

With a View to Fear Extinction and to Brain Functional Change: A Neurotheoretical Hypothesis About Traditional Health-Preserving Exercises as Treatments for Post-Aortic Dissection Kinesiophobia

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Abstract: Post-aortic dissection kinesiophobia is one of the most frequent clinical problems—greatly impairing rehabilitation adherence—yet its neural underpinnings remain largely unexplained. Taking recent progress in studies of fear-related neural circuits (learning and extinction) as a basis, the present paper suggests that post-aortic dissection kinesiophobia corresponds to a conditioned fear phenomenon essentially of the type described with insufficient vmPFC inhibition and with an overactive amygdala, the present discussion proposes the “fear extinction-brain functional remodeling” neurotheoretical hypothesis: traditional health-preserving exercises—by virtue of their three-dimensional synergy (of body regulation, breath control, and mental calm)—help to regulate top-down vmPFC-amygdala activity, to optimize DMN functional connectivity and internetwork dynamics, to acquire and solidify fear extinction learning, and so to relieve kinesiophobia and to improve long-term prognosis. It now gives a systematic account of indirect evidence for this hypothesis and outlines multimodal fMRI studies as well as a mediation analysis research agenda.

Keywords: Aortic dissection; Kinesiophobia; Extinction of fear; Standard health-maintaining exercises; Remodeling of brain function

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1. Introduction

1.1. Kinesiophobia: The invisible barrier to post-aortic dissection rehabilitation

Aortic dissection (AD) is a cardiovascular emergency with a sudden onset and very high mortality. With modern surgical methods and better perioperative care, survival following post-AD events has improved

greatly, yet rehabilitation—after surgery—is hampered by an often-neglected problem called kinesiophobia^[1]. The previous surgery-related studies gave an estimate of 46.2% for the incidence of kinesiophobia^[2]. A later 2026 study added further evidence of the heterogeneity of post-AD kinesiophobia: of the 309 examined cases, 73.14% ($n=226$) had kinesiophobia. They could be assigned to three subtypes—Type A (25.2%), Type B (17.7%), or Type C (57.1%). Age above 65 years, Stanford Type A dissection, lack of a spouse, low education level, and highly invasive surgery seem to be connected with higher fear levels^[3]. Kinesiophobia has direct negative effects on participation in rehabilitation exercises and may itself feed back in an unending cycle into poor postoperative functioning, bad mental health, or reduced quality of life. Current research on post-AD kinesiophobia tends mainly to treat it as a psychobehavioral phenomenon^[4]. But, in terms of neuroscience, the clinical manifestations of kinesiophobia—specific hypervigilance toward exercise-related cues, long-standing avoidance acts, and fear that is hard to remove even with medical reassurance—seem to closely match the behavioral traits of conditioned fear responses. Such observations encourage the authors to consider the neural basis of fear learning and of extinction as central to an explanation of post-AD kinesiophobia.

1.2. Advances in research on the neural circuits of fear extinction

Within the past two decades, work on the neural basis of fear learning and of its extinction has achieved major advances^[5–6]. According to the classical model, the amygdala, hippocampus, and medial prefrontal cortex are central to fear processing, with many mutual connections—both forward and backward—among them^[7–8]. With regard to conditioning of fear, the basolateral amygdala (BLA) is the primary location for formation and for manifestation of such memories; the hippocampus helps to supply contextual details which regulate fear responses; and the ventromedial prefrontal cortex (vmPFC) is centrally involved in fear extinction^[9–10]. The essential neural basis of extinction learning is top-down suppression of the amygdala by the vmPFC^[11]. Functional magnetic resonance imaging (fMRI) data repeatedly confirm that retrieval of an extinction memory—when it succeeds—is associated with enhanced functional connectivity between the vmPFC and amygdala^[12]. An important study has shown that those people whose vmPFC-amygdala functional connectivity was stronger following a fear memory recall had more effective fear suppression in the extinction trials that followed^[13]. Further mechanistic work has now clarified the role of theta oscillations—of information flow between the vmPFC and amygdala—as an electrophysiological basis for extinction learning—theta transmission from the vmPFC to the amygdala helps to acquire a sense of safety, whereas retention of threat data in the amygdala is slowly lost during extinction^[14].

