International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Scholarly Publisher
RS Global Sp. z O.O.
ISNI: 0000 0004 8495 2390

Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

ARTICLE TITLE	LONG-TERM ENDOCRINE SEQUELAE AFTER COVID-19: CLINICAL INSIGHTS, DIGITAL HEALTH PERSPECTIVES AND PUBLIC HEALTH CHALLENGES
DOI	https://doi.org/10.31435/ijitss.4(48).2025.4194
RECEIVED	26 October 2025
ACCEPTED	14 December 2025
PUBLISHED	21 December 2025
LICENSE	The article is licensed under a **Creative Commons Attribution 4.0 International License.**

LONG-TERM ENDOCRINE SEQUELAE AFTER COVID-19: CLINICAL INSIGHTS, DIGITAL HEALTH PERSPECTIVES AND PUBLIC HEALTH CHALLENGES

Katarzyna Bielawska *(Corresponding Author, Email: bielawskka@wp.pl)*
Grochowski Hospital, Warsaw, Poland
ORCID ID: 0009-0001-7381-8415

ABSTRACT

The COVID-19 pandemic has revealed that SARS-CoV-2 infection affects not only the respiratory system but also multiple endocrine organs, leading to a broad spectrum of long-term hormonal disturbances. This narrative review aims to summarize the current evidence on endocrine sequelae after COVID-19, emphasizing pathophysiological mechanisms, clinical manifestations, and implications for patient management. A structured literature search was conducted in PubMed, Scopus, Web of Science, and Google Scholar for studies published between January 2020 and October 2025 addressing endocrine or metabolic consequences of COVID-19.

The reviewed data demonstrate that SARS-CoV-2 can impair the hypothalamic–pituitary–adrenal, thyroid, gonadal, pancreatic, and growth hormone axes through direct viral injury, immune-mediated inflammation, and chronic stress-axis dysregulation. Clinical consequences include secondary adrenal insufficiency, autoimmune thyroiditis, new-onset diabetes, hypogonadism, and menstrual disorders. While many abnormalities appear transient, a subset of patients develop persistent dysfunction requiring long-term follow-up.

Tele-endocrinology and digital health solutions have emerged as valuable tools for monitoring endocrine recovery and improving access to care. However, significant research gaps remain, particularly regarding long-term prevalence, reversibility, and mechanisms of endocrine injury. Understanding and addressing these complications will require coordinated, multidisciplinary strategies that integrate clinical endocrinology, public health, and digital innovation.

KEYWORDS

COVID-19, Long COVID, Endocrine Dysfunction, Adrenal Insufficiency, Autoimmune Thyroiditis, Tele-Endocrinology

CITATION

Katarzyna Bielawska. (2025) Long-Term Endocrine Sequelae After COVID-19: Clinical Insights, Digital Health Perspectives and Public Health Challenges. *International Journal of Innovative Technologies in Social Science*. 4(48). doi: 10.31435/ijitss.4(48).2025.4194

Introduction

The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has become one of the most serious health challenges of the 21st century. In the initial phase of research, the focus was mainly on acute complications of the respiratory, cardiovascular, and nervous systems. Over time, and with the emergence of a population of recovered patients, it has become apparent that SARS-CoV-2 infection can lead to long-term multi-organ disorders, collectively referred to as long COVID or post-COVID-19 syndrome. Among these complications, endocrine consequences are becoming increasingly important. They involve direct damage to the endocrine glands as well as secondary hormonal dysregulation as a result of the body's chronic inflammatory and immune response [1–3].

The endocrine system is an important target of SARS-CoV-2 infection due to the rich expression of ACE-2 receptors and TMPRSS2 protease in numerous tissues (thyroid, adrenal glands, pancreas, gonads, pituitary gland) [4, 5]. The virus, by penetrating the epithelial cells of the endocrine glands, causes direct cytotoxic damage and secondary immuno-endocrine disorders resulting from cytokine storm, oxidative stress,

and microthrombi. This leads to numerous hormonal disorders – from transient, subclinical dysfunctions to permanent damage to the glands, requiring chronic replacement therapy [8–9].

In recent years, there have been increasing reports that COVID-19 may lead to long-term consequences in all major hormonal axes. Cases of adrenal insufficiency and secondary pituitary disorders [10–12], autoimmune thyroiditis and non-thyroidal illness syndrome [6, 7], as well as newly diagnosed type 2 diabetes after COVID-19 [15–17]. Hormonal disorders have also been observed in men, including a decrease in testosterone levels and deterioration in semen quality [18, 19]. In women, menstrual cycle and reproductive function disorders have been observed [20]. The common denominator of these disorders appears to be persistent dysregulation of the immune-neuro-endocrine axis, which persists long after the infection has resolved [21, 22].

