









Literature Review

Cabin Fever During Isolation Due to Covid-19: What Should We Do to Overcome it?

Grace Manuela Nurhadi¹ , Alya Shafira Hewiz¹, Jihan Nabila Pranjasdhina¹ , Fiqih Faizara Ustadi¹ , Fathiya Rahma Hermawan¹ , Almira Maharani¹ , Alfira Nailatul Izzah¹ , Muhammad Khafidin Al Alim¹ , Danty Puspitasari¹, Yunias Setiawati² 

¹Faculty of Medicine, Universitas Airlangga Surabaya, Indonesia

²Department of Psychiatry, Faculty of Medicine, Universitas Airlangga - Dr. Soetomo General Academic Hospital, Surabaya Indonesia

Abstracts

Received: April 18, 2024
Accepted : October 1, 2024
Published Online : November 1, 2024

You are free to:

Share — copy and redistribute the material in any medium or format

Adapt — remix, transform, and build upon the material for any purpose, even commercially.

The licensor cannot revoke these freedoms as long as you follow the license terms.

Correspondence Author:

Email: yunias.setiawati@gmail.com

Introduction: Cabin fever is described as some combination of irritability, moodiness, and depression due to isolation during COVID-19. Cabin fever may happen to anyone who has to isolate themselves at home because of the COVID-19 outbreak. We aim to identify the risk factor, the influence of hormone imbalance on mood changes, and how to deal with cabin fever during isolation due to COVID-19. People who are unable to connect with other people physically, unable to do activities that they used to do, unmotivated due to having no work, and anxious about finances due to lack of income may have a higher risk of cabin fever. **Review:** Social isolation experienced by people with cabin fever may result in hormonal imbalances that eventually affect their mood. This is due to the stress that comes from prolonged stay-at-home orders, which are thought to disturb the balance of the hormones in a person's body. Hormones which are able to cause mood changes are cortisol, thyroid, testosterone, estradiol, and progesterone. Cabin fever is not a recognized psychological condition. In this way, there is no standard treatment for cabin fever. **Conclusion:** Therefore, acceptance, reconciliation with oneself, coexistence and trust (intimate interactions, mindfulness, and positive self-shaping as self-control) are keys to overcome cabin fever.

Keywords: Cabin Fever, Isolation, COVID-19, Hormone Imbalance, Psychological wellbeing

Cite this as: Nurhadi. G. M., Hewiz. A. S., et al. "Cabin Fever During Isolation Due to Covid-19: What Should We Do to Overcome it?". *Jurnal Psikiatri Surabaya*, vol. 13, no. 2, pp.238-246, 2024. doi: [10.20473/jps.v13i2.30390](https://doi.org/10.20473/jps.v13i2.30390)

INTRODUCTION

Covid-19 along with SARS-CoV and MERS-CoV are types of Human Coronavirus (HCoV) that can cause acute respiratory diseases, can attack across species and are most rapidly transmitted between humans. According to WHO, SARS had 8,906 cases and 744 deaths as of January 2020, MERS cases were 2,519 and 866 deaths; and by the end of 2020, 79.2 million people were positive for Covid-19 and 1.7 million deaths were reported [1]. As the Covid-19 virus is highly infectious, preventive measures were taken to control the transmission of the virus, such as self-isolation, quarantine, stay-at home order, lockdown, and physical distancing [2, 3]. These methods are considered effective in reducing transmissions, yet its strict and long term implementation can cause risks of increased psychological disorders such as prolonged feeling of being confined and isolated [1]. This condition has its own term, namely cabin fever. Cabin fever is described as some combination of irritability, moodiness, boredom, depression, or feeling of dissatisfaction in response of confinement, bad weather, routine, isolation, or lack of stimulation [4]. Symptoms include being uncomfortable at home or in your room, restless or anxious, bored, angry, gets more emotional and tired quickly. Other non-dominant symptoms include frustration, nervousness, loneliness, difficulty concentrating and difficulty performing daily activities [1]. Studies reveal that mental health deteriorates with social isolation and quarantine; thus, should be further investigated in the context of the pandemic [5]. In the early pandemic, several research has been conducted on the fallout of social isolation adding to the burden of mental health around the world. A survey in United Kingdom that tracks the mood of around 2,000 Britons each week showed a significant rise in boredom and fear and a corresponding decrease in happiness during lockdown. A Kings' College study identified three groups reacting to life under lockdown as accepting (48%), suffering (44%) and

