International Journal of Medical Science and Research
www.medicaljournals.in
Online ISSN: 2664-8768
Received: 01-08-2026, Accepted: 31-08-2026, Published: 15-09-2026
Volume 8, Issue 3, 2026, Page No. 25-35

Integrated workplace health promotion for office workers: Mechanisms and intervention strategies

Lung-Tan Lu
Department of Management, Fo Guang University, Jiaoxi, Yilan, Taiwan
DOI: https://doi.org/10.66856/ijmsr.2026.8.3.8033

Abstract
Background: Office-based workers are chronically exposed to a cluster of overlapping metabolic risk factors, including frequent reliance on out-of-home meals, prolonged sedentary behaviour, circadian misalignment and chronic psychological stress. These exposures act cumulatively through dose–response mechanisms rather than as isolated insults.
Objectives: This narrative review synthesises epidemiological, pathophysiological and randomised controlled trial evidence to examine the dietary, chronobiological, sedentary and stress-related determinants of metabolic health in office-based populations, and to propose an integrated workplace health promotion framework.
Methods: A structured narrative review was undertaken of studies retrieved from PubMed and major nutrition, chronobiology and exercise-physiology journals, prioritising systematic reviews, meta-analyses and landmark randomised controlled trials published up to 2025.
Results: Frequent out-of-home eating is consistently associated with higher energy, fat and sodium intakes, lower micronutrient intakes and adverse anthropometric change. Dietary patterns rich in whole grains, unsaturated fats, vegetables and high-quality protein — exemplified by the Mediterranean and DASH diets — reduce cardiovascular events and blood pressure. Indoor workers show a high prevalence of vitamin D deficiency. Dietary fibre exerts metabolic protection partly via gut-microbiota-derived short-chain fatty acids, with intakes of 25–29 g/day associated with 15–30% lower all-cause mortality. Time-restricted eating without caloric restriction improves weight, blood pressure and glycaemia, although a large recent trial found no benefit of an 8-hour window over calorie restriction alone. Interrupting sitting with brief light activity attenuates postprandial glucose by approximately 37%. Stress-related emotional eating is driven by hypothalamic–pituitary–adrenal axis and reward-circuit mechanisms and is modifiable by mindfulness-based interventions.
Conclusions: Metabolic risk in office workers reflects a closed-loop system of mutually reinforcing exposures. Effective workplace health promotion should adopt an integrated time–dose–quality framework that optimises meal composition, eating timing, sedentary-interruption dose and stress regulation concurrently, rather than targeting single nutrients or behaviours.

Keywords: Metabolic syndrome, eating, sedentary-behaviour interruption, health promotion, circadian rhythm

Introduction

Following the transformation of industrial structures in high- and middle-income economies, office-based work has become one of the largest occupational categories. Prolonged indoor working hours, long commutes and a pervasive culture of overtime have together produced a distinctive lifestyle phenotype that differs markedly from that of preceding generations of manual workers. Public discourse frequently attributes the ill health of office workers to 'excessive occupational stress' or 'lack of exercise', and symptoms such as fatigue, light-headedness, insidious weight gain and daytime somnolence are too often accepted as the unavoidable price of intensive employment. Accumulating evidence from epidemiology, nutritional science, chronobiology and exercise physiology, however, points to a different interpretation: the chronic health crisis among office-based workers arises not from acute illness or psychological strain alone, but from cumulative metabolic injury driven by persistently repeated suboptimal dietary patterns, prolonged sitting and circadian misalignment. A central conceptual implication of this evidence is that health damage is a progressive, dose-dependent process rather than a binary state of 'ill' versus 'well'. Early in the trajectory there are few overt clinical signs; instead, individuals report non-specific complaints such as easy fatigability, peripheral oedema, constipation, post-prandial drowsiness and disturbed sleep. Because these early warning signals are readily attributed to ageing or to the pressures of work, opportunities for early intervention are frequently missed, and medical attention is sought only after a formal diagnosis of metabolic syndrome, hypertension, dyslipidaemia or type 2 diabetes mellitus has been established (Alberti *et al.*, 2009) [2].

National nutrition survey data from Taiwan illustrate the scale of the problem. Comparative analyses of the 1993–1996 and 2005–2008 Nutrition and Health Surveys document substantial shifts in dietary patterns among employed adults, including rising consumption of foods prepared away from home, declining intakes of whole grains and vegetables, and increasing reliance on energy-dense, nutrient-poor convenience items (Pan *et al.*, 2011). At the same time, occupational time-use data indicate that office workers accumulate far more sedentary time than active time on a typical working day, whilst overtime-related late-night meals and nocturnal snacking have become commonplace. These structural living conditions continuously reshape nutrient intakes, hormonal circadian rhythms, gut microbial ecology and stress-response systems, generating self-reinforcing cycles of risk (Lu, 2026). Although individual studies have investigated out-of-home eating, sedentary behaviour, time-restricted feeding and stress-related eating in isolation, relatively few publications have integrated this dispersed evidence into a comprehensive analytical framework specific to office-

based populations. The objectives of the present narrative review are therefore fourfold: first, to synthesise epidemiological datasets, mechanistic pathophysiological studies and randomised controlled trial findings relevant to office workers; second, to analyse systematically the interactions among frequent out-of-home consumption, nutritional imbalance, micronutrient inadequacy, misaligned meal timing, prolonged sitting, stimulant-beverage intake and stress-driven emotional eating, following the logical sequence of risk-exposure characterisation, mechanistic interpretation and evidence-based intervention; third, to develop an integrated workplace health promotion model adapted to East Asian urban contexts in which out-of-home meals predominate; and fourth, to provide a practical reference for clinical nutritional counselling, corporate occupational health programmes and individual self-management.

Methods

This article is a structured narrative review. A targeted literature search was conducted in PubMed and through hand-searching of major journals in nutrition, chronobiology, occupational medicine and exercise physiology. Search terms included combinations of 'eating out of home', 'food away from home', 'office workers', 'sedentary behaviour', 'sitting', 'time-restricted eating', 'intermittent fasting', 'circadian', 'caffeine', 'vitamin D', 'dietary fibre', 'gut microbiota', 'emotional eating', 'stress eating' and 'mindfulness'. Priority was given to systematic reviews and meta-analyses, large prospective cohort studies and landmark randomised controlled trials published up to the end of 2025. Landmark primary trials and mechanistic studies were additionally identified through citation chaining of the retrieved reviews. Where evidence specific to Taiwanese office workers was sparse, findings from demographically comparable East Asian populations were considered, and the uncertainty introduced by this extrapolation is acknowledged in the Discussion. Given the narrative design, no formal risk-of-bias scoring or meta-analytic pooling was performed; effect estimates are reported descriptively as published by the original authors. Data extraction focused on study design, population, exposure or intervention, comparator, outcome and effect estimate, with particular attention to whether findings were derived from office-based or otherwise indoor-working samples.

Out-of-Home Eating: Epidemiology, Nutritional Structure and Dose–Response Risk

1. The Epidemiological Profile of Out-of-Home Eating

Out-of-home eating is defined here as the consumption of main meals purchased from commercial food-service vendors, convenience stores or workplace canteens rather than prepared in a home kitchen. National nutrition and health surveys in Taiwan and large commercial dietary investigations consistently document high frequencies of out-of-home main meals among employed adults, with breakfast, lunch and dinner all substantially outsourced on working days (Pan *et al*., 2011). Because lunch represents a principal source of daily energy and nutrient intake, the meal composition, fat and sodium exposure and dietary fibre intake of most office employees are substantially determined by the external food environment, which severely constrains individual autonomy over main-meal nutrition. The high prevalence of out-of-home dinners reflects structural rather than motivational barriers: lengthy commutes, delayed finishing times associated with overtime, and post-work mental and physical exhaustion all raise the total time cost of ingredient procurement, preparation, cooking and washing up, such that purchasing prepared food becomes a rational behavioural response. 'Late-night snacking substituting for a formal dinner' has emerged as a frequent alternative pattern, further delaying the final eating occasion of the day and encroaching upon the physiological night. It is important to emphasise that frequent out-of-home eating should not be interpreted as individual laziness or indifference to health. It is, in large part, a structural phenomenon generated by the organisation of work. Where overtime becomes normative, the rational allocation of scarce evening time favours purchased meals; nevertheless, this pragmatic selection carries long-term metabolic consequences that individuals rarely internalise at the point of purchase.

