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Infectobesity and Lifestyle Responsiveness: Infectious Modifiers of Adiposity with Implications for Nutrition and Exercise Science: An Integrated Review

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A B S T R A C T

Obesity is a chronic, relapsing, and multifactorial condition with rising global prevalence and substantial cardiometabolic and functional consequences. Although diet and physical activity remain foundational therapies, large inter-individual variability in weight loss and health responses suggests that biological modifiers influence lifestyle responsiveness. The infectobesity hypothesis proposes that specific pathogens can promote adiposity and remodel metabolic phenotypes through effects on adipogenesis, insulin signaling, inflammation, and host–microbiome interactions. Notably, the earliest empirical roots of infectobesity research emerged from avian models, where infection-related adiposity was first documented in chickens. Six pathogens have been reported to cause obesity in animals. The first was an avian retrovirus, which has been shown to cause stunting, obesity, and hyperlipidemia in chickens. The obesity-promoting effect of Borna disease virus was next demonstrated in rats. Scrapie agents were reported to induce obesity in mice and hamsters. The final two reports were of SMAM-1, an avian adenovirus, and Ad-36, a human adenovirus that caused obesity in animals. Additionally, an association with human obesity is a unique feature of SMAM-1 and Ad-36. Human adenovirus 36 (HAdV-36) is the most studied adipogenic pathogen, supported by mechanistic evidence in cell and animal models and by human sero-epidemiological findings, although there are heterogeneous and method-dependent null results. From a sports- and exercise-science perspective, the infection–obesity interface is clinically relevant because training load, energy availability, and dietary patterns can alter immune competence, susceptibility to infection, recovery, and inflammatory tone—factors that may interact with metabolic adaptation and weight-regain biology. This review synthesizes contemporary evidence on infectobesity, highlights methodological pitfalls (diagnostics, temporality, confounding, and publication bias), and proposes an integrated "infection–lifestyle" framework for precision obesity management in clinical and athletic contexts.

Keywords: *Infectobesity; Avian Adenovirus; SMAM-1; Human Adenovirus 36; Metabolic Inflammation; Exercise; Precision Nutrition; Obesity Management*

1. Introduction

Obesity has emerged as a global health crisis, with increasing prevalence worldwide and significant metabolic and functional consequences. Historically, obesity was predominantly attributed to metabolic and lifestyle factors, including poor diet and physical inactivity (1). Over time, however, research has uncovered the complexity of obesity, revealing its multifactorial origins. In addition to genetic predisposition and environmental influences, emerging evidence suggests that infectious agents may also play a role in promoting obesity.

The first documented evidence that viral infection can lead to increased fat accumulation in birds—particularly intra-abdominal/visceral fat—emerged from the early 1990s work of Nikhil V. Dhurandhar and colleagues. Following a field observation of "unusual fat gain" in chickens infected with an avian adenovirus (SMAM-1), this phenomenon was initially reported in a brief communication (2). Subsequently, in a controlled laboratory study, the same group showed that experimental inoculation with the adenovirus could increase adiposity in chickens; importantly, these changes could not be explained solely by differences in feed intake, and even natural transmission from infected to uninfected groups was associated with a similar fat-gain pattern (3). Later, in an account reflecting the progression of this research line, it was explicitly emphasized that SMAM-1–infected chickens developed a distinct syndrome characterized by "excessive intra-abdominal fat deposition," an observation that became one of the foundational elements for the concept of infectious obesity/adiposity in animal models (4). Subsequent studies identified several other pathogens, including Borna disease virus and Scrapie agents, that were found to induce obesity in rodents (4). This animal pattern was then linked to human studies: in a human sample from Bombay, Dhurandhar and colleagues screened serum for antibodies against SMAM-1 and reported that SMAM-1–positive individuals had a higher BMI but lower serum cholesterol and triglycerides than seronegative individuals; moreover, evidence consistent with antigenemia/viremia was suggested by compatible lesions developing after inoculation of certain human serum samples into chicken embryos. Collectively, these findings extended the hypothesis of infection-related obesity/adiposity from avian models to humans and raised the possibility that SMAM-1—or a serologically similar virus—may contribute to obesity in a subset of individuals (4). The concept of infectobesity—which posits that specific pathogens can contribute to adiposity and metabolic dysfunction, has become an important and increasingly recognized area of research. The most widely studied infectious agent in this context is the 3 human adenoviruses, adenovirus (Ad) 36, Ad-37, and Ad-5, that are associated with obesity also affect adipocytes directly. These viruses stimulate enzymes and transcription factors that cause accumulation of triglycerides and differentiation of preadipocytes into mature adipocytes (5). Human Adenovirus 36 (HAdV-36), which has been associated with increased fat accumulation in both animal models and humans (6). These findings suggest that certain viruses can disrupt metabolic pathways, including adipogenesis, insulin signaling, and inflammation, thereby contributing to obesity. Although the role of infectious agents in obesity remains an area of active investigation, the evidence supports the hypothesis that infectobesity represents a novel and important factor in the development of obesity, alongside traditional metabolic and lifestyle risk factors (7). it could be argued that a growing body of evidence suggesting certain viruses may contribute to obesity in humans supports the possibility that viral infections might account for at least a portion of the global obesity epidemic observed over recent decades. In this context, further human studies, particularly in the post–COVID-19 are essential to clarify the many still-unknown dimensions of infectobesity, and especially virus-related mechanisms and pathways.

