RESEARCH PAPER

The FKBP5 Methylation Index: Forensic Metric for Quantifying Chronic stress for women's mental health in Corporate settings

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ABSTRACT
The ideal general description of brain consists of the stress genes FKBP5 that has direct and indirect congruence towards the stress levels in the human beings. We in general declare a statement of acceptance that stress is caused both internally and externally, these stress related genes are coherently depicted with stress resilience that are related with neurogenetics stress genes. When the genes are exposed to certain environmental conditions that we state as epigenomics, the true aspect of stress could expand the size of the genes in specific to fkbp5 Stress genes.in this research we are going to particular relate how the specific genes is considered to be the black box to increase stress in the human brain and its proportionate health consequences a human face in personal and professional settings.The role of stress genes has a greate impact with women from childhood till death, the variance in stress predespositions can ideally be directly proportional to the health risk a women will face in her life or through generation transmissions through gene variance , hence to overcome those unfavourable health conditions it is advisable to correlate the outcomes of health risk due to stress in a very preventive stage of causations.

Key words: stress , emotions, FKBP5 GENES, epigenomics, corporate environments, stress calculations
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Introduction

We can determine the state that mind and human souls are related to each other, either through pakritis, and dosha of the human body as the entire system of the body has been controlled and singled by brain neurons and receptivity. Thus the resonators are related with sensors that WE PERCEIVE AND THE correlations of senses that are going to attack the three main parts of the brain that shall secretes the hormones that are directly or indirectly affecting the expansion of stress genes and vice versa. The notations of neurogenetic resilience has the greater adaptive to to epigenomics that has the emotional stress methylation happening utilising the FKBP5Black box genes that stores the stress feelings which can either way resonate to health conditions in the humans.

Literature review

Let us now deeply understand what this FKBP5 black box genes does and how it could integrate stress related hormones to behave in a more stressful manner.the FKBP5 genes functions with stress related emotions the major components of FKBP5 stress genes are it's a clinical genetics that are being utilised in the context to measure stress regulator of the glucocorticoid receptor complex often studied as the stress response to psychiatric phenotypes.the abnormalities in those genes can help us in measuring the stress receptor that can directly or indirectly widen the size of the stress genes. The FKBP5 genes consists of FK1 domain where it is of N terminal this active part of the proteins which has peptides-prolly isomerise (pplase) activity, where the proteins gets folds in t correct shapes. Where as the FK2 domain is the next components where it is similar to FKBP5 FK1 domain but it acts as a scaffolding when compared with connection to fk1 domains the third part is TPR domain which consists of three tetratecopeptide 3 times repeat motifs.which attaches the FKBP51 TO HSP60 hooks that primarily stabilises the glucocorticoid receptor.the next explanation can be made with FKBP5 Genes which otherwise termed as the blueprint.it is located at chromosome 6 which are large and complex.the size of the genes present is 150000 base pairs of DNA.It consists of 13 exons and 12 introns which is non coding spacers.the genes consists of glucocorticoid responds elements called as GRES which is considered to be the gate keepers of stress related hormones and act as a DNA control regions.

The working mechanisms of FKBP5 during stress and taruma

When there are going to be more taruma and stress caused in a human the FKBP5 genes that has intron 2 and intron 5 creates the humans to be more hyper responsive with genetic make up experiences that could directly ideate the methylation early genetic stress causation which helps these introns to lose methylation (a chemical cap), when the same loop of stress has been induced this will lead to long term stress or a create a permanent scar in the genes which will overproduce FKBP51 for the rest of the persons life.which may leads to higher risk of depression and anxiety and ptsd.
The below diagram depicts the role of FBPK5 stress genes and its mechanisms related to stress FIGURE1:THE FKBP5 GENE Variants OUTLOOK