1.3. Traditional health—Some preserved exercises as mind-body interventions that may affect the nervous system

Among traditional forms of health-preserving exercise (such as Tai Chi, Baduanjin, Qigong, etc.), there is a three-dimensional synergy called “body regulation, breath regulation, and mind regulation.” Over the past few years, some neuroimaging investigations have begun to clarify their special brain regulatory effects^[15]. For people who regularly do Tai Chi, there are definite changes—of low-frequency type—in the default mode network (DMN) and frontoparietal network^[16]. Both Tai Chi and Baduanjin are capable of influencing functional connectivity in the cognitive control network, and Tai Chi practitioners display marked changes—relative to controls—in resting-state functional coupling between the dorsolateral prefrontal cortex and

anterior cingulate cortex ^[17–18]. As far as emotion regulation is concerned, systematic reviews give evidence that Tai Chi reduces amygdala activation, helps to normalize prefrontal-limbic relations, and improves autonomic tone ^[17, 19]. Such observations seem to confirm that traditional forms of health-promoting exercise may affect several nodes of the fear extinction system by means of multi-target neuromodulation, thereby offering a non-drug-based, widely applicable approach to treatment of post-AD kinesiphobia.

1.4. Purpose of this paper

The present paper proposes an integration of findings about the neural circuits of fear extinction, of clinical cases involving post-AD kinesiphobia, and of prior neuroimaging studies of ordinary health-promoting exercises—all of which seem relevant—to formulate the “fear extinction–brain functional remodeling” neurotheoretical hypothesis, and to lay out both theory and program for later systematic testing.

2. Neural mechanisms of fear learning and extinction

2.1. Fear conditioning: Synergy of the amygdala, hippocampus, and prefrontal cortex

Fear conditioning is an associative learning phenomenon that is conserved across species ^[7]. With regard to the classical fear conditioning model, if a neutral stimulus (conditioned stimulus, CS) is paired many times with something unpleasant (unconditioned stimulus, US), then the CS alone may lead to a fear reaction (conditioned response, CR). Such a behavioral outcome depends upon synaptic plasticity of the basolateral amygdala; long-term potentiation (LTP) and NMDA receptor-related protein synthesis are involved. The hippocampus is responsible for contextual modulation of fear conditioning. With regard to contextual fear conditioning, the hippocampus must encode environmental cues, and its projections to the vmPFC help to regulate fear expression through such context-dependent information. The prefrontal cortex—specifically the vmPFC—defines a cap on fear intensity during the conditioning period by limiting amygdala activation, so as not to exaggerate threat-related responses. Dynamic interaction of these three areas is central to the neural basis of fear learning.

2.2. Fear extinction: Top-down inhibition of the amygdala by the vmPFC

Fear extinction should not be regarded as forgetting fear memory but as building a new, competitive safety memory. The central neural mechanism of such a process involves top-down inhibition of the amygdala by the vmPFC. With regard to its glutamatergic pathway leading to amygdala intercalated cells (ITCs), the vmPFC triggers activity in ITCs and blocks fear-related neurons in the BLA, thus suppressing fear responses. On the network level, extinction learning may be understood as involving dynamic reorganization of large-scale brain networks. An fMRI investigation has shown that for extinction learning, functional connectivity in the DMN, in the frontoparietal area, or in the ventral attention system rises appreciably between early and late stages ^[20–21]. This observation is of major theoretical value: it denies that extinction learning is just a matter of local circuit plasticity but shows global network-level reorganization.

2.3. Fear memory reinstatement and extinction retention: The role of the DMN

The fragility of extinction memory presents a basic difficulty for exposure therapy—it tends to be disrupted by fear reinstatement or by spontaneous recovery, renewal ^[22–23]. Now some recent studies are beginning to consider the role of the DMN in retention and in retrieval of extinction memory ^[24]. Prefrontal glucocorticoid

receptors help to regulate the relationship of the salience network with DMN during fear memory formation. A number of studies have shown that in the transition of fear memory—from recent (hippocampus-dependent) to remote (cortex-dependent)—there is a reorganization of the retrieval network going from a “salience network-like” configuration to a “DMN-like” configuration^[25–26]. This seems to indicate that the DMN is central—perhaps essential—to remote fear memory storage and recall. Among the core nodes of the DMN—posterior cingulate/precuneus, medial prefrontal cortex, and hippocampus—each has strong structural or functional links with the vmPFC–amygdala circuit; dysfunction of the DMN may cause both recurrence of fear memory and failure of extinction consolidation.

