ROLE OF ESTROGEN RECEPTOR ALPHA (ESR1) IN MENTAL STRESS: MOLECULAR MECHANISMS AND THERAPEUTIC PERSPECTIVES : REVIEW ARTICLE

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ABSTRACT

Mental stress is a major contributor to the development of neuropsyatric disorders, including depression, anxiety, and cognitive dysfunction. Increasing evidence indicates that the Estrogen Receptor Alpha (ESR1) plays a pivotal role in regulating the molecular and cellular mechanisms underlying stress resilience and emotional homeostasis. ESR1 is abundantly expressed in brain regions involved in stress processing, such as the hypothalamus, hippocampus, amygdala, and prefrontal cortex, where it modulates neuroendocrine signaling, neurotransmitter systems, synaptic plasticity, neuroinflammation, and oxidative stress. Through both genomic and non-genomic signaling pathways, ESR1 regulates the hypothalamic–pituitary–adrenal (HPA) axis, enhances brain-derived neurotrophic factor (BDNF) expression, promotes neuronal survival, and suppresses inflammatory mediators including NF-κB, IL-1β, IL-6, and TNF-α. Genetic polymorphisms in ESR1 have also been associated with individual differences in susceptibility to stress-related psychiatric disorders. Experimental studies using ESR1-deficient animal models and clinical investigations in humans further support its neuroprotective and stress-modulating functions. This review summarizes the structural and biological characteristics of ESR1, its molecular mechanisms in mental stress, evidence from experimental and clinical studies, therapeutic potential, and future research directions. A better understanding of ESR1-mediated signaling may facilitate the development of brain-selective estrogen receptor modulators and personalized therapeutic strategies for stress-associated neuropsychiatric disorders.

Keywords: ESR1, Estrogen Receptor Alpha, Mental Stress, HPA Axis, Neuroinflammation, BDNF, Neuroplasticity, Anxiety, Depression, Neuroprotection.

INTRODUCTION

Mental stress activates adaptive physiological responses that are essential for survival; however, chronic or excessive stress can disrupt neuroendocrine homeostasis and promote psychiatric illness. The estrogen signaling pathway has emerged as an important regulator of emotional behavior and stress resilience. ESR1, one of the principal estrogen receptors, is widely expressed in brain regions involved in mood regulation, including the hypothalamus, hippocampus, amygdala, and prefrontal cortex.[1]

Accumulating evidence indicates that ESR1-mediated signaling influences stress hormone secretion, neurotransmission, neuroplasticity, inflammatory responses, and gene expression, making it a key component in the biological response to mental stress.[2]

Structure and Biological Function of ESR1

ESR1 encodes estrogen receptor alpha (ERα), a member of the nuclear receptor superfamily. Upon binding estrogen, ERα undergoes conformational changes that enable it to regulate target gene transcription through estrogen response elements (EREs) or through interactions with transcription factors such as AP-1 and SP-1.[3,40]

Apart from genomic actions, ESR1 also mediates rapid non-genomic signaling through kinase pathways including PI3K/Akt, MAPK/ERK, and cAMP signaling cascades. These pathways influence neuronal survival, synaptic remodeling, and cellular stress responses.[4]

The Estrogen Receptor Alpha (ESR1) gene encodes estrogen receptor alpha (ERα), a ligand-activated transcription factor belonging to the nuclear receptor superfamily. ERα mediates many of the biological effects of estrogens in reproductive tissues, the cardiovascular system, bone, immune cells, and the central nervous system. In the brain, ESR1 is highly expressed in the

hypothalamus, hippocampus, amygdala, and prefrontal cortex, where it regulates stress responses, emotional behavior, cognition, and synaptic plasticity.[5,6,42]

Structural Organization of ESR1

The human **ESR1** gene is located on **chromosome 6q25.1** and encodes a protein of approximately **595 amino acids**. ERα consists of six functional domains (A–F), each with distinct biological roles.[6,41,43]

Table: 1 Structural Organization of ESR1 [5,6,7]

Domain	Approximate Position	Function
A/B (AF-1 domain)	N-terminal	Ligand-independent transcriptional activation; recruits co-regulatory proteins.
C (DNA-binding domain, DBD)	Highly conserved zinc-finger region	Binds to estrogen response elements (EREs) in target genes.
D (Hinge region)	Connects DBD and LBD	Provides flexibility and contains nuclear localization signal.s
E (Ligand-binding domain, LBD/AF-2)	C-terminal core	Binds estradiol, mediates receptor dimerization, and recruits coactivators.
F domain	Extreme C-terminus	Modulates receptor activity in a tissue-specific manner.

