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Review Article

Smart running as a low-cost health-promoting strategy: The HamGhadam (step-for-good) workplace physical activity program at Gol-Gohar Sirjan club

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Abstract

Physical inactivity is a major global determinant of non-communicable diseases, particularly in industrial and occupational environments where structural and environmental barriers limit regular engagement in physical activity. Although the health benefits of aerobic exercise are well established, less attention has been given to the physiological specificity of exercise intensity and its translation into feasible health promotion strategies in real world settings. Current evidence indicates that moderate intensity running (approximately 50–70% heart rate reserve, 46–63% VO_2max, or Borg RPE 12–14) induces coordinated multisystem signaling responses that support metabolic regulation and inflammatory balance. These responses include favorable modulation of myokines, adipokines, neurotrophic factors, and immunoregulatory mediators involved in metabolic homeostasis. Particular attention is given to the context dependent role of interleukin 6, highlighting the distinction between its transient exercises induced signaling effects and the chronic elevations associated with metabolic and inflammatory diseases. In addition to mechanistic insights, this review discusses translational considerations such as adherence, safety, and long term sustainability of moderate intensity running programs in occupational populations. The HamGhadam (Step for Good) initiative implemented by Gol Gohar Mining and Industrial Company is presented as a descriptive workplace case example illustrating how structured physical activity programs can be incorporated into corporate wellness initiatives. The manuscript does not claim empirical validation of the program's effectiveness but highlights its potential as a practical model for workplace health promotion. Overall, smart running is framed as a biologically efficient and potentially scalable strategy that conceptually bridges molecular exercise biology with population level physical activity promotion.

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Introduction

The pervasive epidemic of physical inactivity stands as a paramount global health determinant, intricately associated with elevated morbidity and mortality across the spectrum of non-communicable diseases, including metabolic syndrome, cardiovascular pathologies, and increasingly recognized neuropsychiatric sequelae (R. Guthold et al., 2018; I. M. Lee et al., 2012). Insufficient engagement in habitual movement not only amplifies the risk profile for chronic illness but critically undermines long-term functional capacity, thereby imposing substantial socio-economic burdens via diminished productivity and increased healthcare utilization (Wang et al., 2025).

Within specific occupational cohorts, such as those prevalent in heavy industrial sectors, the inherent structure of work imposes significant constraints on volitional physical engagement (Kusik et al., 2026). As detailed in our prior analysis concerning the GolGohar complex (Ahmadi Hekmatikar et al., 2025), the intense temporal demands and sedentary requirements associated with operational roles create a proximal barrier to achieving recommended physical activity guidelines among both the workforce and the surrounding community demographics. Recognizing this environmental determinism, we advocated for a pragmatic intervention—smart running—positioned as an accessible, scalable strategy, distinct from traditional high-performance training models (Ahmadi Hekmatikar et al., 2025).

From a Public Health standpoint, physical activity must be operationalized not merely as a recommendation but as a high-yield, low-cost prophylactic agent (R. Guthold et al., 2018). Aerobic exercise, when delivered consistently within the threshold of moderate intensity, elicits profound systemic adaptations, crucially involving improvements in cardiorespiratory efficiency, favorable modulation of body com-

-position (specifically visceral adiposity), and enhanced neurocognitive resilience (MacInnis & Gibala, 2017). The challenge remains translating these known benefits into actionable protocols that guarantee adherence and safety within complex, real-world populations (MacInnis & Gibala, 2017; MacIntosh et al., 2021; Rivera-Brown & Frontera, 2012).

This present discourse endeavors to transition from the macro-level societal context and the initial theoretical proposal toward the core physiological prescription. The necessity now shifts from why movement is critical to how movement should be precisely modulated to maximize therapeutic efficacy while minimizing risk. Therefore, the subsequent sections will systematically delineate the parameters defining moderate-intensity running—the key modulator of the beneficial exerkine/myokine signaling cascades—as the principal non-pharmacological intervention framework for mitigating the adverse physiological consequences of occupational inactivity.

Methods

Literature search and narrative synthesis

This manuscript was conducted as a narrative review with the aim of integrating mechanistic exercise physiology, exerkine biology, and public health perspectives on moderate-intensity running. A targeted literature search was performed in PubMed/MEDLINE, Scopus, and Web of Science, focusing on English-language publications. Search terms included combinations of: “moderate-intensity exercise,” “running,” “exerkines,” “myokines,” “adipokines,” “IL-6,” “BDNF,” “immune modulation,” “physical activity adherence,” and “population health.” Additional relevant studies were identified through reference list screening of key articles. Inclusion criteria comprised: (1) human studies examining moderate-intensity aerobic exercise or running; (2) experimental, observational, or mechanistic studies reporting physiological, immunological, or metabolic outcomes; and (3) reviews providing conceptual or translational insights relevant to population-level physical activity. Studies focusing exclusively on high-intensity training, elite athletes, or pharmacological interventions were excluded. Given the heterogeneity of study designs, outcomes, and populations, a qualitative narrative synthesis was undertaken rather than quantitative aggregation. Evidence was integrated thematically across molecular mechanisms, physiological adaptations, and translational public health implications.

Smart running: A low-cost approach to health promotion

Physical activity and chronic diseases

Chronic diseases constitute the leading cause of mortality worldwide, with a continuously rising prevalence across all age groups, sexes, and socio-economic strata (Abbafati et al., 2020; Collaborators, 2022; Vos et al., 2020; Wu et al., 2021). Although historically perceived as a burden primarily affecting high-income countries, contemporary epidemiological data demonstrate that the majority of chronic disease–related deaths now occur in low- and middle-income nations, while remaining a substantial public health challenge in developed societies (Abbafati et al., 2020; Collaborators, 2022; Vos et al., 2020; Wu et al., 2021). Notably, non-communicable diseases such as cardiovascular disease, type 2 diabetes, obesity, and cancer are no longer confined to adulthood, as their incidence is increasingly reported among children and adolescents (Abbafati et al., 2020; Collaborators, 2022; Vos et al., 2020; Wu et al., 2021).

A central and modifiable contributor to this global disease burden is physical inactivity (I.-M. Lee et al., 2012). Extensive epidemiological and longitudinal evidence consistently associates sedentary behavior with elevated risk for chronic disease development, premature mortality, and reduced functional capacity (Mok et al., 2019; Wen et al., 2011). Conversely, higher levels of physical activity (PA) and cardiorespiratory fitness are strongly linked to lower morbidity and mortality rates, underscoring the protective role of habitual movement across the lifespan (Mok et al., 2019; Wen et al., 2011). These associations persist across diverse populations and are observed in both primary disease prevention and secondary disease management contexts (Clerico et al., 2025; Lear et al., 2017; Mallett, 2025; Mok et al., 2019; Myers et al., 2015; Wen et al., 2011).

From a physiological perspective, PA and structured exercise exert multisystemic benefits that extend well beyond isolated disease endpoints. Regular engagement in aerobic activity favorably influences cardiovascular function, metabolic regulation, musculoskeletal integrity, immune competence, and neurocognitive health (Clerico et al., 2025; Lear et al., 2017; Mallett, 2025; Mok et al., 2019; Myers et al., 2015; Wen et al., 2011). Importantly, these adaptations are achieved through endogenous regulatory mechanisms, distinguishing physical activity from pharmacological therapies that often target single pathways and may introduce adverse side effects. In this regard, exercise may be conceptualized as a non-invasive, systemic intervention capable of optimizing physiological function rather than merely suppressing symptoms (Di Pumpo et al., 2025; Radaelli et al., 2025). Public health guidelines issued by international organizations, including the World Health Organization and professional bodies in sports and exercise medicine, consistently emphasize the accumulation of moderate-intensity aerobic activity as a foundational recommendation for health maintenance and disease prevention (Mok et al., 2025; Ranasinghe et al., 2025). Despite this consensus, global adherence to physical activity guidelines remains suboptimal with

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inactivity prevalence increasing in parallel with technological advancement, occupational sedentariness, and lifestyle constraints (Regina Guthold et al., 2018; Piercy et al., 2018). The resulting health consequences extend beyond individual morbidity, contributing to escalating healthcare expenditures, reduced workforce productivity, and broader socio-economic strain (Regina Guthold et al., 2018; Piercy et al., 2018; Pronk, 2009).

Critically, current evidence supports a clear dose–response relationship between physical activity and health outcomes, characterized by substantial inter-individual variability but consistently demonstrating meaningful risk reduction at moderate levels of activity (Arem et al., 2015; Ekelund et al., 2019). Even modest increases in habitual PA are associated with significant improvements in health markers, whereas prolonged sedentary time independently elevates disease risk (Arem et al., 2015; Ekelund et al., 2019; Lee et al., 2018). These findings reinforce the notion that the greatest public health gains may be achieved not through maximal or high-intensity exercise paradigms, but through sustainable, moderate-intensity interventions that are accessible and maintainable in real-world settings (Arem et al., 2015; Ekelund et al., 2019; Lee et al., 2018).