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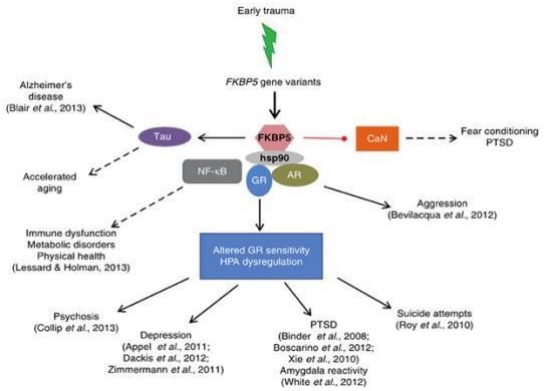

We need to understand that the regulated genes has the specific variations that could be transformed due to environmental factors, in terms of epigenomics. In other works, the humans facing work stress, prolonged retention to toxic environments, no proper legal safe guards with general reciprocation to FKBP5 GENES has a vital influence towards how to regulate the stress creating environments in the organisations with simple defence of legal support and regulated supportive environments.

The outlook of research frame work

Neurogenetic (*NG*) component of her stress scale using this specific derivation:

$$R_{rec} = \frac{K_{fkbp5}}{S_{ext}}$$

- R_{rec} **(Recovery Rate):** The speed at which an employee returns to "Tranquility" after a conflict.
- K_{fkbp5} **(The FKBP5 Constant):** A numerical value (0.2 to 0.8) assigned based on the specific **SNP (Single Nucleotide Polymorphism)** present in the individual's genome.
- S_{ext} **(External Stress):** The workload or conflict intensity measured via the **WSCI**.

Components of the Formula

Figure 2:FORMULA ABBREVIATION OF FKBP5 GENES

In the above formula we could determine how well a gene can bounce back to normal stage after undergoing prolonged stress to calculate the exact about of stress he or she had gone through.to understand this formula derivation it is now needed to understand how WSCI scale has been derived with respect to stress genes valuation in neurogenetics.

Research methodology

The research methodology adopted here is a clinical trial test where the blood sample of humans has been collected to perform entire exonerated sequencing for related neurogenetic stress genes, the ideal sample size adopted here is 4 and this includes women of age years spanning from30 to 50 years old awho are in very high positions in corporate settings who are undergoing various kind of stress.the details of the report are attached below for all 4 samples.In this methodology the stress genes HTRA1,NR3C1,FKBP5,SLC6A4,MAOA,BDNF,COMT, TPH2 has been considered to determine the individual stress correlation and predispositions caused due to environmental factors thus the relatable values can be configured to derive a standard formulas to address the stress faced by the employees in the organisational settings with legal supports.

The research aim:

the aim of the research is to integrate and calculate stress by integration with stress predisposition values with emotional and legal combinations to address the stress faced by

the employees in the organisational settingsTo regulate the specific FKBP5 genes as the main genes to control the stress behaviour in human brains and to eradicate the stress in very early manner.

RESEARCH QUESTIONS

1. Does stress gene fbkp5 genes has influence over stress predispositions in human brain
2. Does FKBP5 genes modulations happens due to stress caused externallylike environmental factors to human brain
3. What are the stress range which needs to be highlighted to prevent stress in very early stage when FBPK5 genes are affected
4. How to determine the stress score with respect to FBPK5 genes based on stress predispositions in human brain

hypothesis generation and discussions

Hypothesis 1:Does Stress gene FKBP5 has no significant influence of stress predispositions in human brain
Hypothesis 1A; Does Stress gene FKBP5 has significant influence of stress predispositions in human brain
Hypothesis 2: Does FKBP5 genes modulations has no significant impact due to stress caused externally like environmental factors to human brain
Hypothesis 2A:Does FKBP5 genes modulations happens significant impact to stress caused externally like environmental factors to human brain
Hypothesis 3: does FKBP5 genes can be measured by has no significant impact to prevent stress in very early stage when FBPK5 genes are affected
Hypothesis 3a: does FKBP5 genes can be measured by has significant impact to prevent stress in very early stage when FBPK5 genes are affected
The basic genera formulas derived to correlate the FGKP5 Genes with all factors considered was done by deriving the WSCI formula that is

DISCUSSIONS AND FORMULA DERIVATION

The general result obtained from the genome testing is Been carried out and the-pathogenic