2.4. The role of the autonomic nervous system and endocrine system in fear responses

Peripheral expression of fear responses is jointly controlled by the autonomic nervous system and the neuroendocrine system^[27]. Activation of the sympathetic nervous system brings about higher heart rate, increased blood pressure, and greater skin conductance; hypothalamic-pituitary-adrenal (HPA) axis activation is associated with cortisol release. With regard to extinction learning, a high vagal tone seems closely bound to the consolidation of extinction memory^[28]. Vagus nerve stimulation (VNS) may help to achieve extinction learning in the BLA through an increase in locus coeruleus noradrenergic activity, with rapid consolidation of extinction memory. Transcutaneous auricular vagus nerve stimulation (tVNS) is now known to facilitate long-term recall of extinction memory in human subjects. Such evidence is helpful in figuring out the neuromodulatory role of the breath-related part of traditional health-preserving exercises.

3. Neuropathological hypothesis of post—Aortic dissection kinesiophobia

3.1. Traumatic events as well as surgery: Robust consolidation of fear memory

The beginning of AD is marked by highly traumatic features. Sudden intense chest pain, an intrusive episode of emergency surgery, and persistent stress following the postoperative ICU phase all fit a “multiple US” fear conditioning context. Within such contexts, it is possible for exercise-linked interoceptive signals (elevated heart rate or more profound breathing) to act as conditioned stimuli and to establish solid connections with fear responses with regard to interoceptive conditioning mechanisms. From a neurobiological standpoint, the huge surge of stress hormones (norepinephrine, cortisol) at AD onset seems to offer neurochemical help for the over-consolidation of fear memory. Animal evidence suggests that stress hormones may facilitate synaptic plasticity in the amygdala, thus helping fear memory to be more tenacious^[29–30]. Post-AD individuals suffer from a fear memory that has already been “super-consolidated,” which is one reason why giving them medical reassurance alone rarely eliminates kinesiophobia.

3.2. Misinterpretation of “avoid exercise” medical advice: Reinforcement of fear conditioning

Post-AD patients are generally told to observe strict limits on exercise, namely no excessive physical effort, no hard training, and no Valsalva maneuvers. Such instructions are medically justified, yet if one lacks both communication and a slow introduction to exercise, the patient may end up thinking that “exercise=danger.” Behaviorally speaking, this cognitive generalization produces an avoidance pattern that applies broadly to various forms of exercise, and such avoidance sustains or worsens fear thanks to negative reinforcement—each instance of avoidance relieves anxiety briefly but denies the possibility of extinction learning. More

importantly, the early postoperative recurrence of avoidance behavior may help to solidify the initial fear memory by means of the “reconsolidation” process. Every time a patient resorts to avoidance in thinking about exercise, the old fear memory comes back and is modified, which complicates its eventual extinction.

3.3. Hypothesis of insufficient vmPFC inhibition and amygdala hyperactivation

With regard to studies of the fear extinction neural circuit, it is now possible to suggest that the basic neuropathological element of post-AD kinesiophobia may be a failure of top-down inhibition of the amygdala by the vmPFC, combined with amygdala hyperactivity and reactivity. Several indirect pieces of evidence back up this hypothesis. Indeed, diminished vmPFC–amygdala coupling (in terms of function) and increased amygdala reactivity seem to be commonly noted phenomena in patients with anxiety disorders. High anxiety together with kinesiophobia in post-AD cases seems to reflect similar neural circuit anomalies. Second, post-AD patients suffer from disturbances of cerebral blood flow perfusion. A study using arterial spin labeling reports reduced cerebral blood flow in a number of areas of post-AD brains—namely, key network sites for thought, attention, or emotional control^[31–32]. Should the vmPFC’s perfusion fail to reach normal levels, its top-down inhibition may not operate fully, and fear extinction could become less likely.

3.4. DMN dysfunction and repeated retrieval of fear memory

Among the prominent clinical features of post-AD kinesiophobia is the “stubbornness” of fear—even though rational arguments confirm the safety of physical exercise, fear responses persist. Such persistence may be linked to a frequent return of fear memory and to trouble with extinction memory brought about by DMN dysfunction. The DMN is centrally involved in self-directed thinking and in retrieval of past events. With abnormal DMN function, self-referential memories of fear about exercise seem to come up too often, generating intrusive ideas such as “I am unsafe” or “exercise will cause dissection recurrence.” At the same time, context-safety associations newly built by extinction learning run a real risk of not being well integrated with self-referential memory via the DMN. Furthermore, post-AD patients may show an impaired dynamic balance between the DMN and frontoparietal system, which complicates their ability to let go of fear thoughts (self-related) and to begin safe, task-oriented exercise experiences.