Within this context, physical activity has been described as a low-cost and broadly accessible health-promoting strategy rather than a truly cost-free intervention. Although aerobic exercise does not inherently require pharmacological expenditure, its successful implementation depends on time availability, behavioral motivation, safe environments, and in some cases structured guidance (Bouchard et al., 2011; Lavie et al., 2022; Pedersen & Saltin, 2015; Ross et al., 2016). When appropriately prescribed, aerobic exercise offers a low-risk, low-cost strategy capable of addressing the multifactorial nature of chronic diseases. However, the effectiveness of this intervention is contingent upon precise modulation of key exercise variables—most notably intensity (MacDonald et al., 2025). Consequently, defining and operationalizing moderate-intensity running emerges as a critical step in translating the well-established benefits of physical activity into a practical health strategy.

Smart running: Physiological & practical considerations

A central premise of smart running is the precise prescription of exercise intensity within the moderate domain, a physiological window in which aerobic metabolism predominates and adaptive signaling is maximized without imposing excessive mechanical or neuroendocrine stress (Jadon et al., 2025; Joisten et al., 2025; Tanrıverdi et al., 2025). In applied terms, this intensity range can be robustly characterized through converging indicators derived from cardiovascular, metabolic, and perceptual markers (Jadon et al., 2025; Joisten et al., 2025; Tanrıverdi et al., 2025).

From a cardiovascular standpoint, moderate intensity running corresponds to approximately 50–70% of heart rate reserve (HRR) (Howley, 2001; Yang et al., 2025). This zone reflects a stable internal load in which cardiac output rises proportionally to metabolic demand while avoiding the exponential ventilatory and lactate responses associated with higher intensities. Metabolically, the same domain aligns with roughly 46–63% of maximal oxygen uptake (VO_2max), a range consistently identified in the literature as representing the upper boundary of sustainable, predominantly oxidative exercise (Howley, 2001; Yang et al., 2025).

Perceptual validation of this physiological state is commonly achieved via the Rating of Perceived Exertion (RPE) (Eston & Connolly, 1996). During smart running, perceived effort typically falls within RPE 12–14 on the Borg 6–20 scale—reflecting a level of exertion that is clearly taxing yet comfortably maintainable for extended durations. Complementing these quantitative markers is the talk test, a pragmatic ventilatory indicator in which the ability to speak in full but not extended sentences confirms that ventilatory drive remains below the first ventilatory threshold (Eston & Connolly, 1996; Foster et al., 2008). The integration of HRR, VO_2max, RPE, and the talk test provides a multidimensional framework for defining moderate intensity running with high ecological validity. By grounding intensity regulation in these convergent metrics, smart running ensures that the imposed physiological load is sufficiently robust to activate adaptive pathways while remaining within a sustainable and recoverable range—an essential prerequisite for long term adherence and systemic health benefits.

Smart running and brain signaling pathways

Moderate intensity running elicits a distinct neurobiological signaling profile that differentiates it from both low intensity physical activity and high intensity exercise. Within this intensity domain, central adaptations are primarily mediated through coordinated neurotrophic, metabolic, and inflammatory signaling pathways, which together support brain health, cognitive function, and stress resilience.

One of the most consistently reported molecular responses to aerobic exercise at moderate intensity is the upregulation of brain derived neurotrophic factor (BDNF) (Foster et al., 2008). Running performed within approximately 50–70% of heart rate reserve or ~46–63% of VO_2max induces a sustained increase in circulating and central BDNF levels, particularly within the hippocampus and prefrontal cortex (Szuhany et al., 2015). BDNF plays a pivotal role in synaptic plasticity, neurogenesis, and neuronal survival, and its exercise induced elevation has been directly linked to improvements in learning, memory, and executive function. Importantly, moderate intensity exercise appears to optimize

Table 1. Physical activity and chronic diseases: Epidemiological evidence, mechanisms, and public health implications

Analytical Domain	Scientific Description	Public Health Implications
Global Burden of Chronic Diseases	Non-communicable diseases, including cardiovascular disease, type 2 diabetes, obesity, and cancer, represent the leading causes of global mortality, with a growing proportion of deaths occurring in low- and middle-income countries across all age groups.	Highlights the need for scalable, population-based prevention strategies targeting lifestyle-related risk factors
Role of Physical Inactivity	Physical inactivity is a central, modifiable determinant of chronic disease risk, consistently associated with increased morbidity, premature mortality, and functional decline in epidemiological and longitudinal studies.	Positions physical activity as a primary target for chronic disease prevention and health promotion
Protective Effects of Physical Activity	Higher levels of habitual physical activity and cardiorespiratory fitness are strongly associated with reduced incidence and mortality from chronic diseases across diverse populations.	Supports promotion of regular physical activity across the lifespan and clinical contexts
Physiological Mechanisms	Regular aerobic exercise induces multisystemic adaptations, including improvements in cardiovascular function, metabolic regulation, musculoskeletal health, immune function, and neurocognitive performance.	Demonstrates the systemic and preventive nature of physical activity beyond disease-specific outcomes
Comparison with Pharmacological Therapies	Unlike pharmacological treatments that often target isolated pathways, physical activity elicits endogenous regulatory adaptations with minimal adverse effects.	Reinforces exercise as a non-invasive, low-risk therapeutic strategy
International Physical Activity Guidelines	Global health organizations, including the World Health Organization, recommend regular accumulation of moderate-intensity aerobic activity for health maintenance and disease prevention.	Indicates strong scientific consensus despite low global adherence rates
Barriers to Guideline Adherence	Technological advancement, occupational sedentariness, urbanization, and time constraints contribute to increasing levels of physical inactivity worldwide.	Emphasizes the need for environmental and policy-level interventions
Dose–Response Relationship	Evidence demonstrates a clear dose–response association between physical activity and health outcomes, with meaningful risk reductions observed at moderate levels of activity.	Suggests that achievable, moderate-intensity interventions can yield substantial population health benefits
Socioeconomic Impact of Inactivity	Physical inactivity contributes to rising healthcare costs, reduced workforce productivity, and broader socioeconomic strain.	Provides economic justification for public investment in physical activity promotion
Physical Activity as a "Cost-Free Medicine"	When appropriately prescribed, aerobic exercise represents a low-cost, accessible intervention; its effectiveness is critically dependent on the precise modulation of exercise intensity.	Supports the need for operational definitions of moderate-intensity exercise in public health practice

BDNF signaling without provoking excessive cortisol responses that may attenuate neurotrophic signaling during prolonged high intensity workloads (Erickson et al., 2011; Szuhany et al., 2015). Metabolic signaling also contributes substantially to brain adaptations during smart running. Moderate intensity running promotes a controlled increase in skeletal muscle derived lactate, which readily crosses the blood–brain barrier and functions as both an energetic substrate and a signaling molecule (Heaney et al., 2014). Lactate has been shown to stimulate BDNF expression via activation of redox sensitive pathways and modulation of neuronal excitability, thereby linking peripheral metabolic stress to central neuroplastic adaptations (Heaney et al., 2014). This lactate mediated brain–muscle crosstalk is particularly relevant at moderate intensities, where lactate production is elevated but remains below levels associated with marked metabolic acidosis (Brooks, 2018).

Exercise induced cytokine signaling further shapes the cerebral response to smart running. Among these mediators, interleukin-6 (IL-6) occupies a unique position. It is essential to emphasize that exerkine responses, including IL-6 signaling, are highly context dependent. Acute, exercise induced IL-6 elevations differ fundamentally from chronic pathological elevations observed in cancer, cachexia, and inflammatory diseases, and should not be interpreted as exerting uniform or directly therapeutic effects across conditions. During moderate intensity running, IL-6 is transiently released from contracting skeletal muscle fibers in an acute, pulsatile manner (Pedersen & Febbraio, 2008). Unlike chronically elevated IL-6 observed in inflammatory and pathological states, exercise induced IL-6 acts as a myokine with neuromodulatory and anti-inflammatory properties. Acute elevations in IL-6 stimulate central anti-inflammatory cascades, including the induction of IL-10 and suppression of tumor necrosis factor-α (TNF-α), thereby contributing to a neuroprotective environment. This distinction between acute, exercise induced IL-6 signaling and chronic IL-6 exposure is critical in understanding why moderate intensity running supports, rather than impairs, brain health (Steensberg et al., 2000).

At the intracellular level, moderate intensity running activates AMP activated protein kinase (AMPK) within both peripheral tissues and the central nervous system (Hardie et al., 2012). In the brain, AMPK functions as a metabolic sensor that integrates energetic status with neuronal activity (Hardie et al., 2012). Activation of AMPK during aerobic exercise enhances mitochondrial biogenesis, improves neuronal energy efficiency, and indirectly supports BDNF signaling. Unlike high intensity and disrupt energy homeostasis, moderate intensity running maintains AMPK activation within a physiological range conducive to adaptive remodeling (Carling, 2017). Collectively, these signaling events converge to reduce neuroinflammation and improve neural resilience. Regular exposure to moderate intensity running has been associated with attenuation of microglial activation, improved blood–brain barrier integrity, and enhanced cerebral perfusion through nitric oxide–dependent vascular adaptations. These effects are particularly relevant in populations exposed to chronic psychosocial or occupational stress, where low grade neuroinflammation and impaired neuroplasticity are prevalent. In the context of smart running, the brain is therefore not a passive recipient of increased physical activity, but an active target organ responding to a finely tuned exercise stimulus. By maintaining intensity within the moderate domain, smart running maximizes neurotrophic and metabolic signaling while minimizing maladaptive stress responses, positioning it as a biologically efficient strategy for preserving cognitive function and mental health across diverse populations.

