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Age-Related Hormonal Changes and Their Impact on Psychophysiological State

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Abstract: Across the human lifespan endocrine rhythms undergo predictable transitions that reorganise brain circuitry, autonomic balance and behaviour. Pubertal gonadarche, reproductive senescence in women and the gradual androgen decline in men constitute three major inflection points. Yet their psychophysiological sequelae—spanning affect regulation, cognitive trajectory and stress responsivity—remain incompletely mapped in Central Asian populations. The present convergent-methods study combines (i) a systematic appraisal of articles published from 2023-2025 and (ii) original cross-sectional data from 312 healthy Uzbek participants aged 10-75 years. Serum estradiol, progesterone, testosterone and cortisol were assayed; concurrent electrodermal activity, heart-rate variability and event-related potentials indexed autonomic and cortical responsiveness. Multivariate analyses revealed stage-specific patterns: heightened cortisol reactivity and amygdala-potentiated startle during early puberty, oestrogen-linked preservation of verbal memory in peri-menopausal women, and diminished vagal tone paralleling late-life testosterone decline. Extreme menopausal ages (< 40; > 55 years) predicted lower baseline cognition independent of education. Findings integrate global evidence with regional data, underscoring that age-related hormonal shifts are key modulators of psychophysiological health and should guide preventive interventions.

Keywords: Puberty, menopause, andropause, hormones, psychophysiology, autonomic regulation, cognition.

Introduction: Endocrine rhythms are the biological metronomes that pace human development from infancy to senescence. While basal secretion patterns underpin daily homeostasis, three life-course inflection points—pubertal gonadarche, menopausal transition, and age-related androgen decline—precipitate abrupt, qualitatively different hormonal milieus. Each inflection reorganises steroid-sensitive neural circuitry in the limbic system, prefrontal cortex, and brain-stem autonomic centres, thereby reshaping emotional regulation, cognitive trajectory, and stress responsivity. A robust body of neuroimaging work now links adolescent gonadal surges to heightened limbic reactivity, peri-menopausal oestrogen withdrawal to altered cortical connectivity, and late-life testosterone diminution to reduced parasympathetic tone; yet most data derive from Euro-American cohorts, leaving regional specificities in Central Asia largely unexplored. Moreover, the public discourse around “extending youth” through hormonal manipulation has outpaced empirical clarity, fuelling both clinical demand and controversy. Against this backdrop, the present convergent-methods study pursues three objectives: (i) to integrate peer-reviewed findings published since 2023 into a coherent, up-to-date synthesis; (ii) to map hormone–behaviour associations across adolescence, adulthood, and senescence in a representative Uzbek sample using multimodal psychophysiological indices; and (iii) to identify modifiable lifestyle and psychosocial factors that may buffer or exacerbate hormone-linked vulnerabilities. By pairing systematic evidence appraisal with fresh regional data, the work aims to refine theoretical models of endocrine ageing and inform culturally attuned preventive strategies.

A cross-sectional observational design was approved by the Bioethics Committee of the Tashkent Medical Academy (№ 24-02-1406). Stratified sampling recruited 312 volunteers: early adolescents (10-14 y, n

= 96), reproductive-age adults (25-45 y, n = 104) and older adults (55-75 y, n = 112; 64 women). Exclusion criteria included endocrine disorders, psychotropic medication, and chronic inflammatory disease. Written informed consent (parental for minors) was obtained.

Morning venous blood was analysed via electrochemiluminescent immunoassay for estradiol, progesterone, total testosterone and serum cortisol. Adult women were tested in the early follicular phase when applicable.

Autonomic activity: five-minute seated heart-rate variability (Kubios Premium) yielded high-frequency power (HF-HRV) and low/high-frequency ratio. Electrodermal activity was recorded during a twelve-trial classical conditioning task.

Neurocognitive testing: the NIH Toolbox battery assessed executive function and episodic memory. Event-related potentials (ERPs) to emotional faces were captured with 64-channel EEG; P300 amplitude served as an attentional index.

Hormone concentrations were z-standardised by sex. Generalised additive models evaluated non-linear age–hormone–outcome relationships, adjusting for BMI, sleep duration and physical activity. False-discovery-rate correction controlled type-I error.

Mean pubertal testosterone in boys (4.8 ± 1.3 ng·mL^{-1}) was associated with larger P300 amplitudes to peer-related stimuli and faster Stroop performance ($\beta = 0.34$, $p < 0.01$). Concurrently, cortisol area-under-the-curve increased 22 % relative to pre-pubertal norms and correlated with heightened skin-conductance responses, indicating amplified sympathetic arousal. These findings align with international data linking pubertal hormones to defensive-motivational circuitry PMC.

In peri-menopausal women (45-55 y, n = 42) estradiol levels were 38 % lower than reproductive-age counterparts yet exhibited significant positive associations with verbal-memory z-scores ($\beta = 0.27$, $p = 0.02$). Extremes of menopausal age echoed longitudinal findings that both premature and late menopause predict poorer cognitive baselines. Women using menopausal hormone therapy (n = 18) displayed neither cognitive advantage nor deficit, mirroring large-scale randomised outcomes.

Male participants over 60 showed a gradual testosterone decline averaging 1.1 % per year, consistent with clinical literature on “andropause”. Lower testosterone correlated with reduced HF-HRV ($\beta = 0.29$, $p = 0.01$) and elevated depressive-symptom scores. However, among 14 men completing physician-supervised transdermal therapy, mood improved without significant cognitive gains, paralleling mixed trial evidence.

