Around the World 3

New Perspectives on BPSD Management: Evolutionary Silence, Use-Dependent Activation, and Applications in Japan

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Highlights:

Shift from pharmacological to non-pharmacological interventions: While traditional research on BPSD has emphasized medications, this study highlights innovative non-drug approaches.

Introduction of new theoretical framework: The paper proposes the concept of the *"evolutionary silence period"* and the strategy of *"use-dependent activation"* as a foundation for understanding and addressing BPSD.

Practical care strategies: It emphasizes interventions such as hydration management, elimination support, physical activity, oral feeding, and social engagement as effective methods to alleviate BPSD and improve quality of life.

1. Behavioral and Psychological Symptoms of Dementia (BPSD)

In addition to core symptoms such as memory impairment and cognitive decline, dementia is frequently accompanied by a range of non-cognitive psychiatric and behavioral problems collectively known as Behavioral and Psychological Symptoms of Dementia (BPSD). These include anxiety, depression, hallucinations, delusions, aggression, wandering, circadian rhythm disturbances, emotional instability, and refusal of care. The prevalence of BPSD is remarkably high; epidemiological studies indicate that more than 80% of individuals with dementia will experience some form of BPSD during the course of the disease.

The impact of BPSD on patients' quality of life is profound. First, it directly undermines daily functioning, leading to further difficulties in eating, sleeping, elimination, and social interaction, thereby diminishing autonomy and a sense of dignity. Second, BPSD intensifies patients' subjective suffering—for example, hallucinations and delusions may trigger fear, while depression fosters despair and helplessness—thus exacerbating the overall disease burden. Third, BPSD is often the primary reason why patients are forced into institutional care, since its unpredictability and risks surpass those posed by memory impairment alone.

At the caregiving level, BPSD substantially increases stress for both families and institutions. Caregivers frequently experience anxiety, insomnia, and burnout as they cope with aggression, wandering, or nighttime awakenings, resulting in escalating caregiving costs and medical expenditures. Although pharmacological treatment can partially relieve symptoms, it is often limited in efficacy and accompanied by adverse effects. Consequently, BPSD has become a central challenge and research hotspot in dementia care. In the future, identifying effective non-pharmacological interventions—such as environmental optimization, use-dependent activation training, and social support—will be crucial not only to improving patients' quality of life but also to alleviating the caregiving burden on families and society.

2. The Concept of Evolutionary Silence and Use-Dependent Activation in Aging

Evolutionary silence is an important concept proposed in the science of aging, referring to the gradual entry of the organism into a low-power operating state

under conditions of energy limitation and evolutionary adaptive pressures. This mechanism manifests across four levels: biologically, cellular metabolism along with autophagy and repair functions gradually downregulate, reducing energy consumption but accumulating damage; physiologically, organ systems prioritize the preservation of survival cores (cardiac and circulatory functions), while non-core functions such as cognition and muscle activity fall silent first; psychologically, attention, emotional regulation, and the ability to learn new information weaken, leading individuals to increasingly rely on habitual and low-energy cognitive strategies; and socially, role participation declines, social networks contract, and behavior tends toward an "energy-conserving" mode. Although this comprehensive silent state has adaptive significance, it simultaneously creates vulnerabilities for dementia, depression, and BPSD.