2. Methods and Materials

2.1. Review Design

This study was conducted as a narrative integrated review, which is well-suited for synthesizing diverse evidence from mechanistic studies, epidemiological research, and applied literature in nutrition and exercise science. The review focuses on conceptual integration rather than quantitative effect estimation, while drawing on systematic reviews and meta-analyses, where available, to support higher-level inferences.

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2.2. *Data Sources and Search Strategy*

A comprehensive literature search was performed using major electronic databases, including PubMed/Medline, Scopus, Web of Science, and Google Scholar. The search covered publications from ۱۹۹۷ to 2025 and utilized combinations of relevant keywords and MeSH terms such as infectobesity, adenovirus, adipogenic virus, obesity, metabolic inflammation, exercise immunology, nutrition and immunity, weight loss resistance lifestyles, and physical exercise. Reference lists from pertinent reviews and seminal articles were manually screened to identify additional studies.

2.3. *Eligibility Criteria*

Included studies met at least one of the following criteria:

1. Experimental evidence linking infectious agents to adipogenesis or metabolic regulation;
2. Human observational or interventional studies examining associations between infection and obesity-related outcomes;
3. Systematic reviews or meta-analyses on infectobesity or related immunometabolic pathways;
4. Applied studies relevant to nutrition, physical activity, immune function, or metabolic adaptation.

Non-peer-reviewed sources, case reports without mechanistic relevance, and studies lacking clear methodological descriptions were excluded.

2.4. *Data Synthesis and Analysis*

Evidence was synthesized thematically across five domains:

(i) Conceptual foundations of infectobesity,

(ii) Infectious agents implicated in adiposity,

(iii) Mechanistic pathways linking infection to metabolic dysregulation,

(iv) Interactions between infection, diet, and physical activity,

(v) Implications for lifestyle responsiveness and precision obesity management.

Methodological issues—such as diagnostic heterogeneity, temporality, confounding, and publication bias—were critically appraised to contextualize the strength and limitations of the available evidence.

3. Metabolic Resistance to Weight Loss and the Role of Infectobesity

The difficulty in achieving sustained weight loss is a well-known challenge for many individuals. Research has shown that despite significant efforts in diet and physical activity, large inter-individual variability exists in the response to weight loss interventions (8). This variability is partially due to genetic factors, but increasingly, studies suggest that infectobesity, the influence of infectious agents on obesity—may play a crucial role in this resistance. Pathogens such as Human Adenovirus 36 (HAdV-36) and SMAM-1, an avian adenovirus, have been shown to promote adiposity by altering metabolic pathways related to fat storage and energy expenditure (6, 7). These viruses have been implicated in disrupting insulin signaling, enhancing adipogenesis, and modulating inflammation, which may contribute to the difficulty in reducing fat mass (9).

Moreover, these pathogens interact with the host microbiome, creating a metabolic environment that favors fat accumulation, even in individuals who are engaging in regular exercise or following a healthy diet (10). This interaction between the microbiome and infectious agents further complicates efforts to manage weight loss, as microbiome dysbiosis can influence how the body processes nutrients and burns fat. In this context, both exercise and diet are foundational to weight management, but their effectiveness can be diminished by underlying infections that alter metabolic responses (11). While exercise is known to improve insulin sensitivity, enhance fat oxidation, and promote muscle mass, and dietary interventions can create a caloric deficit necessary for weight loss, the presence of chronic infections may negate some of these benefits by modifying the body's metabolic set point and resistance to fat loss (12). Therefore, managing infectobesity requires not only addressing lifestyle factors such as exercise and nutrition but also considering the role of infections and their impact on the body's ability to lose weight effectively.