Smart running and skeletal muscle signaling pathways

Moderate intensity running induces a distinct molecular environment in skeletal muscle that favors adaptive remodeling over catabolic stress. Within this intensity domain, muscle contraction serves not only as a mechanical stimulus but also as a potent endocrine trigger, activating intracellular energy sensing pathways and promoting the release of myokines that mediate local and systemic health effects. At the intracellular level, moderate intensity running is characterized by a sustained activation of AMP activated protein kinase (AMPK), reflecting a controlled energetic challenge rather than acute metabolic crisis (Hardie et al., 2012). AMPK activation enhances glucose uptake via GLUT 4 translocation, increases fatty acid oxidation, and suppresses energetically costly anabolic processes during exercise, thereby preserving cellular homeostasis (Richter & Hargreaves, 2013). Importantly, the magnitude and duration of AMPK signaling at moderate intensity appear optimal for initiating downstream adaptations without chronically inhibiting protein synthesis pathways (Richter & Hargreaves, 2013).

One of the most critical downstream targets of AMPK activation is peroxisome proliferator activated receptor gamma coactivator -1 alpha (PGC-1α), a master regulator of mitochondrial biogenesis and oxidative metabolism (Chen et al., 2025; Jäger et al., 2007). Moderate intensity running robustly upregulates PGC-1α expression, leading to increased mitochondrial density, enhanced oxidative enzyme activity, and improved fatigue resistance. This adaptation shifts skeletal muscle phenotype toward a more oxidative profile, improving metabolic flexibility and insulin sensitivity while reducing susceptibility to metabolic stress (Coletti et al., 2022).

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In parallel, moderate intensity running exerts a regulatory effect on muscle protein turnover. Unlike high intensity or prolonged exhaustive exercise, which may excessively activate proteolytic pathways, moderate running maintains a favorable balance between synthesis and degradation (Atherton & Smith, 2012). Evidence suggests that this intensity range attenuates chronic activation of muscle atrophy mediators such as myostatin, while supporting anabolic permissiveness through reduced inflammatory signaling and improved insulin like growth factor-1 (IGF-1) responsiveness (Hittel et al., 2010). This balance is particularly relevant in populations at risk of muscle wasting, where excessive training stress may exacerbate catabolic signaling.

Beyond intracellular pathways, skeletal muscle acts as an active secretory organ during moderate intensity running, releasing a spectrum of myokines that orchestrate muscle–organ cross talk. Among these, interleukin 6 (IL-6) plays a central but context dependent role (Pedersen & Febbraio, 2008). During moderate intensity running, IL-6 is released in an acute, transient, and contraction dependent manner, functioning as a myokine rather than a pro inflammatory cytokine (Pedersen & Febbraio, 2008). This exercise induced IL-6 pulse stimulates glucose availability, enhances lipid metabolism, and initiates anti-inflammatory cascades, including the upregulation of IL-10 and suppression of tumor necrosis factor-α (TNF-α) (Fang et al., 2025). The temporal nature of IL-6 signaling is critical, as it distinguishes beneficial exercise induced responses from the deleterious effects associated with chronic low-grade inflammation (Fang et al., 2025). Moderate intensity running also influences the secretion of other myokines involved in muscle mass regulation and metabolic health. Reductions in circulating myostatin, coupled with relative increases in follistatin, create an endocrine environment permissive to muscle maintenance and remodeling. Additionally, exercise induced modulation of irisin contributes to improved muscle–adipose tissue communication, enhancing lipid utilization and metabolic efficiency without imposing excessive energetic demands on skeletal muscle (Fang et al., 2025). Collectively, these signaling events position moderate intensity running as a biologically efficient stimulus that aligns mechanical load, metabolic stress, and endocrine output within an adaptive window. In the framework of smart running, skeletal muscle is not merely trained for performance but strategically engaged as a central regulator of systemic health through precisely modulated signaling pathways.

Smart running and adipose tissue signaling pathways

Moderate intensity running exerts profound regulatory effects on adipose tissue by modulating both lipid metabolism and adipokine secretion, thereby influencing systemic inflammation, metabolic health, and disease risk. Within this intensity domain, adipose tissue responds not merely through energy depletion but via coordinated endocrine and paracrine signaling that distinguishes adaptive fat remodeling from pathological adipose loss (Kershaw & Flier, 2004).

From a metabolic standpoint, running performed at moderate intensity promotes sustained lipolysis through sympathetic activation while preserving metabolic homeostasis (Trayhurn & Wood, 2004). This intensity range enhances fatty acid mobilization and oxidation without eliciting excessive counter regulatory stress hormone responses, allowing for gradual reductions in adipose tissue mass—particularly visceral fat depots, which are strongly associated with cardiometabolic disease risk. Importantly, these changes occur independently of extreme caloric deficits, highlighting the signaling driven rather than purely energetic nature of adipose adaptation (Trayhurn & Wood, 2004). At the endocrine level, moderate intensity running induces a favorable shift in adipokine profiles, characterized by reductions in pro inflammatory mediators and restoration of anti-inflammatory signaling (Horowitz, 2003). One of the most consistently observed responses is a decrease in circulating leptin, particularly when exercise is performed chronically within moderate intensity ranges (Horowitz, 2003). While leptin is primarily recognized as an energy regulating hormone, its role as a pro inflammatory adipokine is increasingly evident. Elevated leptin levels activate immune cells via JAK–STAT and NF-κB pathways, promoting the production of inflammatory cytokines such as TNF-α and IL-6 (La Cava & Matarese, 2004). By reducing leptin concentrations and leptin resistance, moderate intensity running attenuates adipose derived inflammatory signaling and contributes to improved immune–metabolic balance (La Cava & Matarese, 2004; Otero et al., 2005).

Concurrently, moderate intensity running is associated with increases in adiponectin, a key anti-inflammatory and insulin sensitizing adipokine. Adiponectin enhances fatty acid oxidation and glucose utilization through activation of AMPK dependent pathways and suppresses pro inflammatory cytokine production in macrophages and endothelial cells (Otero et al., 2005). The exercise induced elevation of adiponectin is particularly relevant in populations with obesity or metabolic dysfunction, where low adiponectin levels are closely linked to chronic low grade inflammation, insulin resistance, and increased cardiovascular risk (Otero et al., 2005; Yamauchi et al., 2002). By restoring adiponectin signaling, smart running promotes a shift toward an anti-inflammatory adipose tissue phenotype. Beyond leptin and adiponectin, moderate intensity running influences emerging adipokines implicated in adipose tissue inflammation and remodeling (Yamauchi et al., 2002). Chronic aerobic exercise within moderate intensity ranges has been shown to reduce circulating levels of chemerin, an adipokine associated with adipose tissue inflammation, macrophage recruitment, and insulin resistance. Lower chemerin levels are consistently observed in physically active individuals and are associated with improvements in metabolic health markers (Ouchi et al., 2011). Similarly, moderate intensity running may modulate omentin, an adipokine with anti-inflammatory and vasoprotective properties, although responses appear to be intensity and population dependent (Ouchi et al., 2011).

At the tissue level, these adipokine shifts reflect qualitative remodeling of adipose tissue rather than indiscriminate fat loss (Sirico et al., 2018). Moderate intensity running reduces adipocyte hypertrophy, limits hypoxia induced inflammatory signaling, and suppresses the infiltration and activation of pro inflammatory M1 macrophages. This remodeling process results in decreased secretion of TNF-α and IL-6 from adipose tissue, thereby reducing systemic inflammatory burden—a central driver of non-communicable diseases, including type 2 diabetes, cardiovascular disease, and certain cancers (Sirico et al., 2018). The anti-inflammatory effects of moderate intensity running are particularly relevant in the context of chronic disease prevention (Sirico et al., 2018). Adipose tissue–derived inflammation is a major contributor to insulin resistance, endothelial dysfunction, and atherosclerosis. By simultaneously reducing fat mass, improving adipokine profiles, and attenuating inflammatory signaling, smart running acts as a multi target intervention that addresses both the cause and consequence of adipose tissue dysfunction (Sirico et al., 2018). Within the framework of smart running, adipose tissue is therefore not simply a passive energy reservoir but an active endocrine organ whose signaling profile can be reshaped through precisely prescribed exercise intensity. Moderate intensity running occupies a critical physiological window in which adipose tissue remodeling, systemic inflammation reduction, and disease risk attenuation converge, reinforcing its role as a biologically efficient and sustainable strategy for long term health promotion.

Smart running and immune system signaling pathways

Moderate intensity running induces a highly regulated immunological response that differs fundamentally from both sed-

-entary behavior and excessive exercise stress. Within this intensity domain, immune adaptations are characterized by transient activation followed by long term immune regulation, resulting in enhanced immune surveillance, reduced chronic inflammation, and improved host defense without immunosuppression. One of the defining features of moderate intensity running is its effect on exercise induced cytokine signaling, particularly the context dependent role of interleukin 6 (IL-6). During moderate aerobic running, IL-6 is released predominantly from contracting skeletal muscle in an acute, pulsatile manner, acting as a myokine rather than a pro inflammatory cytokine. This transient IL-6 response initiates downstream anti-inflammatory cascades, including the induction of IL-10 and IL-1 receptor antagonist, while suppressing tumor necrosis factor α (TNF-α) signaling. Importantly, this exercise induced IL-6 profile contrasts sharply with the chronically elevated IL-6 observed in cancer and cachexia, where sustained exposure drives systemic inflammation, immune dysfunction, and tissue catabolism.