Understanding these mechanisms is important not only clinically, but also socially and economically. Endocrine disorders after COVID-19 lead to a deterioration in quality of life, chronic fatigue, reduced work productivity, and increased healthcare costs. In response, the role of tele-endocrinology and digital tools enabling remote monitoring of patients with hormonal disorders after COVID-19 is growing [24, 25]. The integration of clinical care with e-health and artificial intelligence solutions may be a breakthrough in the long-term management of the effects of the pandemic in the endocrinology patient population.

The aim of this paper is to present the current state of knowledge on the long-term endocrine consequences of COVID-19, taking into account pathophysiological mechanisms, clinical symptoms, modern diagnostic methods, and the prospects for the use of digital technologies in monitoring and caring for patients. The review focuses on key hormonal axes (HPA, HPT, HPG, pituitary, and pancreatic) and analyzes the consequences of these disorders for public health and clinical practice.

Methodology

This narrative review was conducted through a structured search of PubMed, Scopus, Web of Science, and Google Scholar for peer-reviewed articles published between January 2020 and October 2025. The search terms included “COVID-19”, “SARS-CoV-2”, “endocrine”, “hormonal dysfunction”, “thyroid”, “adrenal”, “pituitary”, “gonadal”, “diabetes”, and “long COVID”. Only human studies written in English were included, focusing on endocrine or metabolic consequences of COVID-19. Non-peer-reviewed and animal studies were excluded. The extracted data were analyzed qualitatively and organized according to affected endocrine axes and relevant clinical outcomes. Due to methodological heterogeneity, the findings are presented descriptively without meta-analysis.

Results

Hypothalamic–Pituitary–Adrenal (HPA) Axis Dysregulation

HPA axis dysfunction is the most common long-term endocrine consequence of COVID-19 infection. Numerous observational studies and reviews indicate that SARS-CoV-2 infection can lead to both transient and persistent adrenal insufficiency [10–12]. The mechanisms of this dysfunction are complex. They include direct damage to adrenal cortex cells by the virus, secondary damage to the pituitary gland, as well as chronic activation of the stress axis and dysregulation of cortisol secretion [13]. Clinical studies have shown reduced morning cortisol and ACTH concentrations in some patients after COVID-19, which may indicate the central nature of the disorders [10, 11]. Long-term observations, covering a period of up to 24 months after infection, confirm the possibility of reversible, partial HPA axis failure in some recovered patients [10]. Table 1 summarizes the major endocrine axes affected by SARS-CoV-2 and their main clinical manifestations.

Table 1. Major endocrine axes affected by COVID-19 and their clinical manifestations

Endocrine axis	Reported disorders	Proposed mechanisms	Clinical implications
Hypothalamic–pituitary–adrenal (HPA)	Secondary adrenal insufficiency, relative hypocortisolism	Direct viral injury, pituitary inflammation, chronic cytokine activation	Fatigue, hypotension, weakness, depressive symptoms
Thyroid (HPT)	Subacute thyroiditis, autoimmune hypothyroidism, NTIS	Viral tropism for thyrocytes, cytokine storm, oxidative stress	Thyrotoxicosis, hypothyroidism, prolonged fatigue
Pancreatic (metabolic)	New-onset diabetes, insulin resistance	β-cell damage, chronic inflammation, glucocorticoid therapy	Hyperglycemia, weight gain, metabolic syndrome
Gonadal (HPG)	Hypogonadism, menstrual irregularities	Leydig/Sertoli cell damage, hypothalamic dysfunction, stress	Infertility, libido loss, menstrual changes
Pituitary and GH axis	Hypopituitarism, GH deficiency	Autoimmune and inflammatory injury, microvascular changes	Weakness, reduced exercise tolerance, metabolic abnormalities

Table 1 summarizes the main endocrine axes affected by COVID-19, the mechanisms involved, and their key clinical consequences.

An important pathophysiological aspect is the impact of chronic inflammatory response and pro-inflammatory cytokines (IL-6, TNF-α) on pituitary and adrenal function. Excessive activation of the stress axis in the acute phase of infection can lead to its secondary "burnout" and relative hypocortisolemia in the convalescence phase [12–13]. Cases of pituitary inflammation and transient hypopituitarism have also been described, which may explain the observed symptoms of chronic fatigue, hypotension, mood disorders, and exercise intolerance in some patients with long COVID [13, 14].