3.5. Autonomic imbalance and enhanced cardiovascular stress responses

Post-AD patients commonly show signs of autonomic nervous system dysfunction. Heart rate variability data make it clear that low-frequency power, high-frequency power, and LF/HF ratio are very poorly developed in post-AD people—a sign of cardiac sympathetic and parasympathetic denervation^[33–34]. The present autonomic imbalance is harmful to cardiovascular health and may encourage fear by means of interoceptive interaction. With regard to interoceptive predictive coding, the cardiovascular system of post-AD patients—after surgery and with denervation—generates abnormal “interoceptive noise.” The brain may misread such interoceptive irregularities as “danger signals,” and then begin to feel fear in consequence. On the other hand, heart rate increases, or blood pressure rises brought about by exercise (normal phenomena for healthy people) may now be wrongly associated with “harbingers of dissection recurrence” in post-AD subjects, thus strengthening the link between exercise and fear. Such a vicious cycle—of autonomic disturbance and heightened fear—constitutes a significant peripheral factor in the maintenance of post-AD kinesiophobia.

4. Traditional health-preserving exercises and their neuromodulatory parts

4.1. Body regulation: Use of a safe exercise experience to reverse fear conditioning

Among traditional health-preserving exercises, one finds low-intensity, slow, fluid movements, with an unmistakable “safe exercise experience” for post-AD patients^[35]. With regard to fear extinction, body regulation is basically an instance of interoceptive exposure^[36]. For those who do Tai Chi or Baduanjin, the physical effects of the exercise they experience (mild acceleration of the heart, more vigorous breathing) occur in a safe, controlled situation with no aversive outcome. Such a process matches well the basic logic of extinction learning: with the CS (interoceptive signals from exercise) still active, the US (recurrence of dissection/danger) is absent, hence creating a new CS-safety link^[37]. More importantly, the movement design of traditional health-preserving exercises makes it possible to avoid Valsalva maneuvers and abrupt changes in blood pressure, thus helping the patient to keep hemodynamics more or less stable during exercise. Such safety is not merely medical but also neurobehaviorally helpful: extinction training—i.e., the key to extinction learning—is facilitated by the certainty of US absence during CS exposure, and traditional health-preserving exercises yield exactly this “certainty of safe experience.”

4.2. Breath regulation: Vagal activation and autonomic balance

Breath regulation is a central element of traditional health-preserving exercises, and it calls for slow, deep breathing from the abdomen. Such breathing is known to lead to substantial improvement of vagal tone as well as to an increased high-frequency part of heart rate variability. With regard to fear extinction, the regulation of respiration—probably via the vagus—may help to establish or consolidate extinction memory in a direct fashion. Stimulation of the vagus nerve seems to help consolidate fear extinction—by means of increased locus coeruleus noradrenergic transmission and LTP in the BLA—a fact now well established in animal studies^[38–39]. As far as humans are concerned, transcutaneous auricular vagus nerve stimulation is known to facilitate both immediate suppression of fear and long-term recall of extinction memory. Among the components of traditional health-preserving exercises, breath regulation may foster a neuromodulatory climate favorable to extinction learning—through similar vagal activation mechanisms—with the cooperative release of norepinephrine and acetylcholine helping to improve synaptic flexibility in the vmPFC and amygdala, and thus to encode or consolidate extinction memory^[40]. In addition, the autonomic regulatory effects of slow breathing seem to be capable of correcting post-AD autonomic imbalance^[41]. With higher parasympathetic tone and less sympathetic overactivity, the breath regulation component is likely to ease cardiovascular strain during exercise and to prevent fear responses caused by interoceptive noise.