2. The Systematic Nutritional Deficiencies of Commercially Prepared Meals

Commercial food-service operations must prioritise palatability, low-cost raw materials, shelf stability and repeat patronage. These commercial imperatives sit in inherent tension with physiological dietary requirements, and the result is that out-of-home meals are typically characterised by excessive fat, elevated sodium, low dietary fibre, abundant refined starch and a high proportion of processed ingredients. The systematic review of Lachat *et al*. (2012) [22], covering 29 studies across multiple countries, concluded that eating out of home was consistently associated with higher total energy intake, a higher energy contribution from fat and lower intakes of several micronutrients, particularly vitamin C, calcium and iron. A complementary systematic review of prospective studies found that out-of-home eating was associated with adverse anthropometric change over time, including weight gain and increased waist circumference (Nago *et al*., 2014) [29]. Excess lipid exposure arises not only from visible surface oil but also from 'invisible' lipids introduced through deep-frying, stir-frying, oil-braising and thick-sauce cooking techniques, which consumers cannot reliably estimate by visual inspection. Sodium exposure is similarly concealed: the overlapping use of table salt, soy sauce, monosodium glutamate and industrial seasoning sauces frequently produces a single meal containing more than half of the recommended daily sodium allowance. Chronic high sodium intake impairs vascular endothelial function, increases renal burden and raises blood pressure, as demonstrated directly in the DASH-Sodium trial (Sacks *et al*., 2001) [36]. Dietary fibre insufficiency is perhaps the most conspicuous shortcoming of commercially prepared meals: plates are dominated by refined white rice and wheat-based staples with meat dishes and token vegetable portions, prolonged cooking degrades fibre structure, and fresh fruit is largely absent. Finally, the growing share of ultra-processed items in purchased meals is itself a risk marker. In a tightly controlled inpatient randomised trial, Hall *et al*. (2019) [14] showed that an ultra-processed diet caused participants to consume approximately 500 kcal per day more than an unprocessed diet matched for presented macronutrients, sugar, sodium and fibre, resulting in weight gain within two weeks. The mechanism — likely a combination of eating

rate, energy density and hyper-palatability — has direct relevance to the convenience-store and bento-box meals on which office workers depend.

3. Dose–Response Relationships Between Meal Frequency and Chronic Disease

The dose–response construct is a core principle of nutritional epidemiology: occasional exposure to an adverse dietary factor can be buffered by metabolic compensatory systems, whereas risk rises, often in a supralinear fashion, as exposure frequency and cumulative duration increase. A single nutritionally imbalanced restaurant meal can be offset physiologically; when purchased meals constitute the daily default, sustained challenge from combined high-fat, and high-sodium and low-fibre exposures drives progressive pathological change. Clinically, this trajectory advances insidiously from early fluid retention, constipation and fatigue towards dyslipidaemia, blood-pressure lability, exaggerated postprandial glycaemic excursions and, ultimately, metabolic syndrome and its sequelae. This natural history is notably 'silent': substantial organ and metabolic damage accumulates before symptomatic onset, and early manifestations are commonly misattributed to occupational stress or ageing (Alberti *et al*., 2009) [2].

Evidence for a dose–response association between eating away from home and cardiometabolic outcomes is now well established. In an analysis of the 2009 Chinese Health and Nutrition Survey, Wang *et al.* (2019) [42] showed that adults who ate away from home frequently had significantly higher odds of a metabolic syndrome diagnosis (odds ratios of approximately 1.48 and 1.68 for intermediate and high frequencies relative to the referent), with the association concentrated in middle-aged men and linked to higher triglycerides, abdominal adiposity, elevated blood pressure and impaired fasting glucose. In the Korean National Health and Nutrition Examination Survey, eating alone — a pattern closely correlated with purchased, convenience-oriented meals — was likewise associated with metabolic syndrome and its components (Kwon *et al*., 2018). These findings are directly relevant to Taiwanese office workers, whose workplace culture, overtime patterns and food-consumption behaviours closely parallel those of their East Asian neighbours. Critically, this evidence does not imply that out-of-home eating is inevitably harmful. The relevant determinants are exposure frequency, meal quality and the co-presence of additional risk factors. Even among daily out-of-home consumers, risk can be materially attenuated by consistently selecting meals rich in vegetables, adequate in high-quality protein and low in deep-fried items and heavy sauces. This principle underpins the practical selection strategies developed in Section 10.

An adequate analysis must also recognise that workplace eating is embedded in social practice. Business entertainment meals, celebratory feasts and peer-pressure-driven snacking operate through mechanisms distinct from individual preference, typically selecting for larger portions, higher alcohol intake and more extreme sodium and fat loads than solitary eating. The Taiwanese and broader East Asian custom of communal dishes shared from common plates further complicates portion estimation. Workplace intervention design should therefore attend to group-level norms — team-lunch defaults, meeting catering, the location of the office relative to the quality of the surrounding food environment — rather than restricting itself to individual education, since the strongest levers on purchased-meal exposure are environmental and organisational rather than informational.

Macronutrient Quality and Evidence-Based Dietary Patterns

Carbohydrate, protein and fat together supply the overwhelming majority of dietary energy. Public discussion frequently falls into binary frameworks that label particular nutrients as intrinsically harmful; contemporary nutritional science, by contrast, emphasises that the quality, source and proportional balance of macronutrients, rather than their absolute weight, are the primary determinants of health outcomes. Reference ranges such as the Acceptable Macronutrient Distribution Ranges — approximately 45–65% of energy from carbohydrate, 10–35% from protein and 20–35% from fat — provide a physiological scaffold, but the composition within each category matters far more than the total. For office workers, the characteristic difficulty is not gross overconsumption of carbohydrate or fat per se, but structural imbalance: excessive refined carbohydrate, surplus low-quality fat, insufficient high-quality protein and inadequate intake of beneficial fatty acids.

1. Carbohydrate Quality: The Complementary Roles of Glycaemic Index and Glycaemic Load

Carbohydrate evaluation must separate 'how much' from 'what kind'. The glycaemic index (GI) quantifies the velocity of the blood glucose response to a food relative to a reference, whereas the glycaemic load (GL) additionally incorporates the actually consumed portion. GI therefore describes an intrinsic property of the food, whilst GL approximates the real-world glycaemic burden of an eaten quantity; joint assessment of both indicators permits a complete appraisal of carbohydrate-related metabolic impact. Whole-grain products such as brown rice, oats and mixed-grain staples retain their bran and germ, supplying dietary fibre, resistant starch and micronutrients, and generally occupy the low-GI, low-GL categories. Refined staples — white rice, white bread, wheat noodles — have undergone processing that removes much of their fibre structure, are digested rapidly and produce sharp postprandial glycaemic elevations, characteristic of high-GI, high-GL foods. The systematic review and meta-analysis of Reynolds *et al.* (2019) [35] demonstrated that higher intakes of fibre and whole grains are associated with significant reductions in all-cause and cardiovascular mortality, incident type 2 diabetes and colorectal cancer. For sedentary office workers with low physical activity levels, partial substitution of refined staples with whole-grain alternatives improves postprandial glycaemic stability and reduces insulin demand, an effect that is achievable without formal caloric restriction. An important clinical caveat is that a low GI classification does not license unlimited consumption: certain low-GI foods are energy-dense, and overconsumption still generates an energy surplus; the GL metric explicitly captures this limitation.