Genomic Signalling Pathway
The classical mechanism of ESR1 signaling involves direct regulation of gene transcription:

1. **17β-Estradiol (E2)** binds to ERα in the cytoplasm or nucleus.
2. The receptor undergoes a conformational change and forms **homodimers** or **heterodimers**.
3. Activated ERα binds to **Estrogen Response Elements (EREs)** in DNA.
4. Coactivators (e.g., SRC family proteins and CBP/p300) or corepressors are recruited.
5. Transcription of estrogen-responsive genes is altered, affecting neuronal survival, inflammation, metabolism, and stress adaptation.[8,10,44,]

Non-Genomic (Rapid) Signaling

A subset of ERα is associated with the plasma membrane and initiates rapid intracellular signaling independent of direct DNA binding.

Major pathways include:

- **PI3K/Akt pathway** → promotes neuronal survival and anti-apoptotic signaling.
- **MAPK/ERK pathway** → regulates synaptic plasticity and memory.
- **cAMP/PKA signaling** → modulates neurotransmitter release.
- **Ca^{2+}-dependent pathways** → influence neuronal excitability and calcium homeostasis.

These rapid responses occur within seconds to minutes after estrogen exposure.[10,11,45]

Table: 2 Biological Functions of ESR1 [9,10,49,50]

Biological Function	Mechanism	Physiological Importance
Gene regulation	ERE-mediated transcription	Controls expression of hundreds of estrogen-responsive genes.
Neuroprotection	Activates Akt and ERK signaling	Protects neurons from apoptosis and oxidative stress.
Synaptic plasticity	Increases dendritic spine density and BDNF expression	Improves learning and memory.
Stress regulation	Modulates hypothalamic–pituitary–adrenal (HPA) axis activity	Limits excessive cortisol responses.
Neurotransmitter modulation	Influences serotonin, dopamine, glutamate, and GABA systems	Regulates mood and emotional processing.
Anti-inflammatory action	Suppresses NF-κB signaling and cytokine production	Reduces neuroinflammation.
Antioxidant defense	Enhances cellular antioxidant pathways	Protects against reactive oxygen species.

ESR1 in Mental Stress

In the context of mental stress, ESR1 contributes to resilience through several interconnected mechanisms:

- **Regulation of the HPA axis:** ESR1 modulates corticotropin-releasing hormone (CRH) and glucocorticoid signaling, helping maintain appropriate stress responses.
- **Neurotransmitter balance:** It influences serotonergic, dopaminergic, glutamatergic, and GABAergic neurotransmission, which are critical for mood regulation.
- **Promotion of BDNF expression:** ESR1 enhances brain-derived neurotrophic factor (BDNF), supporting neuronal survival and synaptic remodeling.
- **Reduction of neuroinflammation:** ERα signaling inhibits inflammatory mediators such as **NF-κB**, **IL-1β**, **IL-6**, and **TNF-α**.
- **Protection against oxidative stress:** ESR1 activation improves antioxidant defenses and reduces stress-induced neuronal damage.[12,13 14,,46,47,48]