From a clinical point of view, it is recommended that patients with persistent symptoms of asthenia, hypotension, or metabolic disorders after COVID-19 should be evaluated for HPA axis dysfunction, including measurement of morning cortisol and ACTH stimulation testing [11]. Early identification of these disorders is therapeutically important, as in many cases improvement is observed after the introduction of low-dose glucocorticoid replacement therapy. At the same time, long-term studies suggest that in some patients, HPA axis function gradually normalizes, indicating the potential reversibility of the changes [10, 12].

Thyroid Dysfunction

Thyroid dysfunction is a common endocrine complication of COVID-19, including both transient and persistent forms of gland dysfunction. Observational studies and meta-analyses have shown that SARS-CoV-2 infection can lead to subacute thyroiditis (SAT), non-thyroidal illness syndrome (NTIS), and the onset or exacerbation of autoimmune thyroid diseases [6, 7]. The mechanisms of these disorders are multifactorial: they include direct damage to thyrocytes by the virus, overexpression of ACE-2 receptors in thyroid tissue, as well as secondary effects of cytokine storm, oxidative stress, and microcirculation changes [4, 8].

Systematic studies of the adult convalescent population have shown that even after a mild course of COVID-19, abnormalities in thyroid hormone concentrations are observed. Most often, there is a decrease in FT3 and TSH levels with normal FT4, indicating the phenomenon of low T3 characteristic of NTIS [6]. In turn, some patients develop transient thyrotoxicosis with features of subacute thyroiditis, accompanied by neck pain and elevated CRP [7]. An interesting observation is that these disorders often resolve within a few months, but in some cases, chronic autoimmune changes persist, including an increase in anti-TPO or anti-Tg antibody titers, which may herald the development of permanent hypothyroidism [8, 9].

Currently, attention is being drawn to the need to monitor thyroid function in patients who have had COVID-19. This is particularly important in people with previous autoimmune diseases, women, and patients with symptoms of chronic fatigue, weight gain, or cold intolerance [6, 9]. Modern models of endocrine care, using telemonitoring and remote reporting of TSH results, can facilitate early detection of these disorders and reduce the need for direct outpatient visits [24].

Pancreatic and Metabolic Effects

Increasing evidence suggests that SARS-CoV-2 infection can cause both transient and persistent carbohydrate metabolism disorders, suggesting a role for the pancreas in the long-term endocrine consequences of COVID-19. Population studies and meta-analyses have shown an increased incidence of newly diagnosed type 2 diabetes among people who have had COVID-19, regardless of the severity of the infection [15, 16]. Pathophysiological mechanisms include direct damage to pancreatic β cells resulting from the presence of ACE-2 and TMPRSS2 receptors in these cells, as well as the indirect effects of cytokine storm, hypoxia, microcirculation disorders, and chronic inflammation [17, 26].

Studies have confirmed that the risk of developing diabetes after COVID-19 may be increased by as much as 40-60% compared to the general population. This phenomenon is observed both in people with a previous metabolic predisposition and in previously healthy patients [15,16]. In addition to de novo type 2 diabetes, there is also an increase in insulin resistance, lipid disorders, and features of metabolic syndrome, which may persist for many months after infection [17]. Some of these changes may be reversible – long-term studies have described cases of normalization of glycemia and metabolic parameters within 6–12 months after recovery [15].

The literature also emphasizes the role of iatrogenic factors, such as the use of glucocorticosteroids in COVID-19 therapy, which may accelerate the onset of diabetes in predisposed individuals [26]. On the other hand, chronic stress and activation of the HPA axis can lead to persistent hyperglycemia and insulin resistance, which are part of the so-called post-COVID metabolic syndrome.

Periodic monitoring of glycemia, glycated hemoglobin (HbA1c), and lipid profile is recommended in patients who have had COVID-19, especially in those with symptoms of fatigue, weight gain, or polydipsia. From a public health perspective, the increase in diabetes cases after the pandemic poses a serious burden on healthcare systems [28]. There is a growing emphasis on the need to implement secondary prevention programs and use telemedicine tools for long-term monitoring of metabolic parameters in recovered patients.