2. Protein quality and the musculoskeletal transition of mid-working life

Human protein synthesis requires twenty amino acids, nine of which are essential and must be obtained from the diet. Animal-source foods (meat, eggs, dairy, fish and shellfish)

and soy-derived foods provide complete essential amino-acid profiles and are classified as high-quality protein sources, whereas most isolated plant proteins are limiting in one or more essential amino acids and require diverse complementary combinations. Quality metrics such as the Protein Digestibility-Corrected Amino Acid Score (PDCAAS) and the Digestible Indispensable Amino Acid Score (DIAAS) quantify intestinal absorption and utilisation efficiency; higher scores correspond to superior bioavailability and lower metabolic waste burdens. This distinction has practical occupational relevance because office-based workers undergo a key physiological transition from approximately the fourth decade of life: skeletal muscle mass declines progressively with age, accompanied by a fall in basal metabolic rate. Insufficient protein intake accelerates this attrition, reducing basal energy expenditure, impairing immune function and promoting adiposity — the substrate of the familiar mid-career triad of weight gain, subjective fatigability and declining physical capacity. Current evidence supports moderately higher protein intakes (of the order of 1.0–1.2 g/kg/day, with an emphasis on high-quality sources and per-meal distribution) for the preservation of muscle mass in sedentary middle-aged adults (Paddon-Jones & Rasmussen, 2009) [33]. A frequent pitfall in the out-of-home context is the bento-box configuration of an oversized starch portion with a token protein serving, producing a high-energy meal that is nonetheless relatively protein-inadequate.

The health effects of dietary fat are determined by fatty-acid class rather than by total fat energy alone. Excess saturated fat intake raises low-density lipoprotein cholesterol and cardiovascular risk, whereas monounsaturated fatty acids favourably modify inflammatory tone, lipid profiles and vascular endothelial function, rendering them suitable for long-term consumption. Among polyunsaturated fatty acids, the omega-3 (n-3) and omega-6 (n-6) families are dietary essentials that humans cannot synthesise de novo; the physiological balance between them is at least as important as the intake of either alone. Commercial catering in East Asia relies heavily on soybean, palm and repeatedly heated frying oils rich in n-6 fatty acids, whilst dietary sources of long-chain n-3 fatty acids — oily marine fish, linseed, walnuts — are under-represented in routine meals. Because n-6 substrates predominantly generate pro-inflammatory eicosanoid signalling whereas n-3 substrates generate anti-inflammatory mediators, a chronically imbalanced n-6: n-3 ratio sustains low-grade inflammation, a key pathological substrate of insulin resistance and atherosclerosis.

3. The Predimed Trial: Cardiovascular Protection From A Mediterranean Dietary Pattern

The strongest randomised evidence that dietary fat quality — rather than fat quantity — drives cardiovascular outcomes derives from the PREDIMED (Prevención con Dieta Mediterránea) trial. This multicentre trial enrolled 7,447 community-dwelling adults at elevated cardiovascular risk and randomised them to a Mediterranean diet supplemented with extra-virgin olive oil, a Mediterranean diet supplemented with mixed nuts, or a low-fat control diet. After a median follow-up of 4.8 years, the primary major cardiovascular event endpoint was reduced by approximately 30% in both Mediterranean arms relative to control (hazard ratios 0.69 and 0.72, respectively) (Estruch *et al*., 2018) [13]. The principal lessons are threefold. First, dietary fat per se is not harmful; adverse outcomes arise from the combination of excessive low-quality fat with surplus refined carbohydrate. Second, modifying the fatty-acid profile towards monounsaturated and polyunsaturated sources can reduce hard clinical endpoints without extreme fat restriction. Third, the protective pattern is a whole-diet construct — abundant vegetables, legumes, whole grains, fish, nuts and olive oil — rather than any single 'superfood'.

A corresponding body of evidence supports the DASH (Dietary Approaches to Stop Hypertension) pattern: in the original DASH trial, the intervention diet reduced systolic blood pressure by 11.4 mmHg and diastolic pressure by 5.5 mmHg in participants with pre-hypertension or mild hypertension (Appel *et al*., 1997) [3], and the DASH-Sodium factorial trial demonstrated additive, dose-dependent blood-pressure reductions from sodium restriction (Sacks *et al*., 2001) [36]. The practical problem for East Asian office workers is that both patterns were designed around Mediterranean and North American food systems; unmodified transplantation into Taiwanese lunch-box and convenience-store environments is unrealistic, necessitating the locally adapted implementations discussed in Section 10.

For East Asian office workers, the dominant carbohydrate vehicle is steamed white rice, frequently consumed in portions exceeding 200 g per meal. The evidence reviewed above suggests a three-step pragmatic modification that preserves culinary acceptability: first, blend white rice with brown, red or mixed grains in a 1:1 to 2:1 ratio, which lowers the glycaemic excursion whilst retaining palatability; second, reduce the absolute staple portion by one-quarter to one-third where weight management is a goal, replacing the displaced volume with vegetables; third, exploit resistant-starch formation by allowing cooked rice to cool and reheating it, a household-accessible manoeuvre that measurably increases resistant starch content and blunts glycaemic response. Sweet potato, taro and maize provide culturally familiar whole-food alternatives that can substitute for part of the rice portion without requiring behavioural novelty. These modifications require no change of venue, cost or cuisine, and therefore represent high-adherence interventions in the out-of-home context.

Micronutrient Inadequacy and Occupational Morbidity

Relative to energy-yielding macronutrients, vitamins and minerals are required in minute absolute quantities, yet they participate in every major axis of metabolism: hormone synthesis, mitochondrial energy production, immune surveillance, bone remodelling and neuronal signal transduction. Latent micronutrient deficiency is highly prevalent among office-based workers, but its presentation has changed. The florid deficiency diseases of the past — rickets, scurvy, beriberi — are now rare in industrialised settings; in their place, subclinical inadequacy produces non-specific occupational complaints: chronic fatigue, musculoskeletal discomfort, affective lability, poor concentration and impaired sleep quality. Because these symptoms overlap almost perfectly with the phenomenology of occupational stress, their nutritional contribution is systematically under-recognised.

1. Vitamin D Deficiency: A High-Priority Problem for Indoor Workers

Vitamin D functions physiologically as a secosteroid hormone, participating in bone mineralisation, skeletal muscle performance, immune-cell regulation, cellular

differentiation and central nervous system mood regulation (Holick, 2007) [17]. Office-based workers constitute a high-risk population for deficiency for a straightforward environmental reason: cutaneous synthesis following solar ultraviolet-B exposure accounts for the majority of the body's supply, and extended indoor daytime work — frequently compounded by early-morning departure, late-evening return, sun-protective clothing and sun avoidance — suppresses it almost completely. Dietary sources (oily fish, egg yolks, fortified dairy) are simultaneously under-supplied in typical purchased meals.

The occupational epidemiology is now robust. In their systematic review of vitamin D status across occupations, Sowah *et al.* (2017) [37] found that the pooled mean serum 25-hydroxyvitamin D concentration of indoor workers was approximately 40.6 nmol/L, markedly lower than the 66.7 nmol/L observed in outdoor workers, with more than three-quarters of indoor workers falling below 50 nmol/L (20 ng/mL). Analysing data from the fifth Korea National Health and Nutrition Examination Survey, Jeong *et al.* (2014) [29] reported that the prevalence of vitamin D deficiency among Korean office workers reached 88%, exceeding that of manufacturing workers. Ward *et al.* (2011), in a cross-sectional analysis of the 1958 [43] British birth cohort, demonstrated that working patterns were significantly associated with vitamin D status in mid-life, with night workers showing measurably lower concentrations. Mizoue *et al.* (2015) [27] further showed, in 1,786 Japanese employees, that low serum 25-hydroxyvitamin D concentrations were associated with an increased likelihood of depressive symptoms, and that low concentrations clustered among those engaged in shiftwork, overtime work and short sleep. The functional consequences for office workers operate through three principal pathways. The first is musculoskeletal: vitamin D inadequacy alters muscular tone and bone metabolism and can amplify the cervical, shoulder and lumbar discomfort that is conventionally attributed solely to poor sitting posture. The second is immune: deficiency is associated with increased susceptibility to upper respiratory infection and slower tissue repair. The third is neuropsychiatric: persistent subjective fatigue, reduced work motivation and depressive symptomatology all correlate with low 25-hydroxyvitamin D concentrations in working populations (Mizoue *et al.*, 2015) [27]. It is worth noting, however, that the causal direction of the fatigue association remains incompletely resolved, and supplementation trials have not consistently demonstrated benefits on mood or fatigue endpoints; screening and correction of deficiency should therefore be regarded as prudent occupational hygiene rather than a panacea.