4.3. Mind regulation: Promotion of fear extinction learning through mindful attention

The mind regulation part is concerned with keeping attention directed toward immediate bodily feelings and toward one’s own breathing, without prejudice or evasion. Such mindful attention seems to help in many ways with the process of fear extinction learning. First of all, it fosters an improved awareness of actual sensory facts, which may lead to clearer identification of both “threat-related signals” and safety signals. Extinction learning is basically a form of discriminative learning—learners must come to distinguish between CS+ (threat cues joined with US) and CS– (safety cues). Training in mindfulness—which increases interoceptive accuracy—seems helpful in achieving such discrimination. Second, mindful attention brings about less dependence upon cognitive avoidance or emotion control with regard to fear memory. fMRI work has indicated that mindfulness training facilitates extinction memory retrieval, by means of direct modulation

of subcortical threat-processing areas (like the amygdala) instead of depending on top-down cognitive control ^[42]. This “direct modulation” mode coincides very closely with the mind-regulation traits of known health-promoting exercises—mindful attention is not obliged to push patients toward an active “suppress” of fear but helps extinction through modification of the relationship with fear experiences. Third, mindfulness training seems able to improve hippocampus-mediated contextual retrieval and thus allow flexible extinction memory across contexts ^[43]. Such flexibility is especially significant for post-AD kinesiophobia since people with this disorder tend to have generalized fears of exercise, which call for an extinction memory not limited to one context.

4.4. Three-dimensional synergy: Advantages of multi-target neuromodulation

Among the advantages of traditional health-preserving exercises is the three-dimensional synergy of body regulation, breath regulation, and mind regulation. Compared with either single-exposure treatment or with mindfulness alone, such synergy offers a more favorable setup for extinction learning. Body regulation furnishes a secure environment for interoceptive exposure; breath regulation gives rise to a neuromodulatory environment that encourages extinction consolidation and benefits the mind in its regulation—both of states and of contexts—during extinction learning. The two factors taken together seem to yield a more potent effect than either alone. From the standpoint of network neuroscience, three-dimensional synergy implies joint control of many brain systems: body regulation is largely connected with the sensor. The motor and interoceptive networks; regulation of breath is essentially handled by autonomic regulatory pathways (i.e., by the brainstem and insula); mind regulation is essentially linked to the DMN and to the executive network. Modulation of several networks together seems likely to help with consolidation of extinction memory for long-term recall of extinction memory; one needs widespread synaptic plasticity across many networks.

5. Proposal of the neurotheoretical hypothesis

5.1. Hypothesis 1: Conventional health—Saving exercises may help to improve vmPFC-amygdala connectivity

Consistent practice of traditional health-preserving exercises seems to help in post-AD cases with regard to resting-state or task-state functional connectivity of the vmPFC and amygdala. Such an improvement is likely to result from two interwoven mechanisms. First, the interoceptive exposure of the body regulation part offers many repeated “practice” occasions for vmPFC inhibition of the amygdala; the strengthening of the vmPFC–amygdala pathway is similar to what occurs with extinction training. Second, the combination of breath regulation and mind regulation brings about favorable neuromodulatory and hemodynamic states—specifically higher vagal tone and prefrontal activity—for the vmPFC. From a clinical standpoint, the authors expect that an 8-to 12-week course of traditional health-promoting exercise may lead to substantial improvement in vmPFC–amygdala connectivity for post-AD patients, with such improvement linked closely to falling kinesiophobia scores.

5.2. Hypothesis 2: Conventional health-preserving exercises may help to optimize DMN internal and internetwork connectivity

Standard health-preserving exercises are likely to bring about an improvement in functional connectivity of the DMN and to promote a better dynamic balance—between the DMN and the frontoparietal network

and the salience network. More specifically, the authors anticipate that functional connectivity of the DMN (notably the vmPFC–posterior cingulate or precuneus circuit) is going to increase, with extinction memory being more efficiently integrated, now being considered in connection with self-referential knowledge structures. At the same time, the anticorrelation between the DMN and frontoparietal network seems possibly stronger now, which would help explain both a reduction of self-bound fear and an increase in task-oriented safety (exercise) feelings. With regard to low-frequency amplitude of the DMN and frontoparietal network, Tai Chi users show marked changes—an indirect initial support for this hypothesis.

5.3. Hypothesis 3: Brain functional remodeling mediates the decline in kinesiophobia

Increased vmPFC–amygdala functional connectivity as well as optimized DMN function seem to mediate the relation between conventional health-preserving exercise interventions and reduced kinesiophobia. More specifically, the intervention lowers fear responses by means of top-down suppression of the amygdala—by the vmPFC—and by less frequent recall of fear memories (due to better DMN function), both of which help explain the behavioral relief from kinesiophobia. Such an idea is amenable to mediation analysis: the mediating factor is change in brain connectivity, the independent factor is traditional health-preserving exercise, and TSK-SV Heart score changes are the dependent variables—all to be used in testing the indirect effect’s significance.