Gonadal and Reproductive Disorders

The gonadal axis is another endocrine system whose function may be disrupted by SARS-CoV-2 infection. Numerous publications have described the impact of COVID-19 on gonadal function in males and females, pointing to potential consequences for fertility and reproductive health [18–20]. Pathophysiological mechanisms include direct expression of ACE-2 and TMPRSS2 receptors in Leydig cells, Sertoli cells, and seminiferous tubule epithelium, which allows the virus to enter testicular tissues and cause a local inflammatory response [18]. Moreover, chronic hypoxia, oxidative stress, and systemic inflammation can lead to impaired spermatogenesis and decreased testosterone levels.

A systematic review has shown that men who have had COVID-19 often have reduced testosterone levels, increased LH levels, and impaired semen parameters, including reduced sperm motility and concentration [18]. Some studies suggest that these disorders may be temporary and improve after a few months, but in some patients they persist longer, which may indicate permanent damage to Leydig cells [19].

However, women experience menstrual cycle disorders, such as longer or shorter cycles and changes in bleeding intensity. These abnormalities may result from dysregulation of the hypothalamic-pituitary-gonadal (HPG) axis and emotional and metabolic stress accompanying the infection. Attention is also drawn to the possible link between long COVID and changes in estrogen and progesterone levels, which may explain symptoms such as fatigue, low mood, and sleep disturbances observed in women after the disease [20].

From a clinical practice perspective, it is recommended to monitor gonadal function in patients after COVID-19, especially in cases of persistent symptoms of hypogonadism, infertility, or menstrual disorders. The inclusion of hormonal diagnostics (testosterone, LH, FSH, estradiol) and assessment of semen parameters or the ovulatory cycle may be important for early intervention and improving patients' quality of life. In the long term, the importance of interdisciplinary collaboration between endocrinologists, gynecologists, and reproductive medicine specialists in assessing the impact of COVID-19 on the fertility and hormonal health of the population is growing.

Pituitary and Growth Hormone Axis

An increasing body of evidence indicates that SARS-CoV-2 may affect pituitary function, leading to alterations in the secretion of tropic hormones and secondary dysfunction of peripheral endocrine glands. Clinical and observational studies have described cases of secondary adrenal insufficiency, hypogonadism, and disturbances in the GH–IGF-1 axis among patients recovering from COVID-19 [13, 14]. The underlying mechanisms are likely multifactorial, involving autoimmune processes, microvascular injury within the pituitary gland, and chronic inflammation resulting in damage to somatotroph cells [21].

Di Filippo et al. [13] highlighted a possible bidirectional relationship between long COVID and pituitary dysfunction—on one hand, SARS-CoV-2 may directly impair pituitary tissue, while on the other, neuroendocrine dysregulation could contribute to the persistence of long COVID symptoms such as chronic fatigue and metabolic imbalance. Neuroendocrine studies have also demonstrated reduced serum IGF-1 concentrations in some post-COVID patients, suggesting impaired GH secretion that may underlie symptoms such as asthenia, muscle weakness, and reduced exercise tolerance [14].

Koch [21] and Bansal et al. [22] emphasized that chronic immune-endocrine activation and persistent stress-axis dysregulation may lead to complex neurohormonal alterations involving not only the HPA but also the GH–IGF-1 and gonadotropic axes. These disturbances frequently overlap with the chronic fatigue syndrome (CFS) phenotype observed in long COVID, which further complicates diagnostic assessment.

Given these findings, routine hormonal evaluation should be considered for patients experiencing persistent weakness, orthostatic hypotension, libido changes, or unexplained body weight fluctuations following COVID-19. Testing of ACTH, cortisol, prolactin, LH, FSH, and IGF-1 levels is particularly recommended when hypopituitarism is suspected. Although many cases appear transient, reports of structural pituitary alterations on MRI imaging suggest that long-term impairment may occur in certain individuals [13, 21].

Digital Health and Public Health Aspects

The rapid growth of telemedicine and digital health technologies has become a crucial component of endocrine care in the post-COVID-19 era. The pandemic accelerated the adoption of remote healthcare solutions, allowing for the ongoing assessment and follow-up of endocrine disorders even when in-person consultations were limited [24]. Several studies have shown that tele-endocrinology, which integrates virtual consultations, remote laboratory data sharing, and symptom tracking, can significantly improve access to care and facilitate early detection of hormonal and metabolic complications after COVID-19 [24, 25].

In a systematic review, SeyedAlinaghi et al. [24] emphasized the value of telehealth in managing chronic endocrine sequelae of COVID-19, such as diabetes, hypothyroidism, and HPA axis dysfunction. Remote monitoring of glucose, TSH, or cortisol levels enables physicians to adjust treatment in real time while reducing the need for face-to-face visits. Artificial intelligence–based algorithms are also being explored as tools for identifying subtle hormonal imbalances and predicting the risk of long-term endocrine damage.