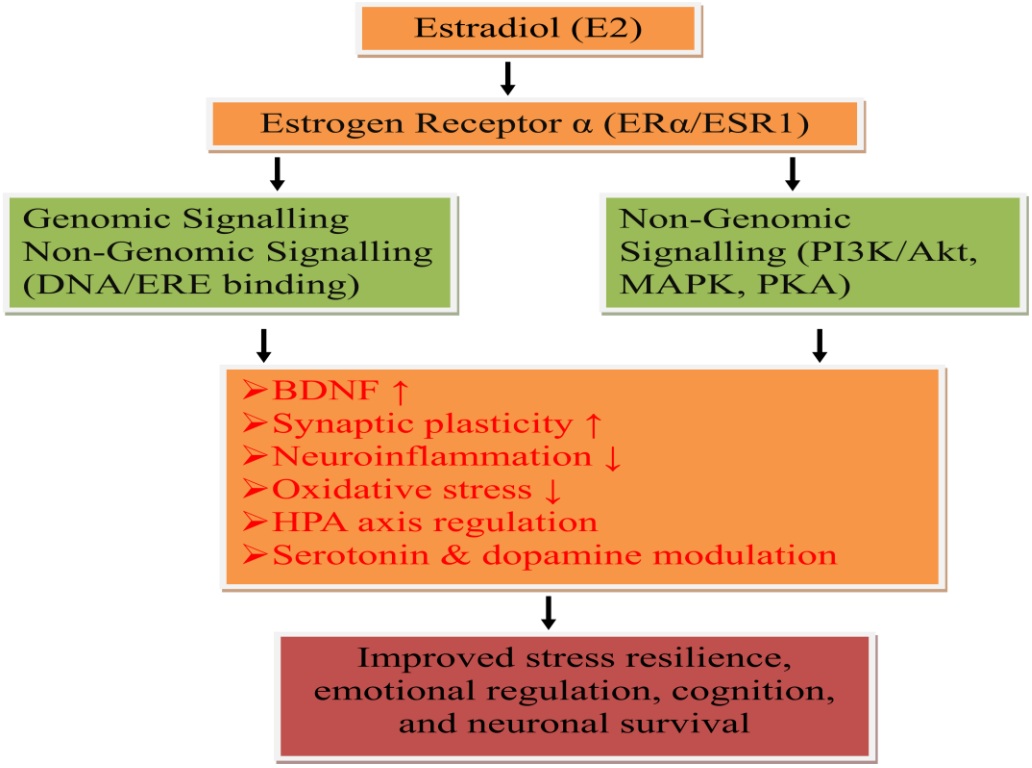

Figure: 1 Mechanism of ESR1 in Mental Stress

ESR1 Expression in the Central Nervous System

ERα is abundantly expressed in several brain regions associated with emotional and cognitive processing:

- Hypothalamus
- Hippocampus
- Amygdala
- Prefrontal cortex
- Bed nucleus of the stria terminalis

Its distribution supports important roles in regulating stress responses, memory formation, reproductive behavior, and neuroendocrine function.[15,16,51,52]

ESR1 and Regulation of the HPA Axis

The hypothalamic–pituitary–adrenal (HPA) axis represents the principal endocrine system activated during stress. Estrogen signaling through ESR1 modulates secretion of corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and glucocorticoids.[17,20,52,54]

Experimental studies suggest that ESR1 influences glucocorticoid feedback sensitivity and alters stress hormone release. Dysregulation of ESR1 signaling may therefore contribute to prolonged cortisol exposure and impaired stress adaptation, which are commonly observed in depression and anxiety disorders.[18,22,55,56]

Molecular Mechanisms Linking ESR1 to Mental Stress

1 Modulation of Neurotransmitter Systems

ESR1 regulates several neurotransmitter pathways implicated in stress resilience:

- Enhances serotonergic neurotransmission
- Influences dopamine synthesis and receptor activity
- Modulates glutamatergic signaling
- Regulates GABAergic inhibitory circuits

These effects collectively impact mood, emotional regulation, and cognitive performance.[19,20,18,32]

2 Neuroprotection and Synaptic Plasticity

Activation of ESR1 promotes neuronal survival by increasing anti-apoptotic signaling and reducing oxidative stress. It also stimulates dendritic spine formation and synaptic remodeling, supporting learning and memory during stressful conditions.[21,38,42]

3 Anti-inflammatory Effects

Chronic stress induces neuroinflammation characterized by microglial activation and elevated cytokines such as IL-1β, IL-6, and TNF-α. ESR1 signaling suppresses inflammatory gene expression through inhibition of NF-κB pathways, thereby protecting neural tissue from inflammatory injury.[22,23,36]

4 Regulation of Brain-Derived Neurotrophic Factor (BDNF)

Estrogen signaling through ESR1 enhances expression of BDNF, a critical mediator of neuronal plasticity and resilience. Reduced BDNF levels have been associated with chronic stress and major depressive disorder, suggesting that ESR1-mediated BDNF regulation contributes to adaptive stress responses.[24,28,31]

Genetic Variations in ESR1 and Stress Susceptibility

Several polymorphisms within the ESR1 gene have been associated with variability in emotional behavior and psychiatric risk. Commonly studied variants include:

- PvuII (rs2234693)
- XbaI (rs9340799)

These polymorphisms may alter receptor expression or transcriptional activity and have been investigated in relation to depression, anxiety, cognitive decline, and stress sensitivity, although findings remain heterogeneous across populations.[24,25,27]

Table 3. Molecular Mechanisms by Which ESR1 Influences Mental Stress[26, 29]

Mechanism	Key Molecular Events	Physiological Outcome
Genomic signaling	Binding to estrogen response elements (EREs)	Regulation of stress-responsive gene transcription
Non-genomic signaling	Activation of PI3K/Akt and MAPK/ERK pathways	Rapid neuroprotective signaling
BDNF induction	Increased BDNF transcription	Enhanced synaptic plasticity and resilience
Anti-inflammatory signaling	Inhibition of NF-κB-mediated cytokine production	Reduced neuroinflammation
Oxidative stress reduction	Increased antioxidant enzyme activity	Protection against neuronal damage

Mechanism	Key Molecular Events	Physiological Outcome
Epigenetic regulation	Interaction with chromatin remodeling proteins	Long-term adaptation to stress

Evidence from Animal Studies

Rodent models demonstrate that disruption of ESR1 signaling produces behavioral abnormalities resembling anxiety and depression. Conversely, pharmacological activation of ERα often reduces stress-induced behavioral deficits and improves cognitive performance.