Moderate intensity running also exerts a regulatory influence on innate immune cell dynamics, particularly natural killer (NK) cells and macrophages. Acute bouts of aerobic exercise within moderate intensity ranges transiently increase circulating NK cell number and cytotoxic activity, enhancing immune surveillance against transformed or malignant cells. With repeated exposure, these adaptations contribute to improved immune readiness without inducing the immune exhaustion or transient suppression associated with prolonged high intensity training. This distinction is critical in oncological and high risk populations, where immune competence must be preserved rather than overstimulated. Macrophage polarization represents another key mechanism through which moderate intensity running modulates immune function. Chronic aerobic exercise at moderate intensity promotes a shift away from pro inflammatory M1 macrophage phenotypes toward anti-inflammatory and tissue repair oriented M2 phenotypes. This shift is mediated in part by reductions in adipose tissue–derived inflammatory signaling and by exercise induced myokines that suppress NF-κB activation. As a consequence, systemic levels of TNF-α, IL-1β, and IL-6 decline over time, leading to attenuation of chronic low-grade inflammation—a central driver of cancer progression, insulin resistance, and cardiovascular disease.

From a systemic perspective, moderate intensity running improves immune–metabolic integration by reducing the inflammatory burden imposed by dysfunctional adipose tissue. As reductions in visceral fat mass are accompanied by favorable adipokine remodeling (↓leptin, ↑adiponectin), immune cell activation within adipose tissue is suppressed, limiting the continuous release of pro inflammatory mediators into circulation. This reduction in background inflammation restores immune sensitivity and responsiveness, which is often impaired in individuals with obesity, metabolic syndrome, or cancer related inflammation. In the context of cancer, these immunomodulatory effects are particularly relevant. Chronic inflammation is a hallmark of tumor progression and cachexia, contributing to immune escape, muscle and fat wasting, and reduced tolerance to therapy. Moderate intensity running, through its capacity to downregulate systemic inflammatory signaling while preserving innate immune function, creates an immunological environment less permissive to tumor associated inflammation. While exercise is not a direct anti-cancer therapy, the regulation of immune signaling through smart running may enhance host resilience, improve inflammatory control, and support the effectiveness of conventional treatments.

Crucially, the intensity of exercise determines the direction of immune adaptation. Excessive or prolonged high intensity training may induce transient immunosuppression, characterized by reductions in lymphocyte function and increased susceptibility to infection. In contrast, moderate intensity running occupies a physiological window in which immune activation and recovery are optimally balanced, reinforcing the importance of precise intensity prescription within the smart running framework.Taken together, moderate intensity running functions as an immune regulatory intervention rather than an immune stressor. By aligning exercise load with physiological signaling thresholds, smart running modulates cytokine networks, innate immune cell function, and systemic inflammation in a manner conducive to long term health and disease risk reduction, including conditions characterized by chronic inflammation such as cancer.

Gol Gohar and the HamGhadam program: An illustrative workplace physical activity case

This section presents the HamGhadam (Step-for-Good) program as an illustrative, descriptive case example intended to contextualize the application of a workplace physical activity intervention in an organizational setting, without claiming causal effectiveness. This section is intended solely as a descriptive case illustration and should not be interpreted as empirical evidence of effectiveness or as validation of the intervention.

Running is widely recognized as one of the most accessible and cost effective forms of physical activity, requiring minimal infrastructure while providing substantial physical and mental health benefits (Bernhart et al., 2025; I. M. Lee et al., 2012). Owing to these characteristics, running has played a central role in the global expansion of mass participation sporting events over recent decades, including charity oriented running initiatives. Beyond their fundraising function, such events have gained increasing social recognition as platforms for promoting active lifestyles, fostering social cohesion, and reinforcing collective responsibility toward health and social causes.

Table 2. Smart running: Exercise intensity domains and proposed physiological pathways.

Target System	Moderate-Intensity Domain (Smart Running)	Key Signaling Pathways	Functional and Clinical Implications
Exercise Intensity Definition	~50–70% heart rate reserve (HRR); ~46–63% VO_2max; RPE 12–14; positive talk test	Integrated cardiovascular, metabolic, and perceptual regulation	Ensures sufficient physiological stimulus while avoiding excessive mechanical and neuroendocrine stress
Central Nervous System	Sustained aerobic metabolic state without excessive cortisol response	↑ BDNF; controlled lactate signaling; AMPK activation; ↓ neuroinflammation	Enhanced neuroplasticity, cognitive function, stress resilience, and preservation of brain health
Brain–Muscle Crosstalk	Moderate lactate accumulation below metabolic acidosis threshold	Lactate-mediated BDNF upregulation; redox-sensitive signaling	Efficient coupling of peripheral metabolic stress to central neuroadaptive responses
Neuroinflammatory Regulation	Acute, transient cytokine response	↑ IL-6 (myokine); ↑ IL-10; ↓ TNF-α; reduced microglial activation	Creation of a neuroprotective and anti-inflammatory cerebral environment
Skeletal Muscle (Energy Sensing)	Controlled energetic challenge	Sustained AMPK activation; GLUT4 translocation	Improved glucose uptake, lipid oxidation, and metabolic flexibility
Mitochondrial Remodeling	Predominantly oxidative workload	↑ PGC-1α expression; mitochondrial biogenesis	Enhanced fatigue resistance and insulin sensitivity
Muscle Protein Turnover	Balanced anabolic–catabolic signaling	↓ Myostatin; ↑ IGF-1 responsiveness; reduced proteolysis	Preservation of muscle mass, especially in at-risk populations
Myokine Secretion	Contraction-dependent endocrine response	IL-6 (acute); ↑ IL-10; ↓ TNF-α; ↑ irisin; ↑ follistatin	Systemic anti-inflammatory effects and improved muscle–adipose communication
Adipose Tissue Metabolism	Sustained lipolysis without excessive stress hormone activation	↑ Fatty acid oxidation; ↓ visceral adiposity	Reduction of cardiometabolic risk factors
Adipokine Profile	Qualitative adipose tissue remodeling	↓ Leptin; ↑ Adiponectin; ↓ Chemerin; ↑ Omentin	Attenuation of adipose-derived inflammation and improved insulin sensitivity
Immune System Regulation	Balanced immune activation and recovery	↑ NK cell activity; M1 ⟶ M2 macrophage polarization; ↓ NF-κB signaling	Enhanced immune surveillance with reduced chronic inflammation
Systemic Inflammatory Load	Reduction in low-grade chronic inflammation	↓ TNF-α; ↓ IL-1β; ↓ chronic IL-6 exposure	Lower risk of non-communicable diseases, including cancer

Importantly, the HamGhadam initiative is discussed in this manuscript as a conceptual and translational example rather than as a validated health intervention. At present, no independently audited data are available regarding participant adherence, intensity regulation, injury risk, or long-term behavioral sustainability. As with other digitally mediated physical activity platforms, potential limitations include digital exclusion among lower-literacy populations, data privacy considerations, motivational decay over time, and the risk of unsupervised exercise-related injury. These considerations underscore the need for cautious interpretation and highlight the necessity of independent evaluation before broader public health generalization. The HamGhadam initiative is presented in this manuscript as an illustrative translational example rather than as an evidence-validated intervention. At present, no independently published evaluations are available regarding its effectiveness on health outcomes, adherence trajectories, injury incidence, or long-term sustainability. Accordingly, the discussion of HamGhadam is intentionally descriptive and hypothesis-generating. Its inclusion serves to contextualize how principles of smart running might be operationalized within a real-world occupational and corporate social responsibility framework, rather than to assert causal or comparative effectiveness.Charity running events constitute a distinctive form of physical activity engagement in which individual health behaviors are explicitly linked to collective social outcomes (Bernhart et al., 2025; I. M. Lee et al., 2012). Existing evidence suggests that participation in charity runs is driven by an interplay of health related, psychological, social, and altruistic motivations, including support for meaningful causes, strengthening of social ties, and the reinforcement of personal identity as both physically active and socially responsible. Importantly, these events may function as forms of embodied or "living" media, through which health norms and social values are communicated via active participation rather than passive information exchange (Stamatakis et al., 2023). Within this global context, Gol Gohar Mining and Industrial Company has introduced the "HamGhadam" (Step for Good) initiative as part of its corporate social responsibility and sustainability strategy. Unlike conventional charity races that are limited to single events, HamGhadam operationalizes the concept of charity running through a digital, continuous, and scalable platform, whereby participants accumulate steps or running distance via a mobile application, and each step is translated into a tangible financial contribution to charitable causes. This model effectively integrates smart running—defined by accessible, moderate intensity, and sustainable physical activity—with structured philanthropic impact.