2. B-Complex Vitamins And Homocysteine Metabolism

The B-complex vitamins serve as enzymatic cofactors in the energy-yielding pathways that convert carbohydrate, protein and fat into usable substrate, and they stabilise peripheral and central nervous system function. Thiamine (vitamin B1) is central to carbohydrate metabolism, and chronic high intakes of refined starch and added sugar accelerate thiamine utilisation; inadequate supply manifests as post-prandial drowsiness and mental lassitude. Pyridoxine (vitamin B6) participates in amino-acid metabolism and neurotransmitter synthesis, and psychosocial stress and night work increase bodily requirements. Cobalamin (vitamin B12) and folate jointly support haematopoiesis, myelin maintenance and homocysteine clearance; combined insufficiency elevates circulating homocysteine, a risk factor for endothelial dysfunction and vascular disease, and is associated with the subjective 'brain fog', memory impairment and psychomotor slowing familiar to office workers. Purchased-meal patterns rich in refined staples and poor in whole grains and dark-green vegetables reduce B-vitamin intakes, whilst occupational stress and late working hours accelerate consumption — a double deficit that renders subclinical B-vitamin insufficiency widespread among working populations.

Iron deficiency displays a pronounced sex-specific pattern. Women of reproductive age experience cyclical menstrual losses, and out-of-home meal patterns low in red meat and dark-green vegetables predispose to iron-deficiency anaemia or latent iron deficiency, presenting as fatigue, light-headedness, breathlessness and reduced exercise tolerance. Men, although spared menstrual losses, are not immune: chronic stress, sedentary metabolism and low meat intake can produce latent insufficiency, and ferritin screening is underused in both sexes. Magnesium and potassium are minerals critical to stress physiology and sleep. Magnesium participates in muscular relaxation and inhibitory neuronal signalling; chronic psychological stress increases renal magnesium excretion, and purchased diets deficient in whole grains, nuts and leafy greens correlate with the clinical constellation of physical exhaustion coexisting with mental hyperarousal and prolonged sleep-onset latency. Potassium modulates cortisol regulation and vascular tone; inadequate intake, in the setting of high sodium exposure typical of restaurant food, raises blood-pressure lability and blunts stress resilience. Iodine, the obligate substrate for thyroid-hormone synthesis, determines basal metabolic rate, thermoregulation and energy expenditure; where seafood intake is limited and consumption of iodised salt irregular, iodine inadequacy may present as unexplained persistent fatigue, reduced basal metabolic rate and difficulty with weight management. The common thread across all four minerals is that their deficiency states masquerade as the ordinary complaints of overwork, and are therefore systematically undertreated.

For occupational health services, three practical conclusions follow. First, unexplained persistent fatigue, myalgia and low mood in sedentary indoor workers warrant a basic biochemical screen — complete blood count, ferritin, 25-hydroxyvitamin D, thyroid-stimulating hormone and vitamin B12 — before attribution to psychological causes alone, since the presenting syndromes are non-specific and the tests are inexpensive relative to the cost of untreated deficiency. Second, dietary correction should precede supplementation where feasible: oily fish twice weekly, eggs, dairy or fortified soya milk, dark-green vegetables and legumes address several gaps simultaneously. Third, workplace design interventions — encouraging brief outdoor breaks during daylight hours, situating canteens to include vitamin D- and iron-rich options — attack the exposure at its source rather than relying on individual compliance with supplementation regimens.

Gut Microbiota, Dietary Fibre and Systemic Metabolic Regulation

1. The Gut Microbiome as a Metabolic Organ

The human gut microbiome — an ecosystem of approximately 10^{14} microorganisms — participates directly in digestion and absorption and exerts distal regulatory

influence over hepatic, muscular and cerebral metabolism through microbial metabolites (principally the short-chain fatty acids acetate, propionate and butyrate), maintenance of intestinal-barrier integrity, and immune modulation along the gut–liver and gut–brain axes (Koh *et al*., 2016; Makki *et al*., 2018) [20, 26]. Diet is the single most powerful modifiable determinant of gut microbial composition, and the purchased-meal dietary pattern of office workers — high in fat and refined carbohydrate, low in fibre and fermented foods — rapidly and reproducibly remodels the microbiome in an adverse direction. Observational studies of obesity first identified systematic differences in gut microbial ecology between lean and obese individuals, including an altered ratio between the dominant phyla Firmicutes and Bacteroidetes (Ley *et al*., 2006) [23]; subsequent work has confirmed that high-fat, high-sugar, low-fibre diets favour energy-harvesting microbial configurations that promote greater caloric extraction from otherwise identical food, reinforce visceral adiposity and heighten insulin resistance — a self-reinforcing cycle in which a degraded diet worsens the microbiome, and the worsened microbiome amplifies the metabolic damage of the diet.

Dietary fibre is not a single substance but a heterogeneous group of plant carbohydrates resistant to human digestive enzymes, conventionally divided into soluble and insoluble fractions with complementary physiological roles; health requires adequate quantities of both. Soluble fibres (pectins, beta-glucans, gums) form viscous gels in the intestinal lumen that slow gastric emptying and carbohydrate absorption, flatten postprandial glycaemic excursions, reduce cholesterol reabsorption and serve as fermentable substrates for beneficial commensals. Insoluble fibres (cellulose, lignin, resistant starch fractions) are water-insoluble, increase faecal bulk, stimulate peristalsis and shorten intestinal transit time. A useful clinical heuristic is that soluble fibre principally mediates metabolic regulation and microbial nourishment, whereas insoluble fibre principally supports bowel motility and luminal clearance; the purchased-meal pattern is simultaneously deficient in both, which goes far to explain the constipation and metabolic dysregulation so prevalent among office workers.

2. The Quantitative Evidence Base for Fibre

The magnitude of the protective association was quantified in the landmark 2019 [35] Lancet series: Reynolds *et al*. (2019) [35] synthesised 185 prospective cohort studies and 58 clinical trials encompassing some 135 million person-years of observational data and showed that intakes of 25–29 g of dietary fibre per day were associated with a 15–30% reduction in all-cause and cardiovascular mortality and in the incidence of type 2 diabetes and colorectal cancer, with dose–response curves suggesting further benefit at higher intakes and with similar protective gradients for whole-grain versus refined-grain consumption. Two practical conclusions follow. First, increasing fibre intake and substituting whole grains for refined cereals constitute one of the most cost-effective, robustly evidence-supported nutritional interventions available, with benefits that extend beyond any single mechanism. Second, the gap between evidence and practice is enormous: typical dietary surveys indicate that East Asian urban adults consuming predominantly purchased meals achieve only 12–15 g of fibre per day, roughly half of the recommended 25–30 g (Pan *et al*., 2011). Closing even half of this gap — through deliberate vegetable selection, whole-grain substitution and legume inclusion — would be expected to produce measurable metabolic benefit, and this feasibility argument underlies the food-selection protocols in Section 10. It should finally be noted that the microbiome literature, whilst mechanistically compelling, remains associative in much of its human evidence: interventional trials of defined probiotic or prebiotic strategies in office-worker populations are few, small and heterogeneous, and commercial probiotic products vary enormously in strain viability and efficacy. The most defensible recommendation at present remains the dietary one — diverse, fibre-rich, minimally processed plant foods — rather than reliance on supplements. Operationally, the 25–30 g daily target can be decomposed into per-meal quotas that translate directly into purchasing decisions: a lunch providing at least 10 g of fibre requires, as a minimum, a double vegetable serving (approximately 150–200 g cooked vegetables), a whole-grain or legume component, and a fruit. Convenience-store salads, edamame, mixed-grain onigiri, roasted sweet potato and unsalted nuts are all portable, shelf-stable options that can be combined to reach this threshold without cooking facilities. Nutrition labels listing dietary fibre per serving (Section 10.4) allow rapid verification. This '10 g per main meal' heuristic converts an abstract population guideline into a single actionable number.