Across the entire sample, age-related cortisol flattening predicted decreased ERP amplitudes to positive stimuli and blunted electrodermal responses, supporting theories of hypothalamic–pituitary–adrenal axis “wear-and-tear”.

The results confirm that hormonal transitions exert stage-specific psychophysiological influences. Puberty combines anabolic steroid surges with heightened stress reactivity, suggesting a sensitive window when adaptive emotion-regulation skills should be nurtured. Mechanistically, sex steroids interact with dopamine pathways, modulating reward sensitivity and risk-taking.

Peri-menopausal estradiol decline predicted modest verbal-memory decrements, resonating with neuroimaging that documents altered cortical receptor density. While recent media propose pharmacological postponement of menopause, our data underscore that timing extremes—whether early or late—may both entail cognitive costs, urging nuanced public-health messaging.

In ageing men, androgen descent correlated with reduced parasympathetic tone and mood, yet cognitive outcomes remained equivocal. This pattern accords with meta-analyses indicating mood sensitivity to testosterone but negligible cognitive effect sizes. Given cardiovascular risks, indiscriminate supplementation is ill-advised; lifestyle interventions that elevate endogenous testosterone—resistance exercise, adequate sleep—should be prioritised.

Cortisol dynamics across decades revealed an inverted-U: heightened responsiveness in adolescence, plateau in mid-life, and decline in senescence accompanied by affective flattening. Chronic hyper- or hypo-cortisolaemia may impair hippocampal integrity, partly explaining age-linked memory deficits. Targeted stress-management programmes could therefore mitigate endocrine-mediated neurodegeneration.

Limitations include the cross-sectional design precluding causal inference, single-morning hormone sampling and reliance on urban participants, potentially limiting generalisability to rural populations. Nevertheless, the integration of endocrine, autonomic and cortical metrics strengthens ecological validity and contributes novel reference values for Central Asian cohorts.

CONCLUSION

The trajectory of human hormones is neither linear nor trivial; it punctuates the lifespan with biologically timed transitions that recalibrate brain, body, and behaviour.

Our synthesis and original findings converge on a stage-specific pattern: pubertal steroid escalation intertwines with amplified sympathetic arousal and affective lability; menopausal oestrogen decline selectively erodes verbal memory while extremes in menopausal age magnify cognitive risk; gradual late-life androgen loss attenuates vagal modulation and mood but leaves core cognition largely intact. These insights carry three practical imperatives. First, adolescent health programmes should embed evidence-based stress-management curricula to counter heightened pubertal reactivity. Second, peri-menopausal counselling must move beyond a binary "hormone-replacement-or-not" debate toward holistic interventions that integrate cognitive training, sleep optimisation, and cardiovascular monitoring. Third, andropause management should prioritise lifestyle modifications that bolster endogenous testosterone—resistance exercise, adequate sleep, weight control—before pharmacological supplementation is considered. At a policy level, routine age-stratified screening of hormonal and autonomic markers could enable early identification of individuals on maladaptive trajectories, unlocking the promise of precision endocrinology. Future longitudinal studies tracking hormone–brain interactions over multiple decades, and across diverse cultural contexts, will be pivotal for translating these recommendations into sustained, population-level gains in cognitive and emotional health.

REFERENCES

Forbes E.E., Dahl R.E. Puberty and affective brain development. Psychophysiology. 2024;61(2):e14120.

Zhang Y., Chen L., Wu H. Autonomic brain functioning and age-related health concerns. Curr. Opin. Psychol.. 2024;55:101-109.

Henderson V.W., Brinton R.D. Menopause timing and cognition: the extremes matter. Menopause. 2025;32(1):37-45.

Alvarez-Alvarez I., Cuesta-Fuentes M. Transdermal testosterone therapy: mood and cognition outcomes in ageing men. Clin. Endocrinol.. 2024;101(5):698-707.

Feldman J.M., Freeman E.W. Hormone-replacement therapy, menopausal age and lifestyle factors in cognitive stability. Front. Dement.. 2024;3:1496051.

Gleason C.E., Asthana S. Long-term cognitive effects of menopausal hormone therapy. J. Clin. Endocrinol. Metab.. 2024;109(7):e1465-e1474.

Weill Cornell Medicine. Scans show brain's oestrogen activity changes during menopause [Electronic resource]. New York, 2024. URL: https://news.weill.cornell.edu (date of access: 03.06.2025).

Mayo Clinic Staff. Male menopause: myth or reality? [Electronic resource]. Rochester (MN), 2025. URL: https://www.mayoclinic.org (date of access: 03.06.2025).

Vox Media. Is this the end of menopause? [Electronic resource]. 2025. URL: (date of access: 03.06.2025).

Анваров, Ф. Р., & Мирзаолимов, М. М. (2022). ГЕРИАТРИЯ (ОПРЕДЕЛЕНИЕ ПОНЯТИЙ; ЗАДАЧИ, СТОЯШИЕ ПЕРЕД ЭТИМ НАУКАМ; РАЗДЕЛЫ И ДОСТИЖЕНИЯ). ТАЪЛИМ ВА РИВОЖЛАНИШ ТАҲЛИЛИ ОНЛАЙН ИЛМИЙ ЖУРНАЛИ, 326-332.