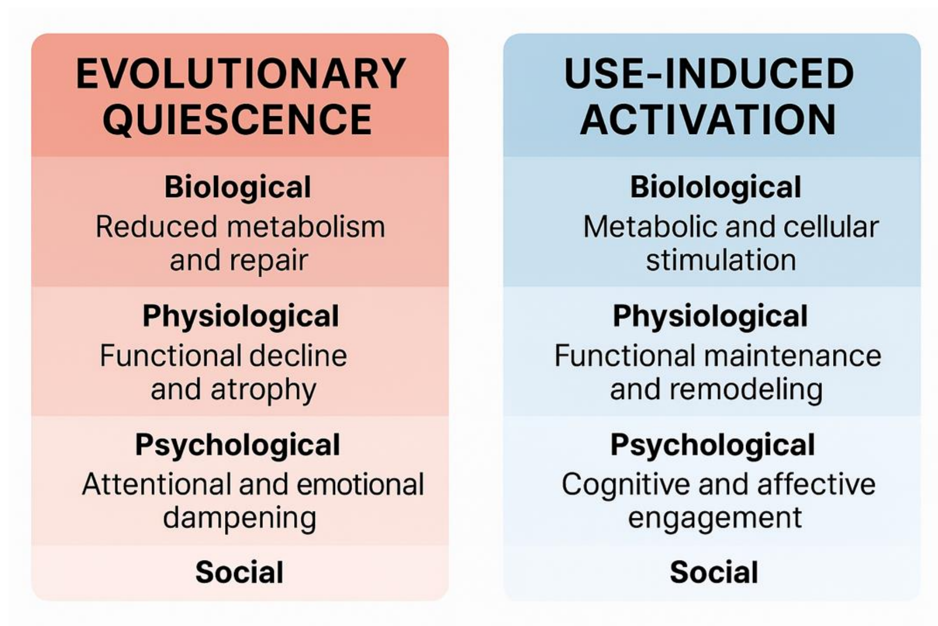

Figure 1. Evolutionary Silence and Use-Dependent Activation

In contrast, use-dependent activation (UDA) emphasizes maintaining and reshaping functions through continuous utilization and external stimulation. Its theoretical basis lies in the principle of "use it or lose it": physical exercise promotes the

preservation of muscle and mitochondrial function; cognitive challenges and learning delay the silencing of neural networks; and social participation activates psychological and social functions. In other words, UDA provides a reversible pathway to counter evolutionary silence—not only sustaining physiological and cognitive vitality but also promoting positive aging at psychological and social levels. Taken together, the two concepts illuminate the dynamic balance of aging and offer new pathways for the prevention and intervention of dementia and BPSD (Figure 1).

3. The Impact of Evolutionary Silence on BPSD

Within the nervous system, circuits responsible for emotional regulation, complex behavioral control, and sensory–cognitive integration are highly energy-demanding but not directly essential for survival. Consequently, these circuits are more likely to "fall silent" during aging. When their activity declines, patients may present with emotional instability, impulsive behaviors, hallucinations, or depression—hallmarks of BPSD. If BPSD is understood as the manifestation of silenced functions across biological, physiological, psychological, and social domains, then the core of intervention should extend beyond symptomatic management to include delaying or reactivating silent circuits and maintaining functional availability.

3.1. Biological Mechanisms

Evolutionary silence exerts profound effects on BPSD through its low-power strategy. As aging progresses into the silent phase, cerebral energy supply is preferentially allocated to basic survival functions, while higher-order cognitive and emotional regulatory regions (e.g., prefrontal cortex and limbic system) are downregulated, resulting in decreased neural network efficiency. Simultaneously, autophagy and protein clearance systems become less effective, leading to the accumulation of abnormal proteins (Aβ, Tau) and inflammatory mediators, which further impair synaptic transmission. In addition, the metabolic balance of neurotransmitters (dopamine, serotonin, acetylcholine) is disrupted, giving rise to emotional instability, impulsivity, hallucinations, and aggressive behaviors. Key biological factors contributing to BPSD include:

(1) Energy insufficiency: Declines in cerebral glucose metabolism impair integration within the prefrontal–hippocampal circuit, triggering anxiety and delusions.

(2) Phenotype–genotype interactions:

APOE ε4: more prone to cognitive decline accompanied by hallucinations and delusions;

SNCA variants: heightened risk of anxiety and impulsive behaviors;

GBA mutations: more frequent depression and apathy;

5-HTTLPR S/S genotype: strong emotional lability and anxiety responses.

(3) Neuroinflammation: Insufficient clearance of Aβ and Tau under low-energy conditions promotes inflammation and impairs emotional regulation.

These biological factors shape the brain's capacity to respond to stress, inflammation, and metabolic challenges during evolutionary silence, thereby increasing both the likelihood and intensity of BPSD. Importantly, evolutionary silence does not directly generate BPSD; rather, through the chain of *energy insufficiency → protein aggregation → network decoupling*, it enhances susceptibility and severity. Thus, BPSD may be viewed as an "external signal" of biological silence, reminding us that use-dependent activation (e.g., exercise, cognitive training, metabolic enhancement) can reopen partially silent neural pathways and alleviate symptoms.

3.2. Physiological Mechanisms

At the physiological level, evolutionary silence primarily affects BPSD through the downregulation of autonomic and homeostatic systems. As aging advances into the silent phase, cardiac and pulmonary function, cerebral perfusion, and cerebrovascular autoregulation decline, leading to insufficient oxygen and energy supply in the brain—especially within the prefrontal and hippocampal regions. At the same time, the endocrine–immune network becomes hypo-reactive yet chronically inflamed, while hormonal rhythms (e.g., cortisol, pineal melatonin

secretion, insulin-like growth factor) become dysregulated, further disturbing the sleep–wake system and emotional stability. Sleep disorders and circadian rhythm disruption often serve as major triggers for BPSD. Dysregulation of the sympathetic–parasympathetic balance (autonomic decoupling) may also induce excessive anxiety, blood pressure fluctuations, and impulsive or aggressive behaviors. Moreover, the silencing of gastrointestinal, excretory, and metabolic functions results in energy fluctuations and physical discomfort, amplifying anxiety, irritability, hallucinations, and other behavioral symptoms.