Zupa et al. [25] conducted a qualitative study examining the experiences of patients and clinicians using telemedicine in type 2 diabetes care. Both groups generally viewed these digital approaches positively, though they noted technical limitations and the importance of maintaining strong data security and patient engagement. In the context of long COVID, remote endocrine monitoring may play a key role in coordinating multidisciplinary care between endocrinologists, primary physicians, and rehabilitation specialists.

From a broader public health perspective, integrating tele-endocrinology into standard healthcare systems could enhance care quality, lower costs, and provide faster responses to newly emerging cases of post-COVID endocrine dysfunction. In the long term, these digital strategies may form the backbone of an integrated, patient-centered model for managing chronic endocrine conditions in the aftermath of the pandemic.

Discussion

The findings presented in this review demonstrate that SARS-CoV-2 infection can lead to long-lasting and diverse endocrine abnormalities affecting nearly all major hormonal axes. These changes have been observed both after severe and mild cases of COVID-19, suggesting that the mechanisms responsible for endocrine dysregulation extend beyond the acute phase of infection [1–5, 10–22]. Growing evidence supports the view that COVID-19 should be regarded as a multisystem disease capable of inducing sustained disturbances in endocrine function.

Pathophysiological mechanisms of endocrine dysfunction after COVID-19

Among the proposed mechanisms, direct viral injury to endocrine cells appears to play a central role. This is enabled by the expression of ACE-2 receptors and the TMPRSS2 protease in various endocrine tissues [4, 17, 18]. SARS-CoV-2 has been shown to infect thyroid follicular cells, pancreatic β-cells, Leydig cells, and even pituitary cells, disrupting hormone secretion and triggering local inflammation and apoptosis. Autopsy studies have confirmed the presence of viral genetic material in the thyroid, pancreas, and adrenal glands, supporting the hypothesis of endocrine tropism [5, 17]. Table 2 summarizes the main mechanisms proposed to explain post-COVID endocrine dysfunction.

Table 2. Pathophysiological mechanisms underlying endocrine dysfunction after COVID-19

Mechanism	Affected glands	Key mediators	Supporting evidence
Direct viral cytotoxicity	Thyroid, pancreas, adrenal glands	ACE-2, TMPRSS2 expression	Autopsy and in vitro studies
Autoimmune response	Thyroid, pituitary, pancreas	Anti-TPO, anti-Tg, anti-islet antibodies	Case series and observational data
Chronic inflammation	Multiple axes	IL-6, TNF-α, IL-1β	Longitudinal cohort studies
Microvascular injury	Pituitary, adrenal glands	Endothelial dysfunction, hypoxia	Imaging and histopathology
Iatrogenic therapy	Pancreas, adrenal glands	Glucocorticoid exposure	Clinical observations

Table 2 outlines the proposed mechanisms contributing to endocrine dysfunction after COVID-19 infection.

Another key mechanism involves chronic immune activation and secondary autoimmunity. Many patients recovering from COVID-19 develop new or increased titers of anti-thyroid (anti-TPO, anti-Tg), anti-pituitary, or anti-islet antibodies [8, 13, 14]. This autoimmune response, likely driven by prolonged cytokine activation (IL-6, TNF-α, IL-1β), may disturb hypothalamic–pituitary regulation and promote secondary endocrine failure [4, 21]. Additionally, oxidative stress and microvascular thrombosis can cause local ischemia and cell damage, particularly in the adrenal and pituitary glands [10, 11, 13].

Differences across hormonal axes

The hypothalamic–pituitary–adrenal (HPA) axis appears to be one of the most frequently affected. Several studies have reported low serum cortisol and ACTH levels, consistent with secondary adrenal insufficiency [10–13]. While many of these alterations are transient, persistent cases requiring glucocorticoid replacement have also been described.

Thyroid dysfunction is another common manifestation, ranging from non-thyroidal illness syndrome to subacute thyroiditis and autoimmune hypothyroidism [6–9]. In some individuals, persistent elevation of thyroid autoantibodies suggests that SARS-CoV-2 may trigger or exacerbate autoimmune thyroid disease.

With respect to metabolic regulation, multiple studies have confirmed an increased incidence of new-onset type 2 diabetes and insulin resistance following COVID-19 [15–17]. The proposed mechanisms include direct β-cell injury, inflammatory and lipid metabolism disturbances, and the iatrogenic effects of glucocorticoid therapy [26, 28]. This phenomenon could contribute to a long-term rise in global diabetes prevalence.