ESR1 knockout mice exhibit altered HPA axis responses, impaired emotional regulation, and changes in synaptic plasticity, supporting a direct mechanistic role for ESR1 in stress adaptation.[26,28]

Table 4: Evidence from Experimental Studies[27,28,39,49]

Model	Major Findings	Implications
ESR1 knockout mice	Increased anxiety-like behavior and altered HPA axis regulation	ESR1 is protective against stress
Chronic restraint stress models	Reduced ESR1 expression with prolonged stress	Stress may impair estrogen signaling
Estrogen-treated rodents	Improved cognition and reduced depressive-like behaviors	ESR1 activation enhances resilience
Cell culture studies	Increased neuronal survival following ESR1 activation	Supports neuroprotective functions

Clinical Evidence in Humans

Clinical studies indicate that estrogen fluctuations during puberty, postpartum periods, and menopause are associated with altered mood and stress vulnerability. Women carrying specific ESR1 polymorphisms may exhibit increased susceptibility to depressive symptoms under stressful conditions.

Neuroimaging and molecular studies also suggest that ESR1-related signaling influences functional connectivity in limbic brain circuits involved in emotional regulation.[29,30,39]

Table 5: Clinical Evidence Linking ESR1 to Mental Stress[31,32,38,57]

Clinical Observation	ESR1-Related Finding
Major depressive disorder	Altered ESR1 expression and polymorphism associations
Generalized anxiety disorder	Potential contribution of ESR1 variants to susceptibility
Menopause-associated mood disorders	Reduced estrogen signaling linked to increased stress sensitivity
Postpartum depression	Hormonal fluctuations involving ESR1 pathways may contribute
Cognitive decline under chronic stress	ESR1-mediated neuroprotection may mitigate impairment

Therapeutic Implications

Targeting ESR1 represents a promising strategy for managing stress-related neuropsychiatric disorders. Potential approaches include:

- Selective estrogen receptor modulators (SERMs)
- Brain-selective ERα agonists
- Combination therapies with antidepressants
- Personalized interventions based on ESR1 genotype

However, systemic estrogen therapy carries risks, including cardiovascular and hormone-sensitive cancer concerns, emphasizing the need for tissue-selective modulation.[33,34,56]

Table 6: Therapeutic Strategies Targeting ESR1[35,36,60]

Strategy	Proposed Mechanism	Advantages	Limitations
Estrogen replacement therapy	Restores estrogen signaling	Improves mood in selected	Systemic adverse effects

Strategy	Proposed Mechanism	Advantages	Limitations
		populations	
Selective estrogen receptor modulators (SERMs)	Tissue-selective activation/inhibition	Reduced peripheral risks	Variable CNS efficacy
Brain-selective ERα agonists	Target CNS ESR1 pathways	Potential neuroprotection	Mostly experimental
Combination with antidepressants	Synergistic enhancement of neuroplasticity	May improve treatment response	Requires further clinical trials
Personalized medicine	Therapy guided by ESR1 genotype	Precision treatment	Limited clinical implementation

Future Perspectives

Future research should focus on:

- Sex-specific mechanisms of ESR1 signaling
- Epigenetic regulation of ESR1 during chronic stress
- Interactions between ESR1 and glucocorticoid receptors
- Development of CNS-selective ERα modulators
- Large-scale genomic studies investigating ESR1 polymorphisms and psychiatric disorders[37,38,58,59,]

CONCLUSION

ESR1 has emerged as a key molecular regulator of the brain's response to mental stress through its diverse actions on neuroendocrine, neurotransmitter, inflammatory, and neuroplasticity pathways. By modulating the HPA axis, regulating serotonergic, dopaminergic, glutamatergic, and GABAergic neurotransmission, enhancing BDNF-mediated synaptic plasticity, and suppressing oxidative stress and neuroinflammation, ESR1 contributes significantly to stress resilience and maintenance of emotional homeostasis. Experimental studies consistently demonstrate that impairment of ESR1 signaling increases vulnerability to anxiety- and depression-like behaviors, whereas activation of ERα provides neuroprotective and antidepressant-like effects. Clinical evidence further suggests that ESR1 polymorphisms and altered receptor expression influence susceptibility to stress-related psychiatric disorders, particularly during periods of hormonal fluctuation such as puberty, pregnancy, postpartum, and menopause.

Despite substantial progress, several challenges remain regarding the precise mechanisms of ESR1 signaling in different brain regions, sex-specific responses, epigenetic regulation, and interactions with other stress-related signaling pathways. Future research integrating molecular biology, genomics, neuroimaging, and clinical trials will be essential for translating these findings into therapeutic applications. The development of brain-selective ERα agonists, selective estrogen receptor modulators (SERMs), and personalized treatment strategies based on ESR1 genetic profiles represents a promising direction for improving the prevention and treatment of stress-associated neuropsychiatric disorders. Overall, ESR1 represents an attractive therapeutic target with significant potential to advance precision medicine approaches for mental stress and related psychiatric diseases.

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