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4. Infectious Agents Implicated in Obesity

4.1. Human Adenoviruses and Adiposity

Among the candidate pathogens, Human Adenovirus 36 (HAdV-36) has accumulated the most evidence linking it to adiposity and obesity. Early clinical reports found a positive association between HAdV-36 seropositivity and higher body weight, paradoxically with lower serum lipids in some cohorts (6). Subsequent studies observed variable associations across different regions and age groups, with considerable heterogeneity driven by differences in diagnostic assays and study designs (7). Despite these inconsistencies, experimental work provides biological plausibility: in vitro and animal models have demonstrated that HAdV-36 promotes adipogenesis and lipid accumulation, while sometimes improving glycemic control, suggesting metabolic phenotype shifts that are not entirely captured by weight alone (9). Recent meta-analyses and systematic reviews (2015–2020 emphasis) continue to support a positive association between HAdV-36 seropositivity and obesity, though causality remains uncertain and likely context-dependent (12, 13). While seropositivity in obese individuals remains higher than in controls, this relationship appears influenced by factors such as geographical location and study methodology. Further studies are needed to standardize diagnostic techniques and improve temporality in the context of obesity research.

Newer human studies continue to explore the cardiometabolic correlates of HAdV-36 exposure, including insulin resistance profiles in school-aged children and inflammatory/oxidative signatures in adults (14). These studies underscore the idea that the infection's impact on obesity may not be purely anthropometric but rather metabolic in nature, potentially influencing broader aspects of metabolic health, such as inflammation and oxidative stress, which further complicates the traditional view of obesity as a purely energy imbalance-related disorder.

4.2. Other Viral and Microbial Candidates

In addition to HAdV-36, other viral and microbial candidates have been implicated in the development of obesity. SMAM-1, an avian adenovirus, has been associated with increased fat deposition and obesity in chickens, suggesting that avian adenoviruses may also play a role in metabolic regulation (4). Furthermore, Borna disease virus has been found to promote obesity in rats by inducing changes in central nervous system function that alter food intake and energy expenditure, highlighting the potential neurological effects of viral infections on obesity (12). Similarly, Scrapie agents, which cause prion diseases in animals, have been linked to obesity in mice and hamsters, demonstrating that infectious agents in the broader microbial landscape can influence fat storage and metabolic health (15). Moreover, emerging evidence suggests that other microbial infections, such as Helicobacter pylori and certain gut microbiota dysbiosis, could contribute to obesity by altering host metabolism and immune responses, with potential implications for weight gain and metabolic disease (10, 16). The role of these pathogens underscores the complex interaction between infection, metabolic pathways, and obesity, highlighting the need for further research to explore the potential for infections as environmental factors in the obesity epidemic.

5. Mechanistic Pathways Linking Infection to Obesity and Metabolic Dysregulation

Infectious agents may contribute to obesity and metabolic dysregulation through multiple interconnected mechanistic pathways that extend beyond simple energy imbalance.

"Ad36 infection has been shown to increase adiposity and metabolic risk markers in human populations, with seropositive individuals exhibiting greater fat accumulation and altered metabolic profiles compared to seronegative controls (17); Yamada, Hara & Kadowaki, 2012).

One primary mechanism is direct modulation of adipocyte biology: pathogens such as Human Adenovirus-36 (HAdV-36) can enhance pre-adipocyte differentiation and lipid storage by activating transcription factors like PPAR-γ and increasing intracellular lipid accumulation, providing biological plausibility for infection-induced adiposity in experimental models (van Ginneken et al., 2009; (6, 7). Moreover, HAdV-36 and related adenoviruses can alter insulin signaling, affecting glucose transport and cellular metabolism in adipose tissue and muscle, which may exacerbate insulin resistance and metabolic dysfunction (6, 7); van Ginneken et al., 2009).

Another key pathway involves chronic low-grade inflammation, a hallmark of many infections and metabolic

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disorders. Immune activation triggered by viral or microbial agents leads to increased production of pro-inflammatory cytokines such as TNF-α and IL-6, which impair insulin receptor signaling and promote adipose tissue dysfunction (12). This inflammatory state also influences adipose immune-cell infiltration, perpetuating a cycle of inflammation and metabolic derangement. Additionally, gut microbiota dysbiosis -disruption of the normal microbial ecosystem- has emerged as a critical mediator of obesity risk, influencing host energy harvest, intestinal permeability, systemic inflammation, and fat storage (11, 18, 19). Viral components of the gut microbiome (the virome) and other non-bacterial microbes further contribute to metabolic outcomes by modifying bacterial communities and host immune responses, with evidence showing distinct virome profiles in obesity (18).

A growing body of research in immunometabolism highlights how metabolic reprogramming of host immune responses to infection can itself influence systemic nutrient metabolism, suggesting that immune–metabolic crosstalk may amplify infection-driven obesity and related disorders (20). Taken together, these mechanisms illustrate that infection can act as both a direct adipogenic stimulus and an indirect modifier of metabolic regulation, emphasizing the need for integrated approaches in obesity research that consider infectious, immune, microbial, and metabolic pathways simultaneously.