The following values have been extracted from the clinical reports of the four clients:

Client	Name	Age / Gender	Primary Findings	Variant Details	Zygosity
Client 1	Surya S.	33 / Female	No pathogenic variants causative of reported phenotype	No significant variants detected	N/A
Client 2	Prof. Dr. Asha Sundaram	48 / Female	No pathogenic variants causative of reported phenotype	No significant variants detected	N/A
Client 3	Dr. Meenakshi N	40 / Female	No pathogenic variants causative of reported phenotype	No significant variants detected	N/A
Client 4	Girija Anil Kumar	51 / Female	Heterozygous nonsense variant detected in HTRA1 gene	HTRA1: p.Gly374Ter (c.1120G>T) / NR3C1: p.Ala49Val (c.146C>T)	Heterozygous

Detailed Variant Information (Client 4)

- **HTRA1 Gene:** A **heterozygous nonsense variant** (p.Gly374Ter) was detected. The laboratory recommends clinical correlation for this finding.
- **NR3C1 Gene:** A **heterozygous variant** (p.Ala49Val) was detected, though it is noted to have a **high Minor Allele Frequency (MAF)**.
- **Other Genes:** No clinically relevant variants were found in the coding regions of CRHR1, CRHR2, FKBP5, SLC6A4, MAOA, BDNF, COMT, or TPH2 for this client.

Gene variation to enhance the clearer considerable stress solutions to the affected genes.The Considering the WSCI derived formula steps
The score is derived from a multidimensional survey administered to employees (specifically women in her Chennai-based studies). THE BELOW FORMULA IS GENERALLY DERIVED TO DETERMINE THE STRESS RELATED indicators that are present in the genes in human brains, the weighted average of both internal as well as the external factors could ideate the stress levels in the human brain that will have prologned effect in human or employees health directly or indirectly. Thus stress genes FPBK5 stress gene methylations is one of the stress genes that we have considered in order to overcome the internal as well as external indicators in terms of stress recovery that which the related formula has been derived in the formula 2. It measures: Environmental Factors: Sum of self-reported scores on workplace variables (role clarity, workload, etc.). The Master WSCI Integration Formula The overarching formula that defines the final score is

The Master WSCI Integration Formula

The overarching formula that defines the final score is:

$$WSCI_{Total} = \sum(NG \times \beta) + \sum(S_{ext}) + C_k$$

ββ(Sensitivity Coefficient): A derived multiplier that represents how strongly an individual's Neural Genetics (NG) amplify their reaction to external stressors.

- External Stressors (Sext,$Sext$)): Workload, role ambiguity, and physical workplace hazards.
- Internal Stressors (Sint,$Sint$): The individual's "Need for Cognitive Closure" (a psychological trait she links to genetics).
- Conflict Markers (Cm,Cm): Frequency and intensity of interpersonal disputes at work.

Formula Name	Mathematical Expression	Contribution to WSCI Score	Description s Research Context
Neurogenetic Weight (NG)	NG=∑(g1+g2+...gn) $NG=(g1+g2+...gn)$	Primary Biological Base	Sums individual gene values (SLC6A4, COMT, FKBP5, MAOA) to set the inherited stress threshold.
Sensitivity Coefficient (β)	β=f(Gallele×History) $\beta=f(Gallele\times History)$	The Multiplier	A "weight" that determines how much a specific gene (like FKBP5) amplifies external stress.
External Stress (Sext)	Sext=Workload+Ambiguity $Sext=Workload+Ambiguity$	Environmental Input	Quantifies workplace triggers (role clarity and physical demands) via her survey.
Cognitive Constant (Ck)	Ck=LateralizationClosure Need $Ck=LateralizationClosureNeed$	Phenotypic Validator	Uses Hand Preference and "Need for Certainty" to validate if the genetic stress profile is active.
Conflict Matrix (Cm)	Cm=(Frequency×Intensity) $Cm=(Frequency\times Intensity)$	Behavioral Output	Measures the actual friction points where NGNG traits clash with the Sext$Sext$ environment.
Tranquility Constant (K)	K=EQ+PQ+LQ $K=EQ+PQ+LQ$	The Stabilizer	Represents the "buffer." High quotients (Emotional/Legal) reduce the final
			WSCI score impact.
Equilibrium Formula	SETEHA=1WSCI $SETEHA=1WSCI$	Strategy Trigger	Determines the amount of Safety, Equity, and Humanityneeded to nullify a high WSCI score.