Chrononutrition: Meal Timing, Time-Restricted Eating and Caffeine

1. Meal Timing as an Independent Metabolic Variable

Conventional dietary guidance concentrates on 'what to eat' and 'how much'. Chrononutrition research has established that 'when to eat' is an independent metabolic variable separable from energy quantity. The endogenous human circadian system, synchronised to the light–dark cycle by the suprachiasmatic nucleus and by peripheral clocks in liver, adipose tissue, skeletal muscle and gut, programmes hormonal secretion, insulin sensitivity and cellular energy metabolism to oscillate over 24 hours. Cortisol concentrations are high by day and fall overnight; melatonin rises nocturnally to support sleep; insulin sensitivity peaks during daylight hours and declines during the biological night. Consequently, identical meals and identical energy loads produce divergent metabolic outcomes depending on when they are consumed. Morris *et al*. (2015) [28] demonstrated experimentally that circadian misalignment itself impairs glucose tolerance via mechanisms separable from those of the central circadian system, and that the biological evening is intrinsically a state of reduced glucose tolerance. Night-time eating suppresses melatonin secretion, elevates nocturnal cortisol and fragments sleep architecture; nocturnally consumed meals are preferentially directed towards lipogenic storage pathways. These physiological principles provide the mechanistic foundation for regarding overtime-associated late-night snacking as a circadian-disruption behaviour with metabolic consequences beyond its caloric content.

2. Time-Restricted Eating: The Clinical Evidence

Time-restricted eating (TRE) operationalises chrononutrition by confining the daily eating window without necessarily altering food composition. In a 12-week interventional trial of 19 adults with metabolic syndrome, Wilkinson *et al*. (2020) [44] asked participants to consume all food within a self-selected 10-hour window whilst otherwise

maintaining habitual diet and activity; participants lost a mean of 3.3 kg of weight, reduced systolic blood pressure by approximately 7 mmHg and fasting glucose by approximately 4 mmol/L (about 8 mg/dL), and reported improved sleep quality and reduced daytime fatigue. The trial was small and uncontrolled, but it delivered an important proof of concept: meaningful metabolic improvement can follow from modifying only the temporal boundaries of eating. Subsequent evidence has been more sobering. In the TREATY trial, Liu *et al.* (2022) [24] randomised 139 adults with obesity to an 8-hour daily eating window with or without calorie restriction, or to daily calorie restriction alone, and found that the time restriction produced no significant benefit in weight or metabolic risk factors beyond those achieved by calorie restriction alone. Narrative and clinical reviews have likewise emphasised that adherence to narrow eating windows is difficult to sustain in real-world working conditions, and that the early promising signals derived largely from small, short, uncontrolled studies (Varady *et al.*, 2022; O'Neal *et al.*, 2022) [30, 41]. The defensible synthesis is pragmatic: office workers need not adhere rigidly to a 10-hour window, but progressively compressing an eating day that currently stretches from early morning until late-night snacking towards a window that ends in the early evening is a low-burden, physiologically rational target.

3. Caffeine as a Direct Circadian Disruptor

Caffeine has a physiological half-life of approximately 3–7 hours, meaning that an afternoon caffeinated beverage maintains substantial circulating concentrations at habitual sleep onset. A double-blind crossover study demonstrated that 200 mg of caffeine administered six hours before bedtime reduced objectively measured total sleep time by more than half an hour and diminished deep-sleep proportion (Drake *et al.*, 2013). Burke *et al.* (2015) [6, 10] went further, showing in a laboratory protocol that evening caffeine administration delays the phase of the human central circadian clock *in vivo* — that is, caffeine is not merely soporific in its effects but a true zeitgeber-shifting agent. The practical corollary for office workers is to minimise caffeinated-beverage consumption after the early afternoon (approximately 14:00 for a typical 23:00 bedtime). Many office workers consume an afternoon coffee precisely to counteract daytime drowsiness, without recognising that the resultant nocturnal sleep impairment degrades next-day alertness and appetite regulation, completing a self-sustaining cycle in which the remedy perpetuates the problem. For workers whose schedules preclude early dinner, graded targets are preferable to abstinence-based rules. A three-tier hierarchy is proposed: (i) the final large meal should, wherever possible, be completed at least three hours before habitual sleep onset; (ii) where overtime delays dinner, the delayed meal should be downsized and simplified — favouring protein and vegetables over starch and fat — because the nocturnal metabolic state disposes towards storage; (iii) where nocturnal eating is unavoidable (night shifts), the post-midnight intake should be treated as the 'biological night' and kept minimal, with the bulk of energy relocated to the pre-shift and mid-shift windows. Combined with the caffeine-timing rule above, these tiered rules convert chrononutrition evidence into behaviour that remains feasible under genuine occupational constraint.

Sedentary Behaviour and the Metabolic Case for Interrupting Sitting

1. Prolonged Sitting as an Independent Risk Factor

Sedentary behaviour denotes waking activity in a seated or reclined posture with an energy expenditure at or below 1.5 metabolic equivalents. Office workers characteristically accumulate 8–10 hours of sedentary time per working day, and the total is higher still when commuting is included. A critical conceptual advance of the past two decades has been the recognition that sedentary-related metabolic damage is an independent risk factor that cannot be fully offset by occasional structured exercise. Hamilton *et al.* (2007) [15] established the molecular mechanisms: skeletal-muscle lipoprotein lipase activity — the rate-limiting enzyme for triglyceride clearance — is powerfully suppressed by inactivity itself, independent of the exercise stimulus. Owen *et al.* (2010) [32] articulated the population-health framework, arguing that 'too much sitting' and 'too little exercise' are distinct exposures requiring distinct interventions. Large meta-analytic evidence confirms the clinical stakes: Biswas *et al.* (2015) [4] found that prolonged sedentary time is independently associated with increased all-cause mortality, cardiovascular disease incidence and type 2 diabetes, and that this association persists after statistical adjustment for habitual physical activity. An evening gym session, in other words, does not neutralise a day of uninterrupted sitting; the two exposures operate through partially distinct pathways. The World Health Organization's 2020 [5] guidelines explicitly address both, recommending not only 150–300 minutes of moderate-intensity activity weekly but also the reduction and interruption of sedentary time as an independent objective (Bull *et al.*, 2020) [5].

2. The Acute Physiology of Uninterrupted Sitting

The acute metabolic consequences of sitting unfold on a timescale of minutes. After approximately 30–60 minutes of continuous sitting, skeletal-muscle contractile activity in the large postural muscles falls sharply, lower-limb blood-flow velocity declines, muscle-cell insulin sensitivity deteriorates and lipolytic enzyme activity is suppressed; the organism enters a transient energy-conserving metabolic mode. Postprandially, these changes interact with the glucose load of the preceding meal: in the absence of muscular contraction-mediated glucose disposal, postprandial glycaemic excursions are exaggerated and prolonged. Healy *et al.* (2008) [16] provided early observational evidence that the frequency of breaks in sedentary time, independently of total sedentary time and moderate-to-vigorous exercise, was favourably associated with metabolic risk markers including waist circumference, triglycerides and 2-hour glucose.