6. Obesity, Infectious Factors, and Resistance to Lifestyle Interventions in the Context of COVID-19

Resistance to conventional lifestyle interventions, such as dietary modification and increased physical activity, remains a critical challenge in the management of obesity. Despite adherence to evidence-based programs, a significant proportion of individuals fail to achieve meaningful or sustained weight loss, underscoring the complex and multifactorial nature of obesity (21). While genetic, environmental, and behavioral factors are well-known contributors to obesity, emerging research indicates that infectious agents, including prior exposure to human adenovirus-36 (HAdV-36) and the SARS-CoV-2 virus, may influence metabolic responses and contribute to the variability in treatment outcomes (22, 23). These infections may modulate adiposity and metabolic pathways independent of lifestyle factors, making them essential considerations in understanding obesity resistance to treatment.

Numerous epidemiological studies have documented a positive association between HAdV-36 seropositivity and an increased risk of obesity, particularly among children and adolescents (24). Pooled odds ratios for obesity prevalence in seropositive individuals range from 1.84 to 2.44, suggesting that HAdV-36 infection may predispose individuals to obesity (25). Similarly, the COVID-19 pandemic has provided a unique context in which infectious factors could exacerbate obesity, as many individuals experienced weight gain during lockdowns due to reduced physical activity, increased sedentary behavior, and changes in dietary patterns (22). This phenomenon highlights the interaction between infectious diseases and lifestyle factors in shaping obesity trajectories.

Importantly, observational studies have also indicated that viral infections like HAdV-36 and SARS-CoV-2 may modify the metabolic response to lifestyle interventions. For instance, in a study involving adults with non-alcoholic fatty liver disease (NAFLD), HAdV-36 seropositive individuals who participated in a structured diet and physical activity intervention showed significantly greater reductions in BMI and improvements in insulin sensitivity compared to seronegative individuals (26). Conversely, the COVID-19 pandemic has led to increased rates of obesity in some populations, particularly among individuals who had previously experienced disruptions in lifestyle habits due to lockdown measures, contributing to resistance to lifestyle changes (27). These findings suggest that viral exposure can significantly alter how metabolic systems respond to changes in diet and physical activity.

Mechanistically, HAdV-36 and COVID-19 infection may induce alterations in adipogenesis, inflammation, glucose metabolism, and immune responses, all of which can affect the body's ability to adapt to lifestyle changes. For example, HAdV-36 infection has been shown to impact adipocyte differentiation and cytokine profiles, leading to increased inflammation and oxidative stress, which in turn could influence metabolic processes such as insulin resistance (28). Similarly, SARS-CoV-2 has been associated with long-term metabolic dysfunction in some individuals, including insulin resistance and increased abdominal

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obesity, even after recovery from the acute phase of infection (29). These viral-induced metabolic disruptions may either attenuate or amplify the physiological responses to diet and exercise, depending on an individual's infection history and immune system status (30).

The concept of **infectobesity**—the idea that certain infections, such as HAdV-36 and SARS-CoV-2, contribute to the development of obesity and metabolic dysregulation—may help explain why some individuals are resistant to conventional weight-loss interventions. This underscores the need to incorporate a patient's infectious and immunological history when designing personalized obesity treatment plans. Both HAdV-36 and SARS-CoV-2 exemplify how infections can disrupt metabolic pathways, creating conditions in which traditional lifestyle interventions may have reduced efficacy (31). Furthermore, the review by Xu et al. (2022) underscores the complexity of interpreting these associations due to heterogeneity in study designs, diagnostic methods, and the varying prevalence of HAdV-36 across geographic regions (32). Despite these challenges, the evidence supports the hypothesis that HAdV-36 infection may represent an underlying factor contributing to the growing obesity epidemic in children and adolescents, particularly in populations with limited access to effective obesity interventions (Table 1).

Therefore, future research should further explore the intersection between infectious diseases and obesity management, with a focus on identifying effective strategies for overcoming resistance to lifestyle interventions in individuals with a history of infection.