5.4. Hypothesis 4: Brain functional remodeling further mediates long-term prognosis improvement

Functional remodeling of the brain is not merely responsible for an initial reduction of kinesiophobia but may also help to improve long-term prognosis. It seems likely that the amount of improvement—whether in vmPFC–amygdala connectivity or in DMN function—after traditional health-preserving exercise treatment will predict how well one adheres to exercise rehabilitation, how much physical function is restored, and what quality of life results at 6–12 months post-intervention. Such prediction needs validation, preferably by means of a longitudinal study showing time-ordered causality between brain changes and long-term medical results.

5.5. Overall framework of the hypothesis

Among the four hypotheses mentioned, there is a causal chain—from neural mechanisms up to clinical results: three-dimensional synergistic use of traditional health-preserving exercises seems to improve vmPFC–amygdala interaction and DMN optimization; it helps with extinction of fear; it reduces kinesiophobia; it promotes long-term recovery. Each of the links in this causal chain is amenable to individual testing, yet the general hypothesis offers a well-ordered plan for scientific investigation.

6. Indirect evidence as well as theoretical justification for the hypothesis

6.1. On the question of Tai Chi’s ability to improve prefrontal connectivity

Among traditional health-preserving practices, Tai Chi is a clear example having considerable imaging evidence concerning the regulatory effects it may have on prefrontal connectivity. A randomized brain imaging study reports that a remote Tai Chi treatment brings about significant increases in dorsolateral prefrontal functional connectivity for those with knee osteoarthritis^[44]. With regard to fibromyalgia patients,

12 weeks of Tai Chi practice—without concurrent drug therapy—produces substantial improvement in resting-state functional connectivity of the cognitive control network (relative to) the anterior cingulate/medial prefrontal cortex ^[45]. Even if the cited studies were not done in post-AD groups, their findings on the prefrontal–limbic regulation by Tai Chi still help confirm Hypothesis 1.

6.2. Promoting effects of mindfulness meditation on fear extinction

Mindfulness meditation is the basic technique used in the mind-regulation part of traditional health-preserving exercises. A randomized controlled trial—using 7T fMRI—has shown that mindfulness training helps with extinction-related memory retrieval and with the suppression of both behavioral and neural threat responses following extinction ^[42]. It is important to note that the effect of mindfulness training was not obtained by increased activation of the cognitive control areas; rather, it is manifested as a direct decrease—rather than an increase—in subcortical threat-related activation (especially in the amygdala). The “direct modulation” mode implies that mindfulness may encourage extinction by means of neural effects not found in ordinary exposure therapy, which seems to match the special way in which mental regulation operates within traditional health-preserving exercises.

6.3. Enhancing effects of regular exercise on fear extinction learning

Exercise of the regular type is itself helpful for fear extinction learning. Studies with animals reveal that wheel running—given once—after extinction training may help retrieve extinction memories, and such an effect relies on the central mTOR signaling pathway ^[46]. With regard to humans, moderate-intensity aerobic exercise carried out either during or following extinction learning seems to aid consolidation of extinction memory. Traditional health-preserving exercises (which are low-to-moderate-intensity mind-body activities) seem to foster extinction memory consolidation by means of similar mTOR-related processes ^[47]. At the same time, these traditional forms of exercise add—beyond aerobic activity—control of breathing and control of mental state, which may yield extinction-enhancing results not achieved otherwise ^[48].

6.4. Clinical observations of post-aortic dissection kinesiophobia

Clinical features of post-AD kinesiophobia give an indirect behavioral basis for this hypothesis. The high frequency, marked diversity, and inverse relationship between post-AD kinesiophobia and exercise self-efficacy all seem to agree with the behavioral pattern of conditioned fear responses ^[49]. Even more important is the finding that moderate-intensity home-based exercise is not harmful to cardiovascular patients and may “help patients rebuild trust” ^[50]. The present finding accords well with the behavioral logic of fear extinction—safe forms of exercise seem to perform the role of extinction learning. Yet the trial under discussion says nothing about neural processes or about systematic use of extinction learning-oriented treatment protocols. Standard health-promoting exercises—three-dimensionally—seem to encourage extinction learning by virtue of that fact.