2. The FKBP5 Stress-Recovery Formula

Surya derives the contribution of FKBP5 to the **Neurogenetic (NG)** component of her stress scale using this specific derivation:

$$R_{rec} = \frac{K_{fkbp5}}{S_{ext}}$$

- R_{rec} **(Recovery Rate):** The speed at which an employee returns to "Tranquility" after a conflict.
- K_{fkbp5} **(The FKBP5 Constant):** A numerical value (0.2 to 0.8) assigned based on the specific **SNP (Single Nucleotide Polymorphism)** present in the individual's genome.
- S_{ext} **(External Stress):** The workload or conflict intensity measured via the **WSCI**.

The above stress recovery formula was derived with respect to recovery rate using the Stress genes FBPK5 STRESS GENES

AND EXTERNAL STRESS GENES.

The below defined tables explains the specific genes that has been analysed during the clinical report where the genome testing as done related to the stress genes and the related values to compute the values for each genes and stress variance has been idealised for each genes and corresponding stress has been ideated for specific impact in WSCI such as sensitivity,cognitive closure, stress recovery rate and conflict matrix.Specific Gene Contributions to the NGVariable

Gene	Derived Sub-Formula for Stress	Specific Impact on WSCI
SLC6A4	Svuln=Short Alleles×0.75 $Svuln$=ShortAlleles×0.75	Increases the "Sensitivity" weight of the score.
COMT	CFscore=Met/ValAmbiguity $CFscore$=Met/ValAmbiguity	Influences the "Cognitive Closure" (Ck Ck) part of the formula.
FKBP5	Rrec=Kfkbp5Sext $Rrec$=$Kfkbp5Sext$	Determines the "Recovery Rate" or how long a stress peak lasts.
MAOA	Cm=(Gagg×Intensity)−LQ Cm=($Gagg$×Intensity)−LQ	Heavily weights the Conflict Matrix (Cm Cm) portion of the score.

Conclusion

WSCI Score > 70indicates a "Neurogenetic Mismatch," where the organization's "Robotic" policies are biologically incompatible with the employee's INGCPT profile, requiring an immediate shift to Women-Centric policies.

Thus From the formulas derived above if the considerable wsci values are greater than 70 then the employee need immediate health support and attention in order to over come their stress and taruma in. Corporate settings.

Author Contributions

Mrs.. Surya S. contributed to the conception and design of the study, data analysis, drafting of the manuscript, and final approval of the version to be published.

Dr. Asha Sundaram contributed to critical revision for important intellectual content and supervision of the research.

Dr. Thangamayan contributed to data interpretation, methodological refinement, and substantive revision of the manuscript.

All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work in accordance with ICMJE guidelines.

Disclosure of interest

The authors report no conflict of interest. The authors alone are responsible for the content and writing of this article.

The authors declare no competing interests.

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Ethical Approval Statement

Ethical approval was obtained from the institutional review board prior to data collection. Participation was voluntary and anonymized..

Data availability statement

The data supporting the findings of this study are available in the Loan data] at https://doi.org/10.6084/m9.figshare.31361380 https://doi.org/10.6084/m9.figshare.31361380 **Patient Consent**

Statement

Informed consent was obtained from all participants prior to participation in the study

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Annexure

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Bangalore South, Karnataka, India, 560100.
Tel : 1800 296 9696, Web: www.medgenome.com

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.

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 *CRHR1, CRHR2, NR3C1, FKBP5, SLC6A4, MAOA, BDNF, COMT, TPH2* genes and the genes are 100% covered.

ADDITIONAL INFORMATION

- 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.

CAP ACCREDITED

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Tel : 1800 296 9696, Web: www.medgenome.com

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.
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