of Sample Collection:	24th September 2025
Referring Clinician:	Dr. Asha Sundaram, Saveetha School of Law, Chennai	Date of Sample Receipt:	25th September 2025
		Date of Order Booking:	25th September 2025
		Date of Report:	11th November 2025
Test Requested:	**Whole Exome Sequencing**		

CLINICAL DIAGNOSIS / SYMPTOMS / HISTORY

Ms. *Girija Anil Kumar* is suspected to harbour mutations in *CRHR1*, CRHR2, *NR3C1, FKBP5, SLC6A4 (5-HTTLPR), MAOA, BDNF, COMT, TPH2* genes and has been evaluated for pathogenic variations.

RESULTS

NO PATHOGENIC OR LIKELY PATHOGENIC VARIANTS CAUSATIVE OF THE REPORTED PHENOTYPE WERE DETECTED

VARIANT INTERPRETATION AND CLINICAL CORRELATION

No significant variant(s) for the given clinical indications that warrants to be reported was detected.
There are no clinically relevant variants in coding region and exon-intron boundaries of in *CRHR1, CRHR2, FKBP5, SLC6A4 (5-HTTLPR), MAOA, BDNF, COMT, TPH2* genes and the genes are 100% covered.

ADDITIONAL INFORMATION

- **A heterozygous nonsense variant in the *HTRA1* gene (c.1120G>T, p.Gly374Ter) has been detected in this assay. Kindly correlate clinically.**
- **A heterozygous variant (p.Ala49Val; c.146C>T) in the *NR3C1* gene was also detected in this assay. However, it has high MAF.**
- No significant SNV(s)/INDELS or CNV(s) that warrants to be reported were detected. All the genes covered in this assay have been screened for the given clinical indications. To view the coverage of all genes Click here. NGS test methodology details of this assay are given in the appendix.
- With regard to ACMG recommendations for reporting of incidental findings in clinical exome and genome sequencing (PMID: 35802134; ACMG SF v3.1), we report significant pathogenic and/ or likely pathogenic variants in the recommended genes for the recommended phenotypes, only if informed consent is given by the patient.
- Please write an email to genetic.counseling@medgenome.com in case you need assistance for genetic counselling. For any further technical queries please write an email to techsupport@medgenome.com

RECOMMENDATIONS

- Genetic counselling is advised.

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MEDGENOME

DNA TEST REPORT - MEDGENOME LABS

Full Name / Ref No:	**PROF DR. ASHA SUNDARAM**	Order ID/Sample ID:	**1458742/9415283**
Gender:	Female	Sample Type:	Blood
Date of Birth / Age:	48 years	Date of Sample Collection:	24th September 2025
Referring Clinician:	Dr. Asha Sundaram, Saveetha School of Law - Chennai,	Date of Sample Receipt:	25th September 2025
		Date of Order Booking:	25th September 2025
		Date of Report:	11th November 2025
Test Requested:	**Whole exome sequencing (80-100x)[Extended TAT]**		

CLINICAL DIAGNOSIS / SYMPTOMS / HISTORY

Prof Dr. Asha Sundaram is suspected to be harbor mutations in *CRHR1, CRHR2, NR3C1, FKBP5, SLC6A4, MAOA, BDNF, COMT, TPH2* genes and has been evaluated for pathogenic variations.

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295	122.91	0.3	99.66	99.45

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- The sensitivity of NGS assay to detect copy number variants (CNV) is 70-75%. We recommend discussing alternative testing methodology options with MedGenome Tech Support (techsupport@medgenome.com) as required. In case clinician is suspecting CNV as an important genetic etiology, alternate tests like microarray/ MLPA or qPCR may be considered after discussing with the MedGenome TechSupport team.

Sandhya Nair, Ph.D	Balaji Rajashekar, Ph.D	Dr. Sheeba Farooqui, MBBS, DNB (Ob Gyn), DM (Medical Genetics)
Sr. Manager - Variant Interpretation	Director - Clinical Bioinformatics	Consultant - Clinical Geneticist

APPENDIX

TEST METHODOLOGY

Targeted gene sequencing: Selective capture and sequencing of the protein coding regions and clinically relevant in the genome is performed. Variants identified in the exonic regions and splice-site are generally actionable compared to variants that occur in non-coding regions. Targeted sequencing represents a cost-effective approach to detect variants present in multiple/large genes in an individual.

DNA extracted from blood was used to perform targeted gene capture using a custom capture kit. The libraries were sequenced to mean depth of >80-100X on Illumina sequencing platform. We follow the GATK best practices framework for identification of germline variants in the sample using Sentieon [Sentieon]. The sequences obtained are aligned to human reference genome (GRCh38) using BWA aligner [Sentieon, PMID:20080505] and analyzed using Sentieon for removing duplicates, recalibration and re-alignment of indels [Sentieon]. Sentieon haplotype caller is then used to identify variants in the sample. The germline variants identified in the sample is deeply annotated using VariMAT pipeline. Gene annotation of the variants is performed using VEP program [PMID: 20562413] against the Ensembl release 104 human gene model [PMID: 34791404]. In addition to SNVs and small Indels, copy number variants (CNVs) are detected from targeted sequence data using the ExomeDepth method [PMID: 22942019]. This algorithm detects CNVs based on comparison of the read-depths in the sample of interest with the matched aggregate reference dataset.

Clinically relevant mutations in both coding and non-coding regions are annotated using published variants in literature and a set of diseases databases : ClinVar, OMIM, HGMD, LOVD, DECIPHER (population CNV) and SwissVar [PMID: 26582918, 18842627, 28349240, 21520333, 19344873, 20106818]. Common variants are filtered based on allele frequency in 1000Genome Phase 3, gnomAD (v3.1 & 2.1.1), dbSNP (GCF_000001405.38), 1000 Japanese Genome, TOPMed (Freeze_8), Genome Asia, and our internal Indian population database (MedVarDb v4.0) [PMID: 26432245, 32461613, 11125122, 26292667, 33568819, 31802016]. Non-synonymous variants effect is calculated using multiple algorithms such as PolyPhen-2, SIFT, MutationTaster2 and LRT. Clinically significant variants are used for interpretation and reporting.

Average sequencing depth (x)	Average on-target sequencing depth (x)	Percentage target base pairs covered		
		0x	≥5x	≥20x

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Average sequencing depth (x)	Average on-target sequencing depth (x)	Percentage target base pairs covered		
		0x	≥5x	≥20x
277	111.21	0.3	99.65	99.37

Total data generated (Gb)	10.38