From a public health perspective, HamGhadam addresses a critical limitation of many charity running events: their episodic nature. While mass charity runs have been shown to temporarily motivate participation among both active and inactive individuals, evidence suggests that a single event does not necessarily induce long term lifestyle change, particularly among individuals with passive or sedentary lifestyles. By contrast, the HamGhadam model shifts the focus from one time participation to habitual engagement, reinforcing regular physical activity through continuous feedback, social recognition, and altruistic reward. Behavioral science frameworks help explain the potential effectiveness of this approach. Studies on charity sport events demonstrate that participants are driven not only by physical activity motives but also by empathy, social identity, and perceived social value. Research consistently shows that the awareness of contributing to a charitable cause enhances psychological benefits, including satisfaction, sense of purpose, and motivation to remain active. These effects are particularly pronounced when participants perceive their actions as meaningful beyond personal health gains. In this regard, HamGhadam aligns individual exercise behavior with broader social outcomes, strengthening motivation through dual reinforcement: personal health and social contribution.

Moreover, charity based physical activity initiatives can be interpreted through the lens of social capital theory. Participation in collective pro social activities fosters trust, shared norms, and networks that facilitate cooperative behavior. Running for a cause not only promotes physical health but also reinforces a sense of belonging and shared responsibility, thereby generating “bridging social capital” that extends beyond immediate social groups. By embedding charitable running within the organizational and community structure of Gol Gohar, HamGhadam contributes to the creation of a health oriented culture that links employee well-being, community engagement, and corporate sustainability.

Crucially, the concept of smart running within the HamGhadam initiative extends beyond step counting. By implicitly encouraging moderate intensity, regular physical activity—rather than extreme or competitive performance—the initiative aligns with evidence based exercise recommendations that optimize immune regulation, metabolic health, and long term adherence. This is particularly relevant in industrial and occupational settings, where time constraints, work related stress, and sedentary behaviors increase the risk of non-communicable diseases. Smart running, when embedded in a pro social framework, becomes a practical and inclusive intervention capable of reaching individuals who might otherwise disengage from structured exercise programs. In summary, HamGhadam represents an innovative convergence of exercise physiology, public health promotion, and corporate social responsibility. By transforming everyday movement into a socially meaningful act, the initiative redefines charity running as a sustained, health promoting behavior rather than a symbolic event. Within the broader framework of smart running, HamGhadam illustrates how moderate intensity physical activity can simultaneously support individual health, strengthen social capital, and advance sustainable development goals—positioning it as a scalable model for pro social health promotion in industrial communities. In summary, HamGhadam represents an innovative convergence of exercise physiology, public health promotion, and corporate social responsibility. By transforming everyday movement into a socially meaningful act, the initiative redefines charity running as a sustained, health promoting behavior rather than a symbolic event. Within the broader framework of smart running, HamGhadam illustrates how moderate intensity physical activity can simultaneously support individual health, strengthen social capital, and advance sustainable development goals—positioning it as a scalable model for pro social health promotion in industrial communities.

Despite its conceptual appeal, several limitations and potential risks associated with the HamGhadam initiative should be acknowledged. First, reliance on a digital platform may exacerbate digital exclusion, particularly among older workers, individuals with limited technological literacy, or those without consistent access to smartphones and internet connectivity. Second, the collection and processing of activity data raise unresolved concerns regarding data privacy, ownership, and informed consent, especially when health-related behaviors are embedded within corporate infrastructures. Third, gamification-based motivation, while effective in the short term, may be vulnerable to motivational decay once novelty diminishes or external incentives lose salience. Sustained behavior change typically requires complementary strategies, including autonomy-supportive environments and periodic re-engagement mechanisms. Fourth, participation in unsupervised running programs carries a non-trivial risk of musculoskeletal injury, particularly among previously inactive individuals. Without individualized screening or progression guidelines, such initiatives may unintentionally increase injury burden or disengagement.

Discussion

The present article advances the concept of smart running as a biologically efficient, socially scalable, and economically sustainable health intervention, integrating contemporary insights from exercise physiology, exerkine biology, immunometabolism, and public health implementation. By systematically examining moderate intensity running as a central modulator of multi organ signaling pathways, this work positions physical activity not merely as a behavioral recommendation, but as a precision regulated biological stimulus capable of addressing the complex pathophysiology underlying chronic diseases and occupational inactivity. A key strength of this framework lies in its intensity specific orientation. While the health benefits of physical activity are well established, much of the existing literature remains agnostic to the qualitative differences imposed by exercise intensity. The present analysis demonstrates that moderate intensity running occupies distinct physiological window in which

Table 3. HamQadam initiative: Integrating smart running with prosocial health promotion

Key Component	Description	Implications for Health Promotion (Conceptual and Hypothesis-Generating)
Foundation of Initiative	Digital, continuous, scalable platform integrating smart running and charitable contributions	Shift from episodic events to habitual engagement, promoting sustained physical activity
Concept of Smart Running	Encourages moderate-intensity, regular activity aligned with evidence-based recommendations	Optimizes immune regulation, metabolic health, and adherence, especially in sedentary populations
Corporate Social Responsibility	Developed by Gol Gohar Mining and Industrial Company as part of sustainability strategy	Links employee well-being with community engagement and corporate responsibility
Motivational Drivers	Health, altruism, social identity, empathy, perceived social value	Enhances psychological benefits, sense of purpose, and motivation through dual reinforcement: health and social contribution
Behavioral Science Foundations	Empathy and social identity drive participation; aligns with social capital theory	Fosters trust, shared norms, networks, and cooperative behavior, generating “bridging social capital”
Public Health Perspectives	Addresses limitations of single-event charity runs; promotes regular activity	Reinforces lifestyle change, reducing risk of non-communicable diseases in occupational settings
Social Capital and Community Engagement	Participation strengthens sense of belonging and shared responsibility	Supports creation of health-oriented culture within industrial community structure
Broader Public Health Outcomes	Health and social outcomes are aligned: individual health, social capital, and sustainable development	Positions HamQadam as scalable model for industrial communities, promoting inclusive health

adaptive signaling outweighs stress induced dysregulation. Across neural, muscular, adipose, and immune systems, this intensity range consistently promotes favorable exerkine profiles—characterized by transient, pulsatile signaling rather than chronic elevation—thereby supporting systemic homeostasis.

Public health considerations, equity, and translational limitations

From a public health perspective, the translation of biologically plausible exercise strategies into population-level benefit is contingent upon equity of access. Although running is often described as a low-cost and accessible form of physical activity, structural barriers—including occupational demands, baseline health status, digital literacy, age, disability, and gender-related constraints—may limit participation for certain subgroups. In industrial settings, shift work, fatigue, and unequal access to supportive environments may further exacerbate disparities in engagement. Consequently, smart running should not be assumed to be universally accessible, and targeted adaptations are required to avoid reinforcing existing health inequities. Adherence represents a central challenge in all physical activity interventions. While moderate-intensity running offers advantages in terms of feasibility and reduced injury risk compared with high-intensity or highly structured programs, long-term adherence remains highly variable. Motivation, social support, prior exercise experience, and perceived meaning strongly influence sustained engagement. Prosocial frameworks, such as charity-based initiatives, may enhance adherence by linking physical activity to social value; however, empirical evidence supporting long-term behavioral maintenance at scale remains limited and context-specific.

Safety considerations are equally critical. Although moderate-intensity running is generally well tolerated, it is not risk-free, particularly among previously inactive individuals, those with cardiometabolic disease, musculoskeletal limitations, or advanced age. Inadequate progression, insufficient recovery, or inappropriate intensity prescription may increase the risk of injury or adverse events. Therefore, smart running should be framed as a guided and adaptable approach rather than a uniform prescription, emphasizing gradual progression, individual monitoring, and context-sensitive implementation. Individual variability in physiological and behavioral responses further complicates translation. Exerkine responses, cardiorespiratory adaptations, and immune modulation exhibit substantial inter-individual heterogeneity influenced by genetics, sex, age, baseline fitness, metabolic health, and disease status. As a result, favorable biological responses observed at the group level may not uniformly translate to benefit for all individuals. Recognition of this variability is essential to avoid overgeneralization and to support personalized, rather than one-size-fits-all, interpretations of smart running frameworks.

Critically, the present work distinguishes between biological plausibility and proven population-level outcomes. While substantial mechanistic evidence supports the role of moderate-intensity running in modulating multisystem signaling pathways, including exerkine-mediated immune and metabolic regulation, such mechanisms alone do not constitute evidence of effectiveness at the population level. Demonstration of public health impact requires longitudinal, controlled evaluations assessing adherence, safety, equity, and clinically meaningful outcomes across diverse populations. Accordingly, the framework presented here should be interpreted as hypothesis-generating and translational, rather than as definitive evidence of population-wide effectiveness.

Exerkine centered integration across organ systems

One of the central contributions of this work is the unified exerkine perspective. Moderate intensity running induces coordinated secretion of myokines (e.g., IL-6, irisin, follistatin), adipokines (↓leptin, ↑adiponectin), and neurotrophic mediators (↑BDNF), which collectively regulate inflammation, metabolism, and tissue remodeling. Importantly, the dual nature of IL-6 is explicitly addressed: while chronically elevated IL-6 is implicated in cancer progression, cachexia, and metabolic dysfunction, the acute IL-6 surge induced by moderate exercise functions as an anti-inflammatory and metabolic regulator. This distinction is critical and frequently misunderstood in both clinical and public discourse. The immunological implications of this signaling pattern are particularly relevant. Moderate intensity running enhances innate immune surveillance—most notably through transient increases in NK cell mobilization—while simultaneously suppressing chronic low grade inflammation via macrophage polarization toward the M2 phenotype. Such adaptations are highly relevant in populations exposed to persistent inflammatory stressors, including industrial workers, individuals with obesity or metabolic syndrome, and cancer survivors. Importantly, the avoidance of excessive training stress mitigates the risk of exercise induced immunosuppression, reinforcing the necessity of precision intensity prescription.