In summary, physiological silence increases BPSD susceptibility through a decline chain involving circulation, metabolism, and neuroendocrine regulation. Appropriate use-dependent activation—such as physical exercise, regular dietary patterns, and sleep interventions—can partially restore homeostatic control and reduce both the occurrence and severity of BPSD.

3.3. Psychological Mechanisms

Evolutionary silence influences the onset and manifestation of BPSD at the psychological level by progressively reducing cognitive resources and weakening emotional regulation systems. As aging advances into the silent phase, dopaminergic neuronal activity declines, leading to reduced investment of brain resources in attention, executive functioning, and emotional control. Motivation and reward circuits become silenced, resulting in apathy, depression, and anhedonia. The decreased coupling between the prefrontal cortex and limbic system further impairs self-awareness and emotional regulation.

This low-power state allows impulses and negative emotions—previously moderated by rational control or social norms—to surface more readily, manifesting as diminished cognitive control, reduced executive function, and an inability to suppress aggression or inappropriate behaviors. Consequently, patients may exhibit anxiety, depression, irritability, or aggression as forms of BPSD. In parallel, the silencing of memory and language systems hampers information processing and communication, preventing patients from clearly expressing their needs or discomfort. This often provokes heightened agitation and resistance. Psychological silence also lowers tolerance thresholds for environmental stimuli, meaning even minor changes can trigger disproportionate reactions.

In other words, evolutionary silence at the psychological level reduces emotional tolerance and cognitive regulation, making BPSD a visible manifestation of a low-power psychological system. Through use-dependent activation (e.g., emotion regulation training, cognitive stimulation, social support), psychological resilience can be partially restored, thereby alleviating BPSD.

3.4. Social Mechanisms

Evolutionary silence also exacerbates BPSD through the decline of social roles, interaction networks, and support systems. As aging enters the silent phase, individuals' roles within the division of labor weaken, while their economic and family functions diminish, leading to passivity, helplessness, and a reduced sense of purpose. This "deactivation" of social roles is often accompanied by loneliness and social isolation, which serve as important triggers for BPSD.

Meanwhile, decreased frequency of social interaction deprives the cognitive and emotional systems of use-dependent activation, reinforcing the low-power state. When patients lose autonomy or choice in daily life within care settings, they often express resistance, stubbornness, aggression, or refusal of care as behavioral symptoms of BPSD. Moreover, insufficient social support or caregiver stress can amplify BPSD through negative interaction feedback loops.

In short, evolutionary silence at the social level functions as an "external driver" of BPSD, with behavioral problems becoming compensatory expressions of loss and social disconnection. Socially oriented use-dependent activation—such as social activities, group participation, and empowerment-based caregiving—not only improves emotional and behavioral outcomes but also buffers the adverse impact of social silence on BPSD.

4. Strategies for Improving BPSD through Use-Dependent Activation

Behavioral and Psychological Symptoms of Dementia (BPSD) severely compromise patients' quality of life and impose substantial caregiving burdens. Traditional interventions have largely focused on pharmacological treatment and environmental modifications, but their effects remain limited. Professor Takahito Takeuchi, a Japanese scholar of care science, proposed the theory of *independence-supportive care*, an approach that contrasts with the conventional "disease–drug

model." This model emphasizes hydration management, physical activity/exercise, oral feeding, elimination support, and social engagement as core components to help individuals with dementia maintain daily functional capacity and social participation as much as possible. By employing *use-dependent activation (UDA)* to delay functional decline, this approach has demonstrated significant effectiveness in alleviating BPSD (Figure 2).