The gonadal axis is also affected, with reduced testosterone levels and impaired semen parameters in men, as well as menstrual irregularities in women [18–20]. These changes may reflect both direct gonadal infection and disruption of the hypothalamic–pituitary–gonadal (HPG) axis. In addition, alterations in the growth hormone–IGF-1 axis have been observed, including reduced IGF-1 levels and clinical features of GH deficiency, which may underlie fatigue and metabolic abnormalities in long COVID [13, 21, 22].

Clinical and societal implications

From a clinical perspective, these findings highlight the need for systematic endocrine monitoring in individuals recovering from COVID-19. Hormonal assessment— including cortisol, TSH, FT3, FT4, glucose, HbA1c, and, in men, testosterone and semen parameters—should be considered in cases of unexplained fatigue, weight changes, or persistent metabolic symptoms [10–20]. Early identification of endocrine abnormalities allows timely therapeutic intervention, often improving patient well-being and functional recovery.

However, the impact of post-COVID endocrine dysfunction extends beyond biochemistry. Symptoms such as chronic fatigue, sexual dysfunction, menstrual irregularities, and metabolic disturbances significantly affect mental health and quality of life. Many patients benefit from a multidisciplinary approach that combines endocrinology, psychology, nutrition, and physical rehabilitation [19, 20, 25].

In this context, telemedicine and digital health tools are becoming increasingly valuable. Studies have shown that tele-endocrinology enables effective management of post-COVID endocrine disorders, particularly diabetes, thyroid disease, and adrenal insufficiency [24, 25]. Remote data monitoring and AI-assisted interpretation can improve diagnostic accuracy and support long-term care while reducing the burden on healthcare systems. Table 3 presents examples of digital and telemedicine tools that can support long-term endocrine care in patients recovering from COVID-19.

Table 3. Emerging digital tools and strategies for post-COVID endocrine care

Focus area	Digital/clinical tool	Application	Expected benefits
Diabetes monitoring	Tele-endocrinology, continuous glucose tracking	Real-time data sharing and therapy adjustment	Improved glycemic control, fewer in-person visits
Thyroid follow-up	Virtual consultations, e-TSH reporting systems	Early detection of hypothyroidism or relapse	Timely dose optimization
Adrenal evaluation	Wearable stress and HRV monitors	Remote detection of adrenal fatigue	Prevents adrenal crisis
Integrated care	AI-based decision dashboards	Coordination across specialties	Personalized long-term management

Table 3 presents examples of digital and telemedicine tools that can support long-term endocrine care in patients recovering from COVID-19.

Research gaps and future directions

Despite the growing body of evidence, many aspects of the post-COVID endocrine syndrome remain insufficiently understood. There is a lack of large, prospective, long-term studies that would clarify the prevalence, duration, and reversibility of endocrine abnormalities [1, 5, 14]. It also remains uncertain to what extent these changes are driven by direct viral effects versus immune or therapeutic factors.

Future research should integrate imaging modalities (MRI, ultrasound, PET) with hormonal profiling and immunological analyses to better characterize organ-specific damage. Genetic and molecular studies may help identify individuals at higher risk for persistent endocrine sequelae. Moreover, the development of biomarkers for early detection and prognosis could guide patient monitoring and treatment strategies.

From a public health standpoint, it is essential to establish integrated long-term care models for patients with long COVID that include endocrine evaluation, metabolic screening, and psychological support. Telehealth platforms and data-driven monitoring systems may play a central role in ensuring continuity of care and improving outcomes in this growing patient population.

Conclusions

The long-term endocrine effects of COVID-19 represent an emerging field of clinical and scientific importance. Evidence accumulated over the past few years clearly indicates that SARS-CoV-2 can disrupt multiple hormonal axes, either through direct viral injury, immune-mediated inflammation, or the secondary consequences of systemic illness. While many of these alterations appear to be reversible, a subset of patients experience persistent dysfunction involving the adrenal, thyroid, pituitary, pancreatic, or gonadal systems.

These findings highlight the need for ongoing endocrine follow-up in individuals recovering from COVID-19, especially those presenting with unexplained fatigue, metabolic changes, or reproductive disturbances. Early recognition and management of endocrine sequelae may improve quality of life and reduce the long-term burden on healthcare systems.

At the same time, the heterogeneity of available data underlines the importance of well-designed, longitudinal studies to clarify the mechanisms, prevalence, and prognosis of post-COVID endocrine syndromes. Integrating digital health technologies and tele-endocrinology into routine care may provide practical tools for early detection and personalized management of these disorders.