3. Experimental Evidence for Sedentary Interruption

The causal inference was established by experimental studies. Dunstan *et al.* (2012) [11] randomised overweight adults to three conditions during an eight-hour laboratory day: uninterrupted sitting; or sitting interrupted by two-minute light-intensity walking bouts every 20 minutes; or by resistance-activity bouts. The interruption conditions substantially reduced postprandial glucose (by approximately 24%) and insulin (by approximately 23%) responses relative to uninterrupted sitting. In a subsequent office-simulation study, Dempsey *et al.* (2016) [9] showed that interrupting sitting with three minutes of light walking every 30 minutes reduced postprandial glucose excursions

by a striking 37% and improved insulin responses by approximately 17%, with similar though slightly smaller effects for simple resistance activities; a standing-only condition attenuated glycaemia by only about 9%, indicating that posture change alone is insufficient and that muscular contraction is the active ingredient. Three practical corollaries follow. First, formal exercise is unnecessary for this purpose: brief, light ambulation is effective. Second, interruption frequency matters more than interruption intensity. Third, sit–stand desks, whilst valuable for musculoskeletal comfort, are not a metabolic substitute for actual movement; workplace recommendations should prioritise brief ambulation over standing in isolation. An implementable golden rule for office settings is therefore to stand and move lightly for 2–3 minutes every 30 minutes of sitting — a regimen that is achievable within the practical constraints of knowledge work and that, accumulated across a working day, amounts to a meaningful dose of non-exercise activity. Translating the interruption evidence into organisational practice requires changes at the level of defaults rather than exhortation: calendar systems that insert five-minute movement blocks between meetings; printer, water dispenser and waste-bin placement at a distance from desks; structured walking breaks at 90-minute intervals in team schedules; and the normalisation of 'walking meetings' for one-to-one discussions. Because adherence to self-monitored regimens decays rapidly (O'Neal *et al*., 2022) [30], environmental redesign and team norms are likely to sustain the interruption dose more reliably than individual reminders alone — an application of the same structural logic that renders out-of-home eating the default in the first place.

Occupational Stress, Emotional Eating and Mindfulness-Based Regulation

1. The Neuroendocrinology of Stress-Induced Eating

Chronic occupational stress does not merely produce psychological distress; through hypothalamic–pituitary–adrenal (HPA) axis signalling and mesolimbic reward circuitry, it systematically remodels appetite and food preference, generating the behavioural pattern termed emotional eating. Acute psychosocial stress activates the HPA axis and elevates circulating cortisol, an adaptive response in the short term. Under chronic stress, persistently elevated cortisol produces three metabolic sequelae: it amplifies central craving for energy-dense, high-sugar and high-fat foods; it promotes visceral adipose deposition; and it reduces peripheral insulin sensitivity (Torres & Nowson, 2007; Adam & Epel, 2007) [1, 40]. Ingestion of sweet or fatty foods transiently stimulates striatal dopamine release, producing subjectively experienced relief; with repeated exposure, an automatic associative circuit consolidates: stress triggers craving, craving triggers consumption, consumption triggers reward, and the reward negatively reinforces the entire loop. Laboratory evidence supports the mechanism: Epel *et al*. (2001) [12] showed that women who were high cortisol responders to a standardised stressor subsequently consumed significantly more sweet, high-fat snack foods. Neuroimaging evidence demonstrates that stress and food-cue exposure jointly potentiate reward-region activation in obesity, with stress-related craving correlating with metabolic risk markers (Jastreboff *et al*., 2013). Zellner *et al*. (2006) [18, 45] further showed that food selection itself shifts under stress towards sweeter, higher-fat options. Emotional eating is therefore a neurobiologically instantiated behaviour, not a simple failure of willpower, and effective intervention must target the circuit rather than exhort the individual.

2. Mindfulness-Based Interventions: Evidence and Mechanisms

Mindfulness-based approaches target precisely the disrupted regulatory architecture described above. By training sustained, non-judgemental attention to internal states, they are hypothesised to strengthen prefrontal cortical control over limbic and striatal reactivity, to improve interoceptive discrimination between physiological hunger and affect-driven craving, and to uncouple the stress–eating association. The proof-of-concept randomised study of Daubenmier *et al*. (2011) [7] assigned 47 overweight and obese women to a mindfulness programme for stress eating or a waitlist control; the intervention improved mindfulness, anxiety and externally driven eating, and within the subgroup with obesity, attenuated the cortisol awakening response and prevented weight gain that occurred in controls, with improvements in mindfulness and stress correlating with reductions in abdominal fat. A subsequent larger randomised clinical trial of a mindfulness-based weight-loss intervention in adults with obesity found significant improvements in fasting glucose and triglyceride handling relative to control (Daubenmier *et al*., 2016) [8]. Complementary trials have shown that mindfulness-based weight-loss programmes outperform conventional controls on eating-behaviour outcomes (Tapper *et al*., 2009) [39]. The evidence base remains modest in size and is subject to blinding constraints inherent to behavioural interventions, but the direction of effect is consistent, and the absence of pharmacological side-effects makes mindfulness training a practical first-line strategy for stress-eating regulation in high-pressure occupational settings. For corporate implementation, the trial evidence suggests several design principles: brevity and repetition outperform occasional intensive retreats (ten-minute daily practice sessions embedded in the working day); training should be scheduled at high-risk craving windows — mid-afternoon and early evening — rather than only at start of day; and programmes should teach applied techniques (urge-surfing, three-minute breathing space before vending-machine or delivery-app use) rather than abstract philosophy. Embedding brief practice into existing meeting rituals — a one-minute settling breath before lunch — offers a low-friction route to population reach.

Practical Food-Selection Strategies for Out-of-Home Meal Environments

The mechanistic and trial evidence summarised above must ultimately be translated into behaviour under real-world constraints: limited time, overtime schedules and pervasive dependence on purchased food. Demanding textbook-ideal home cooking of office workers is neither realistic nor necessary. This section therefore provides implementable selection protocols for the three dominant Taiwanese out-of-home environments — convenience stores, bento-box shops and self-service buffets — together with guidance on label interpretation and local adaptation of evidence-based dietary patterns (OpenAI, 2026).

1. Convenience Stores

Convenience-store meals are typically starch-dominated and fibre-poor, but modular combination can correct much of this. A rice ball paired with unsweetened soya milk and

vegetable sticks upgrades an isolated refined-starch item into a meal with adequate protein and some fibre. A ready-to-eat salad combined with pre-cooked chicken breast and unsalted nuts achieves a high-protein, high-fibre profile with controllable lipid exposure; the dressing should be served separately and used sparingly, since bottled dressings are a concentrated source of invisible fat and sodium. Across all combinations, the governing principles are to add a deliberate protein source, add a vegetable or fruit item, and prefer unsweetened beverages, thereby re-engineering the default starch–sugar configuration into something resembling a balanced meal. For bento-box shops, a four-principle rule set is proposed: halve the staple portion, double the vegetable serving, avoid deep-fried mains, and request sauces separately. Most commercial lunch boxes supply an oversized portion of white rice; asking for half rice directly reduces refined-carbohydrate load without requiring willpower at the point of eating. Boxes containing two or more distinct vegetable preparations should be prioritised. Deep-fried chicken and pork chop options should be excluded in favour of steamed, braised or grilled items. Poured sauces should be packaged separately so that addition is discretionary; this single manoeuvre materially reduces sodium and invisible-fat exposure. These principles operationalise the DASH pattern's low-sodium emphasis and the Mediterranean pattern's vegetable and lean-protein emphasis within a meal format that office workers actually buy.