Table 1

Infectious Factors and Their Potential Role in Obesity Pathogenesis and Variability in Response to Lifestyle Interventions

Infectious Factor	Evidence Type	Key Findings	Strength of Evidence	Major Limitations	Key References (APA style)
HAdV-36	Epidemiological (Serology-based; Meta-analyses)	Positive association between HAdV-36 seropositivity and obesity; pooled OR ≈ 1.8–2.7 in children and adults	Moderate (Meta-analytic support)	Predominantly cross-sectional; temporality unclear; residual confounding	(33, 34)
HAdV-36	Mechanistic (Cellular & Animal models)	Promotes adipogenic differentiation (↑ PPARγ, C/EBPs); alters adipokine signaling; modifies glucose uptake	Strong (Experimental evidence)	Limited direct human mechanistic confirmation	(35, 36)
HAdV-36	Clinical intervention study	Seropositive NAFLD patients showed greater reductions in BMI and HOMA-IR after lifestyle intervention	Limited (Single study)	Small sample; specific clinical population	(36)
SARS-CoV-2	Population-level (Pandemic context)	Lockdown-associated weight gain; increased overweight/obesity prevalence	Strong (Systematic reviews/meta-analyses)	Behavioral/environmental confounding; not infection-specific	(37, 38)
SARS-CoV-2	Post-acute/Long COVID	Persistent insulin resistance and glycometabolic dysregulation in subset of individuals	Emerging	Heterogeneous cohorts; evolving longitudinal data	
SARS-CoV-2	Translational/Tissue-level studies	Infection of adipose tissue; induction of inflammatory responses	Strong (Translational evidence)	Limited long-term metabolic follow-up	(39)
Infectobesity hypothesis	Narrative & integrative reviews	Viral exposure proposed as a biological contributor to obesity risk	Conceptual/theoretical	Does not establish causality	(40)

7. Diet–Infection Interactions in Obesity Management

Diet quality plays a pivotal role in shaping host susceptibility and the response to infectious exposures that contribute to infectobesity by modulating immune and metabolic pathways (41). Poor diet quality—characterized by high energy density, low dietary fiber, and micronutrient deficiencies—is linked to impaired innate and adaptive immune responses, increased chronic low-grade inflammation, and disrupted cytokine signaling, creating

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conditions that may facilitate viral persistence and adverse adipose tissue remodeling (42, 43) . Obesity itself is associated with systemic immune dysregulation, including infiltration of proinflammatory immune cells into adipose tissue, which drives chronic inflammation and metabolic dysfunction (44, 45). Diet-induced obesity and metabolic imbalance also impair immune cell metabolism and function, including macrophage polarization and cytokine production, which have been implicated in impaired pathogen clearance and poorer outcomes in infections such as COVID-19 (15, 46)

From an immunometabolic perspective, nutrient availability is a key regulator of immune-cell metabolism, mitochondrial function, and inflammatory programming (47, 48). These processes provide a biological framework through which diet influences infection-related metabolic risks, beyond caloric balance alone (49). A high intake of refined carbohydrates and fats can shift immune cell behavior toward an inflammatory state, contributing to chronic diseases such as insulin resistance, hypertension, and obesity (50). This highlights the importance of dietary quality in not just managing energy balance, but also in modulating the immune system's ability to respond to infections.

The gut microbiota represents a critical mechanistic interface linking diet, infection, and obesity. Research has shown that diet-induced changes in microbial diversity and function can influence energy harvesting, bile acid metabolism, intestinal barrier integrity, and systemic inflammation—key processes that are also central to the development of insulin resistance and adiposity (51). High-fiber, plant-based dietary patterns and targeted prebiotic strategies are associated with improvements in short-chain fatty acid (SCFA) production and anti-inflammatory signaling. In contrast, ultra-processed diets consistently contribute to dysbiosis and metabolic inflammation, further exacerbating the obesity-inflammatory cycle (52). Infections may disrupt this delicate microbial ecosystem, amplifying obesity risk in susceptible individuals. On the other hand, dietary modifications and exercise together may improve microbial resilience and metabolic flexibility, helping to mitigate the negative effects of poor diet and infections (52, 53).

From a clinical and sports-science perspective, infection-related obesity hypotheses support a more personalized approach to lifestyle interventions. For example, adenovirus-36 (Ad36) seropositivity has been linked to systemic inflammation and oxidative stress imbalances independent of body mass index (BMI), suggesting that standard dietary prescriptions may not elicit uniform metabolic responses across individuals (41). Therefore, nutrition strategies emphasizing adequate protein intake, micronutrient sufficiency (e.g., zinc, vitamin D), improved fatty acid quality, dietary antioxidants, and sufficient fiber intake are particularly relevant in individuals with heightened immune-metabolic activation. These strategies are even more effective when combined with structured exercise and appropriate recovery (54). This layered approach does not replace established obesity management strategies but may provide insight into the inter-individual variability in response to diet and physical activity, thus guiding precision lifestyle prescriptions tailored to individual immune-metabolic profiles (Table 2).