Ultimately, understanding how COVID-19 alters endocrine homeostasis is not only a question of endocrinology but also of broader public health. Addressing these long-term consequences requires coordinated, multidisciplinary care that bridges clinical practice, biomedical research, and digital innovation.

Acknowledgments: The author did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Conflict of Interest: The author declares no conflict of interest.

REFERENCES

1. Szczerbiński, Ł., Okruszko, M. A., Szabłowski, M., Sołomacha, S., Sowa, P., Kiszkiel, Ł., Gościk, J., Krętowski, A. J., Moniuszko-Malinowska, A., & Kamiński, K. (2023). *Long-term effects of COVID-19 on the endocrine system – A pilot case-control study. Frontiers in Endocrinology, 14,* 1192174. https://doi.org/10.3389/fendo.2023.1192174
2. Pal, R., Joshi, A., Bhadada, S. K., Banerjee, M., Vaikkakara, S., & Mukhopadhyay, S. (2022). *Endocrine follow-up during post-acute COVID-19: Practical recommendations based on available clinical evidence. Endocrine Practice, 28*(4), 425–432. https://doi.org/10.1016/j.eprac.2022.02.003
3. Clarke, S. A., Abbara, A., & Dhillo, W. S. (2022). *Impact of COVID-19 on the endocrine system: A mini-review. Endocrinology, 163*(1), bqab203. https://doi.org/10.1210/endocr/bqab203
4. Esmaeilzadeh, A., Elahi, R., Siahmansouri, A., Maleki, A. J., & Moradi, A. (2022). *Endocrine and metabolic complications of COVID-19: Lessons learned and future prospects. Journal of Molecular Endocrinology, 69*(3), R125–R150. https://doi.org/10.1530/JME-22-0036
5. Iosef, C., Matusa, A. M., Han, V. K. M., & Fraser, D. D. (2024). *Endocrine dysregulation in COVID-19: Molecular mechanisms and insights. Frontiers in Endocrinology, 15,* 1459724. https://doi.org/10.3389/fendo.2024.1459724
6. Panesar, A., Gharanei, P., Khovanova, N., Young, L., & Grammatopoulos, D. (2025). *Thyroid function during COVID-19 and post-COVID complications in adults: A systematic review. Frontiers in Endocrinology, 15,* 1477389. https://doi.org/10.3389/fendo.2024.1477389
7. Anbardar, N., Dixon, S. L., Munugoti, S., Gaddam, M., Kashfi, K., Kasulis, L., Messersmith, A. L., & Asadipooya, K. (2025). *Thyroid disorders and COVID-19: A comprehensive review of literature. Frontiers in Endocrinology, 16,* 1535169. https://doi.org/10.3389/fendo.2025.1535169
8. Nakano, Y., Sunada, N., Tokumasu, K., et al. (2024). *Occult endocrine disorders newly diagnosed in patients with post-COVID-19 symptoms. Scientific Reports, 14,* 5446. https://doi.org/10.1038/s41598-024-55526-3
9. Kanczkowski, W., Beuschlein, F., & Bornstein, S. R. (2022). *Is there a role for the adrenal glands in long COVID? Nature Reviews Endocrinology, 18,* 451–452. https://doi.org/10.1038/s41574-022-00700-8
10. Sahoo, S. K., Menon, J. C., Tripathy, N., Nayak, M., & Yadav, S. (2024). *Reversible central adrenal insufficiency in survivors of COVID-19: Results from a 24-month longitudinal study. Endocrine Connections, 13*(9), e240086. https://doi.org/10.1530/EC-24-0086
11. Porntharukchareon, T., Dechates, B., Sirisreetreerux, S., Therawit, P., & Tawinprai, K. (2024). *The existence of adrenal insufficiency in patients with COVID-19 pneumonia. Frontiers in Endocrinology, 15,* 1337652. https://doi.org/10.3389/fendo.2024.1337652
12. Alijotas-Reig, J., Anunciacion-Llunell, A., Esteve-Valverde, E., Morales-Pérez, S., Rivero-Santana, S., Trapé, J., González-García, L., Ruiz, D., Marques-Soares, J., & Miro-Mur, F. (2024). *Pituitary-adrenal axis and peripheral immune cell profile in long COVID. Biomedicines, 12*(3), 581. https://doi.org/10.3390/biomedicines12030581
13. Di Filippo, L., Franzese, V., Santoro, S., Doga, M., & Giustina, A. (2024). *Long COVID and pituitary dysfunctions: A bidirectional relationship? Pituitary, 27*(6), 955–969. https://doi.org/10.1007/s11102-024-01442-8
14. Taieb, A., Ben Haj Slama, N., Gorchane, A., Ben Abdelkrim, A., Garma, M., Ben Lasfar, N., Bellazreg, F., Debbabi, W., Hachfi, W., Chadli Chaieb, M., Zaouali, M., Letaief, A., & Ach, K. (2024). *Explaining long COVID: A pioneer cross-sectional study supporting the endocrine hypothesis. Journal of the Endocrine Society, 8*(3), bvae003. https://doi.org/10.1210/jendso/bvae003
15. Kim, S. H., Arora, I., Hsia, D. S., Knowler, W. C., LeBlanc, E., Mylonakis, E., Pratley, R., & Pittas, A. G. (2023). *New-onset diabetes after COVID-19. The Journal of Clinical Endocrinology & Metabolism, 108*(11), e1164–e1174. https://doi.org/10.1210/clinem/dgad284
16. Li, J., Li, Y., Wang, Z., Liu, N., He, L., & Zhang, H. (2023). *Increased risk of new-onset diabetes in patients with COVID-19: A systematic review and meta-analysis. Frontiers in Public Health, 11,* 1170156. https://doi.org/10.3389/fpubh.2023.1170156
17. He, X., Liu, C., Peng, J., et al. (2021). *COVID-19 induces new-onset insulin resistance and lipid metabolic dysregulation via regulation of secreted metabolic factors. Signal Transduction and Targeted Therapy, 6,* 427. https://doi.org/10.1038/s41392-021-00822-x
18. Anastácio, B. M., Monteiro, P. B., & Capelo, M. F. (2024). *The impact of COVID-19 on male reproductive health: A systematic review. JBRA Assisted Reproduction, 28*(3), 483–488. https://doi.org/10.5935/1518-0557.20240028