7. Validation strategy and research agenda

7.1. Multimodal fMRI validation pathway

Testing this hypothesis calls for the use of multimodal neuroimaging techniques. Resting-state fMRI (rs-fMRI) is well suited for studying shifts—before and after treatment—of vmPFC–amygdala functional connectivity

and DMN internal as well as internetwork connectivity. Task-state fMRI lends itself to paradigms involving fear conditioning or to exercise-related interoceptive exposure in order to analyze activation patterns and dynamics of functional connectivity of the vmPFC and amygdala with respect to extinction learning. Diffusion tensor imaging (DTI) offers a means of determining the anatomical quality of the vmPFC–amygdala white matter pathway, and of evaluating—on an individual basis—possible links between microstructural change and functional connectivity. A longitudinal plan is advised: baseline study, 8–12 weeks of standard health-promoting exercise intervention, post-treatment evaluation, and a 6-month follow-up, to trace the time course of such brain effects and of extinction retention phenomena.

7.2. Correlation analysis between TSK-SV heart and brain functional connectivity

Assessment of kinesiophobia should use the Chinese version of the TSK-SV Heart scale, which has already been tested for reliability and validity with regard to Chinese cardiac patients, and which gives a Cronbach's α value of 0.790, having four dimensions—danger perception, kinesiophobia, exercise avoidance, and functional impairment. For correlation analysis, it is recommended to apply seed-based whole-brain functional connectivity and network-based statistics to check the relations between TSK-SV Heart total scores (or dimension scores) and vmPFC–amygdala connectivity, DMN internal connectivity, and DMN–frontoparietal network connectivity. The main hypothesis is: the improvement post-intervention of vmPFC–amygdala functional connectivity correlates positively with the drop in kinesiophobia magnitude.

7.3. Theoretical design of mediation analysis and structural equation modeling

To test the hypotheses labeled “brain functional remodeling mediates the decline in kinesiophobia” and “brain functional remodeling further mediates long-term prognosis improvement,” one uses mediation analysis. The theoretical model to be considered has this form: the independent variable is the treatment group (traditional health-preserving exercises or standard rehabilitation); the mediating variable is brain functional connectivity changes (vmPFC–amygdala and DMN-associated indicators) considered here; dependent variables include kinesiophobia changes (TSK-SV Heart) and long-term prognosis factors (exercise adherence, physical condition, quality of life). Structural equation modeling (SEM) is well suited to check several mediation models at once and to assess the global fit of the model. Bootstrap analysis is advocated for testing the statistical relevance of indirect effects, with age, sex, operation type, and initial fear controlled as covariates.

7.4. Key scientific questions for future research

Systematic validation of this hypothesis brings with it a need to answer the following major scientific questions:

- (1) Is there a dose-response relationship—between traditional health-preserving exercises and the enhancement of vmPFC–amygdala connectivity? What is the smallest effective dose of such an intervention?
- (2) How do the three elements of body regulation, breath regulation, and mind regulation contribute separately?
- (3) How does the brain's functional state change over time? Is there an earlier (pre-behavioral) stage of functional connectivity that predicts improvement?
- (4) Are the various kinesiophobia subtypes (namely “low risk perception-high kinesiophobia” versus “severe

- kinesiophobia”) differently affected by traditional health-promoting exercise intervention?
- (5) Does extinction memory stay retained indefinitely, or is it dependent upon continuous or limited practice?

8. Conclusion

Post-aortic dissection kinesiophobia is a very common clinical problem whose neural basis is not fully known. The present paper argues that the “fear extinction–brain functional remodeling” neurotheoretical hypothesis offers a unified theoretical approach to this particular phenomenon. According to the hypothesis, post-AD kinesiophobia is basically a conditioned fear reaction, and its basic neuropathology lies in inadequate top-down inhibition of the amygdala by the vmPFC and DMN dysfunction. Traditional health-preserving exercises—whether they regulate body states (with safe interoceptive exposure) or breathing (with better vagal tone and extinction consolidation) or the mind (with attentional focus or contextual adaptability)—act three-dimensionally to improve vmPFC–amygdala functional connectivity and to optimize DMN functional connectivity; extinction of kinesiophobia and improvement of long-term prognosis are thereby achieved. The hypothesis is indirectly substantiated by work on neural circuits of fear extinction, by neuroimaging of well-known health-preserving exercises, and by observations of post-AD patients, and lends itself to systematic validation with multimodal fMRI, mediation techniques, or longitudinal investigations. Should it prove correct, traditional health-preserving exercises seem to offer a secure, feasible, and neuroscience-based treatment of post-AD kinesiophobia.

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