The infographic underscores the interconnections between physiological responses, cognitive function, and general health. Exercise, proper diet, and effective management of infectious obesity collectively contribute to improved physiological responses, such as enhanced cardiovascular health and immune function. Moreover, these lifestyle changes positively influence cognitive function by improving neuroplasticity and reducing neuroinflammation, thereby promoting mental well-being and overall quality of life (55). General health is ultimately optimized when these factors are appropriately balanced, contributing to better long-term health and a reduced risk of chronic diseases (Figure. 1).

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Figure 1

Impact of infectious obesity and lifestyle on exercise and diet response

The infographic presents a comprehensive model that outlines the complex interactions between infectious obesity, lifestyle factors (such as exercise and diet), and their collective influence on immune response, inflammation, metabolic function, and overall health. This integrated framework emphasizes how these factors interrelate and affect physiological, cognitive, and metabolic outcomes, particularly in the context of metabolic health and disease prevention.

Table 2

Key Concepts in Infectious Obesity, Diet, Exercise, and Immune-Metabolic Interactions

Domain / proposition	Dietary or clinical context	Key mechanistic pathways (immunometabolic)	Immune / inflammatory implications	Metabolic / obesity implications	Practical implications for lifestyle intervention (nutrition + exercise)	References
Diet quality as a determinant of susceptibility to infection-related metabolic risk ("infectobesity" framework)	Low-quality diets characterized by high energy density, low fiber, and micronutrient insufficiency	Diet-driven modulation of immune and metabolic signaling; promotion of low-grade chronic inflammation; potential facilitation of viral persistence and maladaptive adipose remodeling	Impaired innate and adaptive immune responses; disrupted cytokine signaling; chronic low-grade inflammatory milieu	Increased vulnerability to adipose tissue dysfunction and infection-associated metabolic derangements	Prioritize dietary quality (fiber density, micronutrient adequacy, food processing level) in addition to caloric control, particularly in individuals with heightened immune–metabolic vulnerability	(34, 42, 43)
Obesity as an immunologically dysregulated state	Established obesity phenotype	Infiltration and activation of pro-inflammatory immune cells within adipose tissue; sustained inflammatory signaling contributing to metabolic dysfunction	Systemic immune dysregulation; chronic inflammation	Worsened metabolic homeostasis and amplification of the obesity–inflammation cycle	Frame obesity management as both metabolic and immunological; integrate anti-inflammatory lifestyle strategies (diet quality + structured physical activity)	(44, 45)
Diet-induced obesity and impaired host defense	Diet-induced obesity and metabolic imbalance	Altered immune-cell metabolism and function; macrophage polarization changes; dysregulated cytokine production	Potentially impaired pathogen clearance and worse outcomes in infections (e.g., COVID-19)	Greater insulin resistance and adverse cardiometabolic risk profile	Use combined interventions (nutrition + structured exercise + recovery) to improve immune competence and metabolic flexibility, and explicitly discuss infection-related risk modulation	(15)
Immunometabolism as a unifying explanatory layer	Nutrient availability as an upstream regulator	Regulation of immune-cell metabolic programming, mitochondrial function,	Shifts in inflammatory programming	Diet can influence infection-related metabolic risk via	In reviews, position immunometabolism as the mechanistic rationale for	(47, 48)