Relevance to cancer, cachexia, and chronic inflammation

Grounded in the exerkine literature—including prior work on cancer related cachexia—the present framework cautiously extends its relevance to oncology related contexts. Grounded in the exerkine literature, including prior work on cancer-related cachexia, the present framework cautiously extends its discussion to oncology-related contexts as a biologically plausible, hypothesis-generating perspective. Exercise is not proposed as a therapeutic or disease-modifying intervention in cancer. Rather, moderate-intensity running is discussed in relation to its potential to influence systemic inflammatory tone, muscle signaling integrity, and adipose-derived cytokine profiles—mechanisms that may be relevant to the broader pathophysiology of cachexia and chronic inflammation. These interpretations are indirect and mechanistic in nature and should not be construed as evidence of clinical benefit, tumor control, or oncologic risk modification. This position is intentionally conservative and aligns with current consensus in exercise oncology, avoiding overstatement while highlighting meaningful mechanistic plausibility.

From physiology to population health: The Role of smart running

A persistent challenge in public health is translating mechanistic knowledge into interventions that are adoptable, maintainable, and equitable. High intensity or highly structured exercise progr-ams, despite physiological efficacy, often fail at the population level due to poor adherence, injury risk, and accessibility barriers. Smart running directly addresses this translational gap by emphasizing moderate intensity, low cost, minimal equipment, and high ecological validity. The framing of running as a low-cost health-promoting strategy is not intended to imply the absence of practical or social constraints. Rather, it reflects the relative economic efficiency of moderate-intensity running compared with resource-intensive medical or fitness-based interventions. Time commitment, motivational demands, environmental safety, and injury prevention remain critical determinants of real-world feasibility and adherence. Even modest engagement within the moderate intensity domain yields disproportionate health returns, particularly among previously inactive individuals. This aligns with epidemiological evidence indicating that the greatest reductions in morbidity and mortality occur when sedentary individual's transition to moderate activity levels, rather than when already active individuals pursue maximal training volumes.

Gol Gohar and the HamGhadam initiative: An illustrative case of applied smart running within corporate social responsibility

Within this theoretical and physiological framework, the HamGhadam (Step for Good) initiative implemented by Gol Gohar Mining and Industrial Company represents a notable case of evidence aligned corporate social responsibility. Rather than adopting a symbolic or episodic charity model, Gol Gohar has operationalized smart running through a continuous, app based platform that links everyday movement to measurable social impact.

This approach addresses multiple barriers simultaneously:

1. Time and access constraints inherent to industrial work environments

2. Motivational decay associated with purely health oriented exercise messaging

3. Lack of sustainability observed in one off charity sporting events

By embedding moderate intensity movement within a prosocial framework, HamGhadam introduces dual reinforcement motivation: individuals engage in physical activity not only for personal health benefits but also for altruistic contribution. This structure is strongly supported by behavioral and social science literature on charity sport events, which demonstrates that empathy, social identity, and perceived social value significantly enhance participation and adherence.

Moreover, the initiative contributes to the generation of bridging social capital, strengthening trust and shared norms across orga-

-nizational and community boundaries. In industrial regions where occupational stress, sedentary behavior, and health disparities often coexist, such integrative models are particularly valuable. From an analytical perspective, the initiative can be interpreted as an example of how institutional support may lower certain barriers to physical activity participation. Nevertheless, institutional facilitation does not guarantee equitable access, sustained engagement, or measurable health benefit, particularly in the absence of structured supervision or independent evaluation. Importantly, the mechanistic pathways discussed herein are derived from heterogeneous preclinical, physiological, and observational evidence, and do not permit causal inference regarding cancer outcomes or cachexia reversal. Future controlled clinical studies are required to directly test these hypotheses.

Conclusion

Smart running, when appropriately prescribed within the moderate intensity domain, represents a low cost and accessible approach that is biologically plausible and potentially relevant for population level health promotion, while requiring careful contextualization and empirical validation. Through coordinated exerkine signaling across neural, muscular, adipose, and immune systems, moderate intensity running promotes systemic resilience, attenuates chronic inflammation, and supports long term health maintenance. The HamGhadam initiative exemplifies how such a physiological framework can be effectively translated into real world practice through innovative corporate social responsibility. The HamGhadam initiative illustrates how a physiologically grounded exercise framework may be operationalized within a corporate social responsibility context. However, its inclusion in this review is intended to be illustrative rather than evaluative. At present, no independent data are available regarding participation patterns, physiological outcomes, injury incidence, or long-term adherence. Consequently, the effectiveness, scalability, and sustainability of the initiative remain uncertain and warrant cautious interpretation. Instead, smart running provides a unifying platform through which individual well-being and collective benefit can be simultaneously advanced. Future health strategies—particularly within occupational and industrial settings—may benefit from adopting similar models that integrate precision exercise prescription, digital engagement, and prosocial incentives. In this regard, smart running should be viewed not as an alternative to structured exercise programs, but as a foundational, population level intervention upon which more specialized strategies can be built.

Critical perspectives and limitations of the HamGhadam program

Although the HamGhadam program illustrates a potentially feasible workplace physical activity initiative, several limitations should be considered. Digital health promotion initiatives may unintentionally exclude employees with limited access to technology or lower digital literacy. In addition, the collection and use of activity-related data raise privacy and governance concerns in occupational settings. Gamification strategies may improve engagement in the short term, but they can also create unintended pressure, comparison effects, or motivational fatigue over time. Moreover, unsupervised increases in running volume may increase the risk of musculoskeletal injury, particularly among previously inactive participants. Finally, long-term adherence and sustainability remain uncertain, and should be evaluated in independent studies before broader implementation or translation to other settings.

What is already known on this subject?

Physical inactivity is a major global determinant of non-communicable diseases, particularly in industrial and occupational settings where structural and environmental barriers limit regular physical activity. Although the health benefits of aerobic exercise are well established, less attention has been paid to the physiological specificity of exercise intensity and its translation into feasible health-promotion strategies in real-world contexts. Current evidence shows that moderate-intensity running elicits coordinated multisystem signaling responses that support metabolic regulation and inflammatory balance. In addition, physical inactivity and sedentary behavior are consistently associated with increased risk of chronic disease, premature mortality, and reduced functional capacity, whereas higher levels of physical activity and cardiorespiratory fitness are linked to lower morbidity and mortality.

What this study adds?

This narrative review introduces the concept of “smart running” as a biologically informed and potentially scalable strategy for workplace health promotion. The study integrates current evidence on moderate-intensity running with mechanistic insights related to myokines, adipokines, neurotrophic factors, and inflammatory regulation, particularly the context-dependent role of interleukin-6. In addition, the HamGhadam (Step for Good) initiative is presented as a descriptive workplace case example illustrating how structured physical-activity programs may be incorporated into occupational wellness settings. The manuscript also highlights practical considerations related to feasibility, adherence, safety, and long-term sustainability of workplace running programs.

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Data availability

Data will be made available upon reasonable request.

Compliance with ethical standards

Conflict of interest

The authors were involved in the conceptual development and/or scientific advisory aspects of the HamGhadam program, which is presented in this manuscript as a locally adapted workplace physical activity intervention rather than as a commercial product or digital platform. The authors state that the analyses and interpretations are independent and do not constitute an endorsement of any commercial entity. Nevertheless, to ensure full transparency, this involvement is disclosed as a potential non-financial conflict of interest.

Ethical approval

Review article

Informed consent

Performed.

Author contributions

Conceptualization and development of the smart running framework: A.A.H. and H.A.P.; Scientific input and contextual analysis related to the HamGhadam case example: A.A.H.; Writing—original draft preparation: A.A.H., A.KH., and KH.P.; Review and editing: all authors. All authors have read and approved the final manuscript.