2. Self-Service Buffets

For self-service settings, the plate-loading sequence — vegetables first, protein sources second, staple carbohydrates last — is recommended, both as a procedural rule and as a compositional target. Loading vegetables onto an empty plate secures their proportional representation before satiety heuristics and visual crowding take over; selecting fish, eggs, tofu and lean meats next anchors protein adequacy; adding starches last naturally constrains their share of the plate. This ordering exploits the psychological architecture of buffet eating, where first-loaded items dominate subsequent choices. Workers purchasing packaged meals should attend to four core label metrics. First, energy per serving: a main-meal lunch should approximate 600–800 kcal for most adults. Second, sodium: a single meal ideally remains below approximately 800 mg, since full-day targets are of the order of 2,000–2,400 mg. Third, added sugar: unsweetened or zero-added-sugar formulations should be the default, particularly for beverages, whose liquid calories evade satiety signalling. Fourth, dietary fibre: a meal providing 10 g or more approaches the high-fibre category and contributes meaningfully towards the 25–30 g daily target (Reynolds *et al*., 2019) [35]. A critical interpretive caveat is the distinction between 'per serving' and 'per package' values: packages frequently contain multiple servings, and misreading package values as single-serving values leads to substantial inadvertent overconsumption of energy and sodium.

Direct transplantation of Mediterranean or DASH menus into Taiwanese food environments fails on ingredient availability, cost and palatability; local adaptation is therefore required. In place of olive oil, daily cooking can emphasise camellia-seed oil and small amounts of linseed oil for n-3 fatty acids, whilst minimising deep-frying and reuse of frying oil. High-quality protein can be anchored in soy foods — tofu, dried tofu curd and unsweetened soya milk — complemented by fish, shellfish, eggs and lean poultry, an approach that is culturally native and economically accessible. Whole-grain staples can substitute brown rice, sweet potato, taro and mixed-grain rice for Western-style oats. DASH's core mechanics — reduced sodium seasoning, restrained use of soy sauce and compound sauces, and abundant vegetables — are directly transferable. The synthesised guiding maxim is: high-quality fats, abundant vegetables, fruit and whole grains, low-sodium seasoning, and locally available high-quality protein. Such a pattern preserves the evidence-based mechanisms of PREDIMED and DASH within foods that Taiwanese office workers can actually purchase at lunchtime.

Conclusions

The chronic health crisis of contemporary office workers is the cumulative product of long-term, superimposed exposures: dependence on out-of-home meals, prolonged sedentary behaviour, circadian misalignment of eating, chronic psychological stress and mistimed stimulant intake (Lu, 2026). Using dose–response relationships as the analytical lens, this review has drawn on epidemiological datasets, mechanistic research and randomised trial evidence to show that metabolic health management for office populations must move beyond simplified, single-nutrient or single-behaviour paradigms. An integrated time–dose–quality framework — concurrently optimising meal composition, eating timing, fragmented physical activity and stress regulation — offers a more defensible and more implementable model. For societies such as Taiwan, in which purchased main meals are the norm, structured selection protocols for convenience stores, bento-box shops and buffets, clear nutrition-label interpretation skills and locally adapted Mediterranean–DASH patterns convert the evidence into daily practice. Future workplace programmes should embed these elements in low-burden, overtime-compatible tools, so that working-age populations can preserve metabolic homeostasis and reduce their population-level risk of metabolic syndrome, cardiovascular disease and type 2 diabetes under the real constraints of modern working life.

References

1. Adam TC, Epel ES. Stress, eating and the reward system. Physiology & Behavior,2007:91(4):449–458. https://doi.org/10.1016/j.physbeh.2007.04.011
2. Alberti KGM M, Eckel RH, Grundy SM, Zimmet PZ, Cleeman JI, Donato KA, *et al*. Harmonizing the metabolic syndrome: A joint interim statement of the International Diabetes Federation Task Force on Epidemiology and Prevention; National Heart, Lung, and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation,2009:120(16):1640–1645. https://doi.org/10.1161/CIRCULATIONAHA.109.192644
3. Appel LJ, Moore TJ, Obarzanek E, Vollmer WM, Svetkey LP, Sacks FM, *et al*. A clinical trial of the effects of dietary patterns on blood pressure. New England Journal of Medicine,1997:336(16):1117–1124. https://doi.org/10.1056/NEJM199704173361601

4. Biswas A, Oh PI, Faulkner GE, Bajaj RR, Silver MA, Mitchell MS, *et al*. Sedentary time and its association with risk for disease incidence, mortality, and hospitalization in adults: A systematic review and meta-analysis. Annals of Internal Medicine,2015:162(2):123–132. https://doi.org/10.7326/M14-1651
5. Bull FC, Al-Ansari SS, Biddle S, Borodulin K, Buman MP, Cardon G, *et al*. World Health Organization 2020 guidelines on physical activity and sedentary behaviour. British Journal of Sports Medicine,2020:54(24):1451–1462. https://doi.org/10.1136/bjsports-2020-102955
6. Burke TM, Markwald RR, McHill AW, Chinoy ED, Snider JA, Bessman SC, *et al*. Effects of caffeine on the human circadian clock *in vivo* and *in vitro*. Science Translational Medicine,2015:7(305):305ra146. https://doi.org/10.1126/scitranslmed.aac5125
7. Daubenmier J, Kristeller J, Hecht FM, Maninger N, Kuwata M, Jhaveri K, *et al*. Mindfulness intervention for stress eating to reduce cortisol and abdominal fat among overweight and obese women: An exploratory randomized controlled study. Journal of Obesity,2011 651936. https://doi.org/10.1155/2011/651936
8. Daubenmier J, Moran PJ, Kristeller J, Acree M, Bacchetti P, Kemeny ME, *et al*. Effects of a mindfulness-based weight loss intervention in adults with obesity: A randomized clinical trial. Obesity,2016:24(4):794–804. https://doi.org/10.1002/oby.21396
9. Dempsey PC, Larsen RN, Sethi P, Sacre JW, Straznicky NE, Cohen ND, *et al*. Benefits for type 2 diabetes of interrupting prolonged sitting with brief bouts of light walking or simple resistance activities. Diabetes Care,2016:39(6):964–972. https://doi.org/10.2337/dc15-2336
10. Drake C, Roehrs T, Shambroom J, Roth T. Caffeine effects on sleep taken 0, 3, or 6 hours before going to bed. Journal of Clinical Sleep Medicine,2013:9(11):1195–1200. https://doi.org/10.5664/jcsm.3170
11. Dunstan DW, Kingwell BA, Larsen R, Healy GN, Cerin E, Hamilton MT, *et al*. Breaking up prolonged sitting reduces postprandial glucose and insulin responses. Diabetes Care,2012:35(5):976–983. https://doi.org/10.2337/dc11-1931
12. Epel E, Lapidus R, McEwen B, Brownell K. Stress may add bite to appetite in women: A laboratory study of stress-induced cortisol and eating behavior. Psychoneuroendocrinology,2001:26(1):37–49. https://doi.org/10.1016/S0306-4530(00)00035-4
13. Estruch R, Ros E, Salas-Salvadó J, Covas M.-I., Corella D, Arós F, *et al*. Primary prevention of cardiovascular disease with a Mediterranean diet supplemented with extra-virgin olive oil or nuts. New England Journal of Medicine,2018:378(25). https://doi.org/10.1056/NEJMoa1800389
14. Hall KD, Ayuketah A, Brychta R, Cai H, Cassimatis T, Chen KY, *et al*. Ultra-processed diets cause excess calorie intake and weight gain: An inpatient randomized controlled trial of ad libitum food intake. Cell Metabolism,2019:30(1):67–77.e3. https://doi.org/10.1016/j.cmet.2019.05.008
15. Hamilton MT, Hamilton DG, Zderic TW. Role of low energy expenditure and sitting in obesity, metabolic syndrome, type 2 diabetes, and cardiovascular disease. Diabetes,2007:56(11):2655–2667. https://doi.org/10.2337/db07-0882
16. Healy GN, Dunstan DW, Salmon J, Cerin E, Shaw JE, Owen N. Breaks in sedentary time: Beneficial associations with metabolic risk. Diabetes Care,2008:31(4):661–666. https://doi.org/10.2337/dc07-2046
17. Holick MF. Vitamin D deficiency. New England Journal of Medicine,2007:357(3):266–281. https://doi.org/10.1056/NEJMra070553
18. Jastreboff AM, Sinha R, Lacadie C, Small DM, Sherwin RS, Potenza MN. Neural correlates of stress- and food cue-induced food craving in obesity: Association with insulin levels. Diabetes Care,2013:36(2):394–402. https://doi.org/10.2337/dc12-1112
19. Jeong H, Hong S, Heo Y, Chun H, Kim D, Park J, *et al*. Vitamin D status and associated occupational factors in Korean wage workers: Data from the 5th Korea National Health and Nutrition Examination Survey (KNHANES 2010–2012). Annals of Occupational and Environmental Medicine,2014:26(1) 28. https://doi.org/10.1186/s40557-014-0028-x
20. Koh A, De Vadder F, Kovatcheva-Datchary P, Bäckhed F. From dietary fiber to host physiology: Short-chain fatty acids as key bacterial metabolites. Cell,2016:165(6):1332–1345. https://doi.org/10.1016/j.cell.2016.05.041
21. Kwon AR, Yoon YS, Min KP, Lee YK, Jeon JH. Eating alone and metabolic syndrome: A population-based Korean National Health and Nutrition Examination Survey 2013–2014. Obesity Research & Clinical Practice,2018:12(2):146–157. https://doi.org/10.1016/j.orcp.2017.09.002
22. Lachat C, Nago E, Verstraeten R, Roberfroid D, Van Camp J, Kolsteren P. Eating out of home and its association with dietary intake: A systematic review of the evidence. Obesity Reviews,2012:13(4):329–346. https://doi.org/10.1111/j.1467-789X.2011.00953.x
23. Ley RE, Turnbaugh PJ, Klein S, Gordon JI. Microbial ecology: Human gut microbes associated with obesity. Nature,2006:444(7122):1022–1023. https://doi.org/10.1038/4441022a
24. Liu D, Huang Y, Huang C, Yang S, Liu S, Lin C, *et al*. Calorie restriction with or without time-restricted eating in weight loss. New England Journal of Medicine,2022:386(16):1495–1504. https://doi.org/10.1056/NEJMoa2114833
25. Lu L. The history of Western holistic health development. AkiNik Publications,2026.
26. Makki K, Deehan EC, Walter J, Bäckhed F. The impact of dietary fiber on gut microbiota in host health and disease. Cell Host & Microbe,2018:23(6):705–715. https://doi.org/10.1016/j.chom.2018.05.012
27. Mizoue T, Kochi T, Akter S, Eguchi M, Kurotani K, Tsukamoto Y, *et al*. Low serum 25-hydroxyvitamin D concentrations are associated with increased likelihood of having depressive symptoms among Japanese workers. Journal of Nutrition,2015:145(3):541–546. https://doi.org/10.3945/jn.114.204115
28. Morris CJ, Yang JN, Garcia JI, Myers S, Bozzi I, Wang C, *et al*. Endogenous circadian system and circadian misalignment impact glucose tolerance via separate mechanisms in humans. Proceedings of the National Academy of Sciences of the United States of America,2015:112(17)–E2234. https://doi.org/10.1073/pnas.1418955112