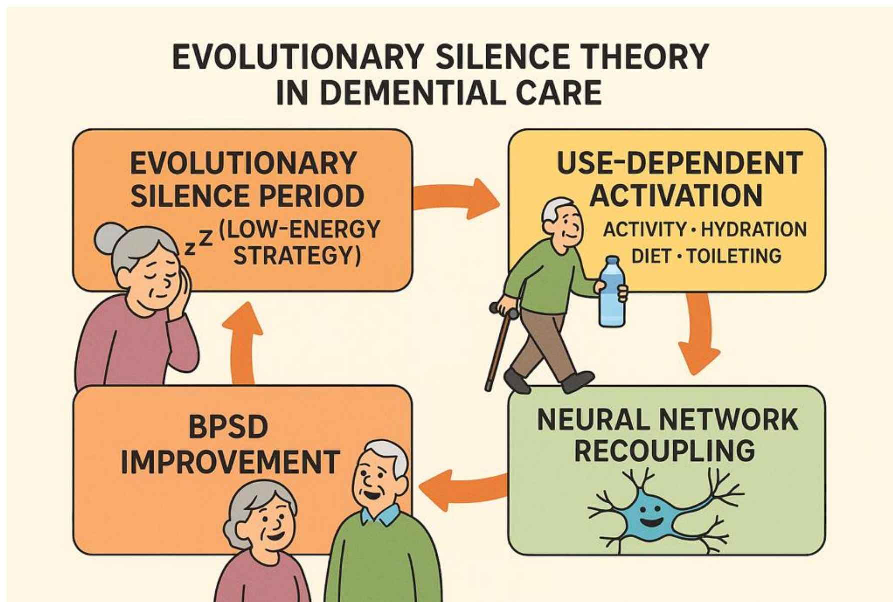

Figure 2. Independence-Supportive Care and Use-Dependent Activation in BPSD

4.1. Hydration Management

During the aging-related phase of evolutionary silence, diminished thirst perception and inadequate fluid intake often result in chronic dehydration. Even mild dehydration can lead to electrolyte imbalance, elevated plasma osmolality, and reduced cerebral blood flow, thereby impairing energy supply to the prefrontal cortex and hippocampus. This exacerbates memory impairment, attentional deficits, and emotional instability, which may manifest as irritability, agitation, and hallucinations—core features of BPSD.

Systematic hydration management (e.g., scheduled and supervised drinking with specified amounts) improves cerebral hemodynamics and cellular metabolic efficiency, stabilizing neuronal synaptic activity. More importantly, drinking behavior itself, as a daily-life form of UDA, reinforces self-management and interaction with the environment, enhancing autonomy and cognitive awareness while reducing anxiety and aggression associated with loss of control. Thus, hydration management contributes to alleviating BPSD through two pathways: maintaining physiological homeostasis and reactivating brain function. By combining water balance maintenance with empowerment through behavioral activation, hydration management effectively buffers the negative impact of evolutionary silence at both physiological and psychological levels, thereby improving BPSD.

4.2. Physical Activity and Exercise

Physical activity and exercise can ameliorate BPSD across physiological, neurological, and psychological dimensions. Physiologically, regular exercise enhances cardiopulmonary function and circulation, increases cerebral blood flow and oxygen supply, and thereby mitigates the energy insufficiency characteristic of the low-power state in evolutionary silence. Neurologically, exercise stimulates brain-derived neurotrophic factor (BDNF) and dopamine release, promotes synaptic plasticity and neural circuit reconstruction—particularly in the prefrontal–hippocampal and limbic networks—and reduces anxiety, depression, and impulsive behaviors associated with BPSD. Furthermore, exercise decreases neuroinflammation and oxidative stress, exerting some inhibitory effects on abnormal Aβ and Tau deposition.

At the psychological and social levels, physical activity provides patients with structured goals and daily rhythms, reduces boredom and confusion, enhances self-efficacy and a sense of accomplishment, and fosters social interaction through group exercise, thereby mitigating loneliness and isolation. As a multidimensional form of UDA, activity and exercise not only restore neural functional activity but also strengthen psychological and social connections, leading to significant improvements in BPSD.

4.3. Oral Feeding

Oral feeding is a fundamental physiological function of human beings. Beyond serving as a process of nutritional supplementation, it represents a multidimensional form of functional activation and has unique mechanisms for improving BPSD. Physiologically, chewing and swallowing promote increased cerebral blood flow, stimulating the trigeminal and glossopharyngeal nerve pathways and activating the limbic system and prefrontal cortex, thereby enhancing attention and emotional regulation. Neurologically, masticatory activity facilitates the release of dopamine and acetylcholine, strengthens hippocampal synaptic plasticity, slows cognitive decline, and reduces BPSD such as anxiety, aggression, and hallucinations that arise from neural network low-power states. Psychologically, eating is among the most familiar and ritualized daily behaviors, providing patients with a sense of safety and satisfaction that alleviates unease and resistance. Socially, communal dining or assisted feeding enhances social interaction, mitigating loneliness and opposition to care. Thus, oral feeding improves BPSD through four integrated pathways—nutritional supplementation, neural circuit activation, emotional soothing, and social bonding—breaking through the functional hypoactivity of evolutionary silence.