19. Alzahrani, M. A., Alkhani, K. O., Alassaf, A. M., Alorainy, J. I., Binsaleh, S., & Almannie, R. (2024). *Updates in the pathophysiology of COVID-19 infection in male reproductive and sexual health: A literature review. Frontiers in Endocrinology, 14,* 1226858. https://doi.org/10.3389/fendo.2023.1226858
20. Maybin, J. A., Walker, C., Watters, M., et al. (2025). *The potential bidirectional relationship between long COVID and menstruation. Nature Communications, 16,* 8187. https://doi.org/10.1038/s41467-025-62965-7
21. Koch, C. A. (2024). *Long COVID: Hormone imbalances and/or rather complex immune dysregulations? Journal of the Endocrine Society, 8*(5), bvae043. https://doi.org/10.1210/jendso/bvae043
22. Bansal, R., Gubbi, S., & Koch, C. A. (2022). *COVID-19 and chronic fatigue syndrome: An endocrine perspective. Journal of Clinical & Translational Endocrinology, 27,* 100284. https://doi.org/10.1016/j.jcte.2021.100284
23. SeyedAlinaghi, S., Yarmohammadi, S., Mirzapour, P., Dehghani, S., Ahmadi, S., Abbaspour, F., Pashaei, A., Molla, A., & Mehraeen, E. (2024). *A systematic review of telehealth applications in endocrinology. Telemedicine Reports, 5*(1), 269–289. https://doi.org/10.1089/tmr.2024.0032
24. Zupa, M., Hamm, M., Alexander, L., & Rosland, A. M. (2025). *Patient and clinician perspectives on the effectiveness of current telemedicine approaches in endocrinology care for type 2 diabetes: A qualitative study. JMIR Diabetes, 10,* e60765. https://doi.org/10.2196/60765
25. Puig-Domingo, M., Marazuela, M., & Giustina, A. (2020). *COVID-19 and endocrine diseases: A statement from the European Society of Endocrinology. Endocrine, 68*(1), 2–5. https://doi.org/10.1007/s12020-020-02294-5
26. Clarke, S. A., Abbara, A., & Dhillo, W. S. (2022). *COVID-19 and endocrine function. Endocrinology, 163*(1), bqab203. https://doi.org/10.1210/endocr/bqab203
27. Lundholm, M. D., Poku, C., Emanuele, N., Emanuele, M. A., & Lopez, N. (2020). *SARS-CoV-2 (COVID-19) and the endocrine system. Journal of the Endocrine Society, 4*(11), bvaa144. https://doi.org/10.1210/jendso/bvaa144