References

Abbafati, C., Machado, D., Cislaghi, B., Salman, O., Karanikolos, M., McKee, M., . . . Trias-Llimós, S. (2020). Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. doi: https://doi.org/10.1016/S0140-6736(20)30925-9

Ahmadi Hekmatikar, A. H., Suzuki, K., & Fernández Lázaro, D. (2025). Physical literacy as the missing link in industrial health: A narrative review and conceptual analysis informed by the Sirjan context and the Golgohar cultural and sports club initiative. Journal of Exercise & Organ Cross Talk, 5(4), 207-214. doi: https://doi.org/10.22122/jeoct.2025.561355.1180

Arem, H., Moore, S. C., Patel, A., Hartge, P., De Gonzalez, A. B., Visvanathan, K., . . . Adami, H. O. (2015). Leisure time physical activity and mortality: a detailed pooled analysis of the dose-response relationship. JAMA internal medicine, 175(6), 959-967. doi: https://doi.org/10.1001/jamainternmed.2015.0533

Atherton, P. J., & Smith, K. (2012). Muscle protein synthesis in response to nutrition and exercise. J Physiol, 590(5), 1049-1057. https://doi.org/10.1113/jphysiol.2011.225003

Bernhart, J. A., Wilcox, S., McKeever, B. W., Ehlers, D. K., & O'Neill, J. R. (2025). A Self-Determination Theory Application to Physical Activity in Charity Sports Events. Am J Lifestyle Med, 19(2), 242-252. https://doi.org/10.1177/15598276221077204

Bouchard, C., Sarzynski, M. A., Rice, T. K., Kraus, W. E., Church, T. S., Sung, Y. J., . . . Rankinen, T. (2011). Genomic predictors of the maximal O2 uptake response to standardized exercise training programs. Journal of applied physiology, 110(5), 1160-1170. doi: https://doi.org/10.1152/japplphysiol.00973.2010

Brooks, G. A. (2018). The Science and Translation of Lactate Shuttle Theory. Cell Metab, 27(4), 757-785. https://doi.org/10.1016/j.cmet.2018.03.008

Carling, D. (2017). AMPK signalling in health and disease. Curr Opin Cell Biol, 45, 31-37. https://doi.org/10.1016/j.ceb.2017.01.005

Chen, J., Liu, B., Yao, X., Yang, X., Sun, J., Yi, J., . . . Sun, H. (2025). AMPK/SIRT1/PGC-1α Signaling Pathway: Molecular Mechanisms and Targeted Strategies From Energy Homeostasis Regulation to Disease Therapy. CNS Neurosci Ther, 31(11), e70657. https://doi.org/10.1111/cns.70657

Clerico, A., Zaninotto, M., Aimo, A., Galli, C., Sandri, M. T., Correale, M., . . . Belloni, L. (2025). Assessment of cardiovascular risk and physical activity: the role of cardiac-specific biomarkers in the general population and athletes: A consensus document by the Study Group on Cardiac Biomarkers from the Italian Society of Clinical Biochemistry (SIBioC) and the European Ligand Assay Society (ELAS). Clinical Chemistry and Laboratory Medicine (CCLM), 63(1), 71-86. doi: https://doi.org/10.1515/cclm-2024-0596

Coletti, C., Acosta, G. F., Keslacy, S., & Coletti, D. (2022). Exercise-mediated reinnervation of skeletal muscle in elderly people: An update. Eur J Transl Myol, 32(1). https://doi.org/10.4081/ejtm.2022.10416

Collaborators, G. M. D. (2022). Global, regional, and national burden of 12 mental disorders in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. The Lancet Psychiatry, 9(2), 137-150. doi: https://doi.org/10.2165/00007256-199621030-00003

Di Pumpo, M., Miatton, A., Riccardi, M., Graps, E., Baldo, V., Buja, A., & Damiani, G. (2025). Digital health interventions to promote physical activity in Community-Dwelling older adults: A systematic review and semiquantitative analysis. International Journal of Public Health, 69, 1607720. doi: https://doi.org/10.1111/cns.70657

Ekelund, U., Tarp, J., Steene-Johannessen, J., Hansen, B. H., Jefferis, B., Fagerland, M. W., . . . Chernofsky, A. (2019). Dose-response associations between accelerometry measured physical activity and sedentary time and all cause mortality: systematic review and harmonised meta-analysis. Bmj, 366. doi: doi: https://doi.org/10.1016/S2215-0366(21)00395-3.

Erickson, K. I., Voss, M. W., Prakash, R. S., Basak, C., Szabo, A., Chaddock, L., . . . Kramer, A. F. (2011). Exercise training increases size of hippocampus and improves memory. Proc Natl Acad Sci U S A, 108(7), 3017-3022. https://doi.org/10.1073/pnas.1015950108

Eston, R., & Connolly, D. (1996). The use of ratings of perceived exertion for exercise prescription in patients receiving beta-blocker therapy. Sports Med, 21(3), 176-190. https://doi.org/10.2165/00007256-199621030-00003

Fang, L., Chang, Y., Liang, Q., Huang, R., Yi, X., & Yao, T. (2025). Phoenixin's influence on HPG axis and inflammation in elite ice hockey athletes: A cross-sectional analysis. Peptides, 184, 171351. https://doi.org/10.1016/j.peptides.2025.171351

Foster, C., Porcari, J. P., Anderson, J., Paulson, M., Smaczny, D., Webber, H., . . . Udermann, B. (2008). The talk test as a marker of exercise training intensity. J Cardiopulm Rehabil Prev, 28(1), 24-30; quiz 31-22. https://doi.org/10.1097/01.Hcr.0000311504.41775.78

Guthold, R., Stevens, G. A., Riley, L. M., & Bull, F. C. (2018). Worldwide trends in insufficient physical activity from 2001 to 2016: a pooled analysis of 358 population-based surveys with 1·9 million participants. Lancet Glob Health, 6(10), e1077-e1086. https://doi.org/10.1016/s2214-109x(18)30357-7

Guthold, R., Stevens, G. A., Riley, L. M., & Bull, F. C. (2018). Worldwide trends in insufficient physical activity from 2001 to 2016: a pooled analysis of 358 population-based surveys with 1· 9 million participants. The lancet global health, 6(10), e1077-e1086. doi: https://doi.org/10.1016/S2215-0366(21)00395-3.

Hardie, D. G., Ross, F. A., & Hawley, S. A. (2012). AMPK: a nutrient and energy sensor that maintains energy homeostasis. Nat Rev Mol Cell Biol, 13(4), 251-262. https://doi.org/10.1038/nrm3311

Heaney, J. L., Carroll, D., & Phillips, A. C. (2014). Physical activity, life events stress, cortisol, and DHEA: preliminary findings that physical activity may buffer against the negative effects of stress. J Aging Phys Act, 22(4), 465-473. https://doi.org/10.1123/japa.2012-0082

Hittel, D. S., Axelson, M., Sarna, N., Shearer, J., Huffman, K. M., & Kraus, W. E. (2010). Myostatin decreases with aerobic exercise and associates with insulin resistance. Med Sci Sports Exerc, 42(11), 2023-2029. https://doi.org/10.1249/MSS.0b013e3181e0b9a8

Horowitz, J. F. (2003). Fatty acid mobilization from adipose tissue during exercise. Trends Endocrinol Metab, 14(8), 386-392. https://doi.org/10.1016/s1043-2760(03)00143-7

Howley, E. T. (2001). Type of activity: resistance, aerobic and leisure versus occupational physical activity. Medicine & Science in Sports & Exercise, 33(6), S364-S369. doi: https://doi.org/10.1038/nrm3311

Jadon, G. R., Muthukrishnan, J., & Datta, K. (2025). Sports and exercise medicine: Beyond injury management. Medical Journal Armed Forces India, 81(1), 1-6. doi: https://doi.org/10.1016/j.jad.2026.121243.

Jäger, S., Handschin, C., St-Pierre, J., & Spiegelman, B. M. (2007). AMP-activated protein kinase (AMPK) action in skeletal muscle via direct phosphorylation of PGC-1alpha. Proc Natl Acad Sci U S A, 104(29), 12017-12022. https://doi.org/10.1073/pnas.0705070104

Joisten, C., Hirschmüller, A., Bauer, P., Baum, E., Behrens, M., Berrisch-Rahmel, S., . . . DeZeuuw, J. (2025). Sports Preparticipation Evaluation for Healthy Adults: A Consensus-Based German Guideline: C. Joisten et al. Sports Medicine, 1-25.

Kershaw, E. E., & Flier, J. S. (2004). Adipose tissue as an endocrine organ. J Clin Endocrinol Metab, 89(6), 2548-2556. https://doi.org/10.1210/jc.2004-0395

Kusik, D., Tokarz, A., & Kłosowska, J. (2026). Antecedents of workaholism and work engagement: A motivational perspective in research on heavy work involvement. Psychological Reports, 129(1), 661-691. doi: https://doi.org/10.1016/s0140-6736(12)61031-9

La Cava, A., & Matarese, G. (2004). The weight of leptin in immunity. Nat Rev Immunol, 4(5), 371-379. https://doi.org/10.1038/nri1350

Lavie, C. J., Arena, R., & Kaminsky, L. A. (2022). Making the case to measure and improve cardiorespiratory fitness in routine clinical practice. Mayo Clinic Proceedings, doi: https://doi.org/10.1016/S2215-0366(21)00395-3

Lear, S. A., Hu, W., Rangarajan, S., Gasevic, D., Leong, D., Iqbal, R., . . . Kumar, R. (2017). The effect of physical activity on mortality and cardiovascular disease in 130 000 people from 17 high-income, middle-income, and low-income countries: the PURE study. The lancet, 390(10113), 2643-2654. doi: https://doi.org/10.1016/s0140-6736(12)61031-9

Lee, I.-M., Shiroma, E. J., Evenson, K. R., Kamada, M., LaCroix, A. Z., & Buring, J. E. (2018). Accelerometer-measured physical activity and sedentary behavior in relation to all-cause mortality: the women's health study. Circulation, 137(2), 203-205.

Lee, I.-M., Shiroma, E. J., Lobelo, F., Puska, P., Blair, S. N., & Katzmarzyk, P. T. (2012). Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. The lancet, 380(9838), 219-229.