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beyond energy balance		and inflammatory set-points	and immune responsiveness	pathways not reducible to calories alone	heterogeneity in response to lifestyle prescriptions	
Refined carbohydrate and poor fat quality as drivers of immune activation	High intake of refined carbohydrates and fats	Skewing immune-cell behavior toward pro-inflammatory states	Elevated inflammatory tone	Increased risk of insulin resistance, hypertension, and obesity progression	Reduce ultra-processed/refined foods; improve fatty-acid quality and carbohydrate quality; track diet quality indices alongside weight outcomes	(56)
Gut microbiota as the mechanistic interface linking diet, infection, and obesity	Diet-induced shifts in microbial diversity and function	Altered energy harvest, bile acid metabolism, gut barrier integrity, and systemic inflammatory signaling	Increased systemic inflammation via impaired barrier integrity and immune activation	Contribution to insulin resistance and adiposity	Include microbiota-targeted mechanisms in synthesis; treat microbiota modulation as a plausible pathway for intervention effects	(57)
High-fiber / plant-based patterns and prebiotic strategies as anti-inflammatory modulators	Increased dietary fiber; prebiotic-focused approaches	Enhanced short-chain fatty acid (SCFA) production; improved anti-inflammatory signaling	Strengthened anti-inflammatory signaling; improved immune–gut homeostasis	Support for metabolic flexibility and improved insulin sensitivity pathways	Recommend fiber-forward, plant-rich patterns and targeted prebiotics where appropriate; integrate with exercise for broader metabolic benefit	(57)
Ultra-processed dietary patterns and dysbiosis	High intake of ultra-processed foods	Promotion of dysbiosis and metabolic inflammation	Sustained metabolic inflammation	Exacerbation of the obesity–inflammation cycle	Emphasize reduction of ultra-processed foods as a core lever in lifestyle-based obesity management	(58)
Infection as a disruptor of microbial resilience	Infections in susceptible individuals	Disruption of gut microbial ecology and resilience; amplification of inflammatory signaling	Heightened inflammatory instability; altered immune–microbiome interactions	Increased obesity risk or worsened metabolic control in vulnerable phenotypes	Highlight bidirectionality: infections may worsen dysbiosis; dietary and exercise strategies may improve resilience	(59)
Synergistic effects of diet and exercise on microbial and metabolic resilience	Dietary modification combined with structured physical activity	Improved microbial resilience and metabolic flexibility	Better inflammatory regulation and immune function	Improved cardiometabolic outcomes and potentially more robust response to lifestyle interventions	Present combined lifestyle prescriptions (diet + exercise + recovery) as the clinically actionable standard, especially under infection-related risk hypotheses	(59)
Inter-individual variability: Ad36 seropositivity and non-uniform response to standard prescriptions	Adenovirus-36 (Ad36) seropositivity as a proposed modifier	Association with systemic inflammation and oxidative stress imbalance independent of BMI	Immune–metabolic activation markers may differ despite similar BMI	Potential heterogeneity in metabolic response to lifestyle change	Support “precision lifestyle” framing: adequate protein; micronutrient sufficiency (e.g., zinc, vitamin D); improved fatty-acid quality; dietary antioxidants; sufficient fiber; plus structured exercise and recovery	(60)
Broader physiological and cognitive outcomes of integrated lifestyle management	Exercise, high-quality diet, and infection-related obesity considerations	Improved cardiovascular and immune function; reduced neuroinflammation; enhanced neuroplasticity	Strengthened immune function and reduced inflammatory burden	Lower long-term chronic disease risk and improved quality of life	In a review, justify multi-domain endpoints (physiological + cognitive + general health) when discussing comprehensive lifestyle frameworks	(61)

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8. Cytokine Changes in Active Individuals with Healthy Diet vs Inactive Individuals with Unhealthy Diet for Weight Loss

Numerous studies have shown that regular physical activity and healthy diet have positive effects on cytokine regulation and improve immune responses in the body. Active individuals with a healthy diet typically have lower levels of inflammatory cytokines such as IL-6, TNF-α, and CRP (C-reactive protein), which are associated with a decreased risk of chronic diseases like obesity, type 2 diabetes, and cardiovascular diseases. In contrast, inactive individuals with an unhealthy diet, especially those with a diet high in saturated fats and simple sugars, usually have higher levels of inflammatory cytokines, leading to chronic inflammation and, ultimately, increased obesity and insulin resistance (62, 63).

8.1. Impact of Physical Activity on Cytokines

Research has shown that regular exercise significantly reduces levels of IL-6, TNF-α, and other inflammatory cytokines. Interestingly, high-intensity physical activity may further enhance these effects. In a study conducted by Pedersen and Hoffman-Goetz, it was shown that regular exercise in individuals with chronic obesity leads to reduced inflammation in adipose tissue and significantly decreases IL-6 and TNF-α levels (62).

8.2. Role of Healthy Diet in Cytokine Regulation

A healthy diet also plays a critical role in regulating cytokines and reducing inflammation. Diets rich in fiber, healthy fats (like omega-3), and antioxidants have been shown to lower inflammatory cytokines and contribute to overall body health. In contrast, diets high in trans fats and added sugars can increase the levels of IL-6, TNF-α, and CRP, leading to chronic inflammation and the development of metabolic disorders like obesity and diabetes (63, 64). In another study, plant-based diets and fiber-rich diets significantly reduced the release of inflammatory cytokines, especially IL-6 and IL-1β (65).

8.3. Cytokine Changes in Weight Loss

In studies comparing active individuals with a healthy diet to inactive individuals with an unhealthy diet, it has been shown that active individuals with a healthy diet not only lose more weight but also have significantly lower levels of inflammatory cytokines like IL-6, TNF-α, and CRP, with a faster rate of weight loss. In contrast, inactive individuals with an unhealthy diet have higher levels of these cytokines, and their weight loss process is significantly slower (66).

8.4. Cytokines and Insulin Resistance in Obesity

Several studies have demonstrated that high levels of inflammatory cytokines such as IL-6 and TNF-α in obese individual contribute to insulin resistance and central obesity. Inactive individuals with an unhealthy diet have higher levels of these cytokines, which directly affect fat storage and lead to increased obesity. In contrast, active individuals with a healthy diet have lower levels of these cytokines and are more metabolically resistant to metabolic diseases (50).