4.4. Elimination Support

Elimination is a normal physiological function and fundamental human need. Providing elimination support is significant for improving BPSD across physiological, psychological, and social dimensions. Physiologically, evolutionary silence during aging often weakens autonomic and sphincter control. When elimination is impaired, conditions such as constipation, urinary retention, or incontinence cause physical discomfort and internal imbalance, aggravating agitation, hallucinations, or aggression associated with BPSD. Structured elimination support (e.g., scheduled toileting, hydration combined with fiber intake) maintains metabolic homeostasis and visceral function, reducing physiological stress. At the neurological and psychological levels, elimination is closely tied to autonomy and dignity; when compromised over the long term, it can provoke shame, anxiety, and depression, worsening BPSD. Appropriate support and training can restore patients' sense of bodily control, reducing distress and resistance due to loss of agency. Socially, effective elimination care minimizes embarrassment and the risk of social withdrawal, enabling greater participation in group activities, reducing isolation, and lowering conflict in caregiving

interactions. Elimination support thus improves BPSD through three UDA pathways—maintaining metabolic homeostasis, restoring bodily control, and enhancing social participation—effectively buffering the low-power and decoupling effects of evolutionary silence.

4.5. Strengthening Social Engagement

Enhancing social engagement plays a central role in alleviating BPSD. At the neurological and cognitive level, social interaction requires the integration of language, memory, emotional interpretation, and executive function—essentially functioning as a "whole-brain activation training" involving the prefrontal–hippocampal–limbic networks. This process promotes the secretion of dopamine, oxytocin, and serotonin, counteracting the low-power state induced by evolutionary silence and boosting emotional stability and cognitive vitality. Psychologically, social activities provide a sense of belonging and purpose, reducing loneliness, anxiety, and depression, and thereby decreasing the likelihood of aggression, wandering, or hallucinations. Socially, participation in roles and interpersonal interactions helps patients preserve their "social self," maintaining dignity and agency—critical for preventing behavioral dysregulation and resistance to care. Moreover, social activities are often embedded in daily contexts—such as communal meals, games, or singing—that simultaneously provide cognitive stimulation and emotional comfort. Strengthening social engagement thus improves BPSD through four pathways—network activation, emotional regulation, self-identity, and belonging—breaking through the low-power operational mode of evolutionary silence and achieving effective symptom relief.

5. Theoretical and Practical Value

Care for BPSD grounded in the theories of evolutionary silence and use-dependent activation (UDA) provides new scientific foundations and practical pathways for dementia care. Traditional approaches have largely emphasized pharmacological treatment or passive management. However, the theory of evolutionary silence reveals that during aging, neurological, psychological, and social systems progressively enter a low-power "silent" state, which is a major driver of BPSD. Sole reliance on medication may disrupt fragile metabolic and neural homeostasis. By contrast, UDA emphasizes the sustained use and stimulation of daily-life

domains—hydration, physical activity, nutrition, elimination, and social engagement—within the framework of independence-supportive care. This approach enables physiological and psychological functions in silent states to regain energy supply and network activation, thereby reducing symptoms such as anxiety, aggression, and hallucinations. Such a model not only lowers reliance on drugs and the risks of side effects but also enhances patients' autonomy and quality of life.

On April 1, 2019, Takaya Saito, director of the Morikaze Uehara Care Center in Japan, presented successful cases of independence-supportive dementia care at the "2019 China International Forum on Elderly Care Industry Development." By integrating hydration, elimination, nutrition, and physical activity management, the facility improved cognitive status among older adults with dementia. From 2012 to 2017, follow-up analysis of several hundred cases showed that after appropriate interventions, BPSD had essentially disappeared in 81% of residents, with 67.3% achieving complete remission and 13.7% near-complete remission. Health economic comparisons between independence-supportive care and the traditional disease–drug model demonstrated significant advantages across core objectives, intervention methods, and value orientation.

Looking ahead, dementia care models informed by these theories may evolve into standardized intervention protocols and individualized activation plans. Integrated with AI-based monitoring and wearable devices, such models could enable dynamic tracking and intervention across the continuum of "low power–activation–recoupling." The prospect is to build a more precise, low-risk, and sustainable dementia care system for aging societies, shifting caregiving from passive management toward active improvement and the reconstruction of daily living.

For Further Readings:

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