Lee, I. M., Shiroma, E. J., Lobelo, F., Puska, P., Blair, S. N., & Katzmarzyk, P. T. (2012). Effect of physical inactivity on major non-communicable diseases worldwide: an analysis of burden of disease and life expectancy. Lancet, 380(9838), 219-229. https://doi.org/10.1016/s0140-6736(12)61031-9

MacDonald, C., Bennekou, M., Midtgaard, J., Langberg, H., & Lieberman, D. (2025). Why exercise may never be effective medicine: an evolutionary perspective on the efficacy versus effectiveness of exercise in treating type 2 diabetes. British Journal of Sports Medicine, 59(2), 118-125. doi: https://doi.org/10.1016/S2415-0466(21)00395-3

MacInnis, M. J., & Gibala, M. J. (2017). Physiological adaptations to interval training and the role of exercise intensity. The Journal of physiology, 595(9), 2915-2930. doi: https://doi.org/10.1152/japplphysiol.00553.2010

MacIntosh, B. R., Murias, J. M., Keir, D. A., & Weir, J. M. (2021). What is moderate to vigorous exercise intensity? Frontiers in Physiology, 12, 682233. doi: https://doi.org/10.1007/s40279-018-0870-1

Mallett, G. (2025). The effect of exercise and physical activity on skeletal muscle epigenetics and metabolic adaptations. European Journal of Applied Physiology, 125(3), 611-627. doi: https://doi.org/10.1007/s40279-016-0509-9

Mok, A., Khaw, K.-T., Luben, R., Wareham, N., & Brage, S. (2019). Physical activity trajectories and mortality: population based cohort study. Bmj, 365. doi: https://doi.org/10.1016/j.mayocp.2015.08.005

Mok, K. C., Liu, M., & Wang, X. (2025). The physical activity and sedentary behavior among pregnant women in Macao: A cross-sectional study. PLoS One, 20(1), e0318352. doi: https://doi.org/10.1016/j.pcad.2014.10.001

Myers, J., McAuley, P., Lavie, C. J., Despres, J.-P., Arena, R., & Kokkinos, P. (2015). Physical activity and cardiorespiratory fitness as major markers of cardiovascular risk: their independent and interwoven importance to health status. Progress in Cardiovascular Diseases, 57(4), 306-314. doi: https://doi.org/10.1016/S2213-8587(16)30070-4

Otero, M., Lago, R., Lago, F., Casanueva, F. F., Dieguez, C., Gómez-Reino, J. J., & Gualillo, O. (2005). Leptin, from fat to inflammation: old questions and new insights. FEBS Lett, 579(2), 295-301. https://doi.org/10.1016/j.febslet.2004.11.024

Ouchi, N., Parker, J. L., Lugus, J. J., & Walsh, K. (2011). Adipokines in inflammation and metabolic disease. Nat Rev Immunol, 11(2), 85-97. https://doi.org/10.1038/nri2921

Pedersen, B. K., & Febbraio, M. A. (2008). Muscle as an endocrine organ: focus on muscle-derived interleukin-6. Physiol Rev, 88(4), 1379-1406. https://doi.org/10.1152/physrev.90100.2007

Pedersen, B. K., & Saltin, B. (2015). Exercise as medicine–evidence for prescribing exercise as therapy in 26 different chronic diseases. Scandinavian journal of medicine & science in sports, 25, 1-72. doi: https://doi.org/10.1016/j.smhs.2020.02.001

Piercy, K. L., Troiano, R. P., Ballard, R. M., Carlson, S. A., Fulton, J. E., Galuska, D. A., . . . Olson, R. D. (2018). The physical activity guidelines for Americans. Jama, 320(19), 2020-2028. doi: https://doi.org/10.1152/physrev.00031.2006

Pronk, N. P. (2009). Physical activity promotion in business and industry: evidence, context, and recommendations for a national plan. Journal of physical activity and health, 6(s2), S220-S235. doi: https://doi.org/10.1136/bjsports-2016-096587

Radaelli, R., Rech, A., Molinari, T., Markarian, A. M., Petropoulou, M., Granacher, U., . . . Lopez, P. (2025). Effects of resistance training volume on physical function, lean body mass and lower-body muscle hypertrophy and strength in older adults: a systematic review and network meta-analysis of 151 randomised trials. Sports Medicine, 55(1), 167-192. doi: https://doi.org/10.1249/MSS.0000000000001241

Ranasinghe, M. P., Andersen, H., Dempsey, R. K., Wexler, N., Davis, R., Francois, M., . . . Dempsey, P. C. (2025). Contemporary national and international guidelines on physical activity and sedentary behaviour for people living with chronic conditions, disability and advanced age: a scoping review. British Journal of Sports Medicine, 59(6), 385-408. doi: https://doi.org/10.1016/j.smhs.2020.02.001

Richter, E. A., & Hargreaves, M. (2013). Exercise, GLUT4, and skeletal muscle glucose uptake. Physiol Rev, 93(3), 993-1017. https://doi.org/10.1152/physrev.00038.2012

Rivera-Brown, A. M., & Frontera, W. R. (2012). Principles of exercise physiology: responses to acute exercise and long-term adaptations to training. Pm&r, 4(11), 797-804. doi: https://doi.org/10.1007/s00421-011-1864-1

Ross, R., Blair, S. N., Arena, R., Church, T. S., Després, J.-P., Franklin, B. A., . . . Lavie, C. J. (2016). Importance of assessing cardiorespiratory fitness in clinical practice: a case for fitness as a clinical vital sign: a scientific statement from the American Heart Association. Circulation, 134(24), e653-e699. doi: https://doi.org/10.1001/jamaoncol.2023.1830

Sirico, F., Bianco, A., D'Alicandro, G., Castaldo, C., Montagnani, S., Spera, R., . . . Nurzynska, D. (2018). Effects of Physical Exercise on Adiponectin, Leptin, and Inflammatory Markers in Childhood Obesity: Systematic Review and Meta-Analysis. Child Obes, 14(4), 207-217. https://doi.org/10.1089/chi.2017.0269

Stamatakis, E., Ahmadi, M. N., Friedenreich, C. M., Blodgett, J. M., Koster, A., Holtermann, A., . . . Hamer, M. (2023). Vigorous Intermittent Lifestyle Physical Activity and Cancer Incidence Among Nonexercising Adults: The UK Biobank Accelerometry Study. JAMA Oncol, 9(9), 1255-1259. https://doi.org/10.1001/jamaoncol.2023.1830

Steensberg, A., van Hall, G., Osada, T., Sacchetti, M., Saltin, B., & Klarlund Pedersen, B. (2000). Production of interleukin-6 in contracting human skeletal muscles can account for the exercise-induced increase in plasma interleukin-6. J Physiol, 529 Pt 1(Pt 1), 237-242. https://doi.org/10.1111/j.1469-7793.2000.00237.x

Szuhany, K. L., Bugatti, M., & Otto, M. W. (2015). A meta-analytic review of the effects of exercise on brain-derived neurotrophic factor. J Psychiatr Res, 60, 56-64. https://doi.org/10.1016/j.jpsychires.2014.10.003

Tanrıverdi, M., Pehlivan, S., & Törpü, G. C. (2025). Differentiating Physical Activity, Exercise, and Physical Fitness: A Narrative Review on Health and Cardiopulmonary Fitness. doi: https://doi.org/10.1016/j.smhs.2020.02.001

Trayhurn, P., & Wood, I. S. (2004). Adipokines: inflammation and the pleiotropic role of white adipose tissue. Br J Nutr, 92(3), 347-355. https://doi.org/10.1079/bjn20041213

Vos, T., Lim, S. S., Abbafati, C., Abbas, K. M., Abbasi, M., Abbasifard, M., . . . Abdelalim, A. (2020). Global burden of 369 diseases and injuries in 204 countries and territories, 1990–2019: a systematic analysis for the Global Burden of Disease Study 2019. The lancet, 396(10258), 1204-1222. doi: https://doi.org/10.1136/bjsports-2016-096587

Wang, T., Laher, I., & Li, S. (2025). Exercise snacks and physical fitness in sedentary populations. Sports Medicine and Health Science, 7(1), 1-7. doi: https://doi.org/10.1016/j.cmet.2012.08.006

Wen, C. P., Wai, J. P. M., Tsai, M. K., Yang, Y. C., Cheng, T. Y. D., Lee, M.-C., . . . Wu, X. (2011). Minimum amount of physical activity for reduced mortality and extended life expectancy: a prospective cohort study. The lancet, 378(9798), 1244-1253. doi: https://doi.org/10.1007/s00421-011-1864-1

Wu, A.-M., Bisignano, C., James, S. L., Abady, G. G., Abedi, A., Abu-Gharbieh, E., . . . Asaad, M. (2021). Global, regional, and national burden of bone fractures in 204 countries and territories, 1990–2019: a systematic analysis from the Global Burden of Disease Study 2019. The Lancet Healthy Longevity, 2(9), e580-e592. doi: https://doi.org/10.1016/S0140-6736(16)30370-1

Yamauchi, T., Kamon, J., Minokoshi, Y., Ito, Y., Waki, H., Uchida, S., . . . Kadowaki, T. (2002). Adiponectin stimulates glucose utilization and fatty-acid oxidation by activating AMP-activated protein kinase. Nat Med, 8(11), 1288-1295. https://doi.org/10.1038/nm788

Yang, J., Chen, X., Chen, X., & Li, L. (2025). Physical activity and arterial stiffness: A narrative review. The Journal of Clinical Hypertension, 27(1), e14941. doi: https://doi.org/10.1016/S0140-6736(12)61031-9