9. Age-Specific Activity Perspectives

9.1. Childhood and Adolescence

Early-life infections and nutritional exposures can significantly influence immune and metabolic pathways, potentially setting the trajectory for obesity and metabolic disorders in later life (67). Childhood obesity, often tracking into adulthood, is associated with increased risk of developing chronic conditions such as cardiovascular diseases and type 2 diabetes (68). Research indicates that early pathogen exposure could influence the development of adiposity by modulating immune responses, potentially altering the programming of fat storage and metabolic regulation (69). However, it remains unclear whether these infectious exposures modify the long-term response to family-based lifestyle interventions aimed at reducing pediatric obesity (67). To address this, future studies should explore the relationship between childhood infections and obesity development, including the interaction with interventions like diet and physical activity, taking into account pathogen-specific effects on immune-metabolic programming (68).

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9.2. *Adulthood*

In adults, several factors including occupational activity, sleep patterns, stress levels, and dietary habits have a profound effect on immune function and inflammatory tone (70). This interaction is central to understanding metabolic heterogeneity among individuals, particularly in the context of obesity (69). The concept of infectobesity, wherein early or chronic infections exacerbate obesity and metabolic dysfunction, may further contribute to this heterogeneity by altering inflammatory and immune responses (69). Given these complexities, interventions in adults could be enhanced by incorporating infectious-status-informed phenotyping (70). This approach, combining serological assessments with metabolic biomarkers, could help tailor diet and exercise strategies more effectively (69). By recognizing the immune-metabolic interactions in response to infections, interventions could be optimized to target individuals who may be more susceptible to metabolic disturbances due to past infections (70).

9.3. *Older Adults*

Older adults face significant challenges related to immunosenescence, the age-related decline in immune system function, and higher rates of sarcopenic obesity (71). Sarcopenic obesity, characterized by the coexistence of excess body fat and reduced muscle mass, is a growing concern in the elderly population (71). This condition not only contributes to frailty but also impairs mobility and increases vulnerability to infections and other health complications (72). As the immune system weakens with age, infection prevention and management become crucial to maintaining physical function and the ability to engage in physical activity (72). Resistance training, combined with adequate protein intake, is essential for preserving muscle mass and function, which can mitigate the effects of sarcopenic obesity (72). Additionally, addressing infectious risk and promoting active management of chronic conditions through preventive measures and timely treatments are critical to protecting the physical and cognitive health of older adults, ensuring their continued participation in physical activities (71).

10. Research Gaps and Future Directions

Key gaps include temporality (does infection precede adiposity gain?), assay validity and comparability across studies, residual confounding, and publication bias. The field would benefit from prospective cohorts with repeated infectious assessments, standardized serology, and harmonized metabolic phenotyping, as well as intervention trials that stratify by infectious status and quantify mechanistic intermediates.In sports and exercise contexts, future research should address how training load, energy availability, and diet composition interact with infectious exposures to shape body composition, performance, and recovery. Methodologically rigorous designs—combining wearable-derived load metrics, validated illness surveillance, and objective biomarkers—are necessary to move beyond plausible narratives.

11. Conclusions

Infectobesity offers a biologically plausible extension to multifactorial obesity models by highlighting that infectious exposures can modify adipocyte biology, inflammation, and metabolic phenotypes. The strongest evidence centers on HAdV-36, supported by experimental data and contemporary meta-analytic synthesis, yet causality and clinical utility remain unresolved. For practitioners in nutrition, obesity medicine, and sports science, an integrated “infection–lifestyle” perspective encourages precision phenotyping, attention to immune competence during weight management, and broader outcome selection beyond body weight. Advancing the field will require standardized diagnostics, longitudinal and interventional designs, and careful integration of infection biology with diet and exercise prescriptions.

Authors’ Contributions

KH. I. contributed to the writing of the original draft, reviewing and editing, resource management, project administration, data curation, and conceptualization. J. R. was involved in writing the original draft, reviewing and editing, supervision, data curation, and conceptualization. M. J. was involved in writing the original draft, reviewing and editing,

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Declaration

AI-assisted tools (including large language models) were used to support English-language editing and clarity of presentation. The authors reviewed, edited, and verified all content and take full responsibility for the accuracy, integrity, and originality of the final manuscript. No AI tool was used to generate or manipulate the study data, analyses, or results.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

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Declaration of Interest

The authors report no conflict of interest.

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Ethics Considerations

This review article was conducted during a sabbatical period in collaboration with the SANA Institute for Avian Health and Diseases Research. The ethical approval was not required, as the study conducted did not involve any ethical concerns or issues.

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