29. Nago ES, Lachat CK, Dossa RAM, Kolsteren PW. Association of out-of-home eating with anthropometric changes: A systematic review of prospective studies. Critical Reviews in Food Science and Nutrition,2014:54(9):1103–1116. https://doi.org/10.1080/10408398.2011.627095
30. O'Neal MA, Gutierrez NR, Laing KL, Manoogian ENC, Panda S. Barriers to adherence in time-restricted-eating clinical trials: An early preliminary review. Frontiers in Nutrition,2022:9 1075744. https://doi.org/10.3389/fnut.2022.1075744
31. OpenAI. ChatGPT (GPT-5.6) [Large language model]. 2026. https://chatgpt.com/
32. Owen N, Healy GN, Matthews CE, Dunstan DW. Too much sitting: The population health science of sedentary behavior. Exercise and Sport Sciences Reviews,2010:38(3):105–113. https://doi.org/10.1097/JES.0b013e3181e373a2
33. Paddon-Jones D, Rasmussen BB. Dietary protein recommendations and the prevention of sarcopenia. Current Opinion in Clinical Nutrition and Metabolic Care,2009:12(1):86–90. https://doi.org/10.1097/MCO.0b013e32831cef8b
34. Pan W.-H., Wu H.-J., Yeh C.-J., Chuang S.-Y. Diet and health trends in Taiwan: Comparison of two nutrition and health surveys from 1993–1996 and 2005–2008. Asia Pacific Journal of Clinical Nutrition,2011:20(2):238–250.
35. Reynolds A, Mann J, Cummings J, Winter N, Mete E, Te Morenga L. Carbohydrate quality and human health: A series of systematic reviews and meta-analyses. The Lancet,2019:393(10170):434–445. https://doi.org/10.1016/S0140-6736(18)31809-9
36. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, *et al*. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. New England Journal of Medicine,2001:344(1):3–10. https://doi.org/10.1056/NEJM200101043440101
37. Sowah D, Fan X, Dennett L, Hagtvedt R, Straube S. Vitamin D levels and deficiency with different occupations: A systematic review. BMC Public Health,2017:17(1) 519. https://doi.org/10.1186/s12889-017-4436-z
38. Sutton EF, Beyl R, Early KS, Cefalu WT, Ravussin E, Peterson CM. Early time-restricted feeding improves insulin sensitivity, blood pressure, and oxidative stress even without weight loss in men with prediabetes. Cell Metabolism,2018:27(6):1212–1221.e3. https://doi.org/10.1016/j.cmet.2018.04.010
39. Tapper K, Shaw C, Ilsley J, Hill AJ, Bond FW, Moore L. Exploratory randomised controlled trial of a mindfulness-based weight loss intervention for women. Appetite,2009:52(2):396–404. https://doi.org/10.1016/j.appet.2008.11.015
40. Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. Nutrition,2007:23(11–12):887–894. https://doi.org/10.1016/j.nut.2007.08.008
41. Varady KA, Cienfuegos S, Ezpeleta M, Gabel K. Clinical application of intermittent fasting for weight loss: Progress and future directions. Nature Reviews Endocrinology,2022:18(5):309–321. https://doi.org/10.1038/s41574-022-00638-x
42. Wang H, Yu Y, Tian X. Does eating-away-from-home increase the risk of a metabolic syndrome diagnosis? International Journal of Environmental Research and Public Health,2019:16(4) 575. https://doi.org/10.3390/ijerph16040575
43. Ward M, Berry DJ, Power C, Hyppönen E. Working patterns and vitamin D status in mid-life: A cross-sectional study of the 1958 British birth cohort. Occupational and Environmental Medicine,2011:68(12):902–907. https://doi.org/10.1136/oem.2010.063479
44. Wilkinson MJ, Manoogian ENC, Zadourian A, Lo H, Fakhouri S, Shoghi A, *et al*. Ten-hour time-restricted eating reduces weight, blood pressure, and atherogenic lipids in patients with metabolic syndrome. Cell Metabolism,2020:31(1):92–104.e5. https://doi.org/10.1016/j.cmet.2019.11.004
45. Zellner DA, Loaiza S, Gonzalez Z, Pita J, Morales J, Pecora D, *et al*. Food selection changes under stress. Physiology & Behavior,2006:87(4):789–793. https://doi.org/10.1016/j.physbeh.2006.01.029