resisting (9%). Of those suffering, 93% reported anxiety and depression, 64% reported reduced or disrupted sleep, 34% reported constant thinking about coronavirus and 64% checking social media on coronavirus daily or more frequently. While the pandemic lockdown benefits in decreasing transmission, it is worth dwelling on the impact of solitary confinement in this case [5].

The cabin fever phenomenon has brought attention to researchers since its affecting most of the world's population due to the pandemic. Further study is needed as more people around the world are experiencing cabin fever and is affecting their mental health. This article aims to learn more about cabin fever, how to identify the risk factors, how it influences hormone imbalance on mood changes, and how to deal with cabin fever during isolation due to Covid-19. Hopefully, this study can help people understand more about cabin fever and improve their lifestyle.

DISCUSSION

What is cabin fever, etiology, risk factor

The pandemic of COVID-19 has brought changes in modern lifestyle. These changes are started by policies made to suppress transmission rate. Some of the policies are the closing of public buildings, physical distancing, self quarantine, and self isolation. These policies are proven to be effective in lowering transmission rate but also trigger social distance and isolation. Restricted and reduced social interaction and increased time spent isolated at home may cause cabin fever. Rather than a specific diagnosis that can be found in DSM V, cabin fever is a group of psychological symptoms caused by prolonged confinement at home [2].

First use of the term was to describe typhoid fever outbreak in poor community living in crowded cabins in Ireland. After the great famine, many irish emigrated to North America and had to quarantine in sheds, thus a new term "fever shed" arised. With the development of sanitary understanding and infectious disease eradication, cabin fever, as

a term, started to shift to psychological syndromes that emerge from winter isolation [5].

Cabin fever may happen to anybody who has to isolate themselves at home because of covid-19 outbreak. People who are unable to connect with other people physically, unable to do activities that they like, feeling burned out, unmotivated due to having little to no work, and anxious about finances due to lack of income may have higher risk of cabin fever [2].

While it is not a certain diagnosis, cabin fever should not be ignored. Cabin fever may lower quality of life. Moreover, people with mental disorders may get worse impact. Mood disturbance caused by cabin fever may trigger generalized anxiety, paranoia, obsessive compulsive disorder, depression, and, even worse, may lead to suicidal thought [2].

The influence of hormone imbalance on mood changes associated with cabin fever during COVID-19

The pandemic of COVID-19 has had a tremendous negative impact on communities worldwide. Because of the severity of the epidemic, local and even national lockdowns were implemented [6]. However, negative emotions have begun to spread as the virus' infectivity and danger grows and the unexpected nature of the situation and uncertainty about when the epidemic will be brought under control [7]. Because of the prolonged lockdown, thousands of people feel helpless, terrified, anxious, and depressed [8]. Furthermore, the pandemic has a significant impact on mental health, as evidenced by data released by the mental health charity Sane only seven weeks after the UK's lockdown, which showed a 200 percent increase in calls to their hotline [5]. This study by the mental health charity Sane, along with other comparable studies, suggests that the isolation felt behind closed doors contributes to cabin fever, a term that has gained popularity in recent years, according to a YouGov survey

conducted in 2020, which tracks the moods of over 2,000 Britons each week. During the lockdown, there was a significant increase in boredom and fear, as well as a significant drop in happiness [5]. Loneliness caused by social isolation is thought to stimulate the hypothalamic-pituitary axis, causing an increase in cortisol, a stress hormone. Individuals who have high levels of stress hormones are less likely to use adaptive coping strategies and are more likely to experience negative moods or emotions due to stress hormones [9].

Stress has been linked to adverse effects on physical health and has been linked to the onset of mental health disorders. Chronic or severe stress due to prolonged lockdown raises the risk of developing psychiatric disorders like depression and substance abuse [10]. Stress is also linked to increased hormone release from the hypothalamus-pituitary-adrenal axis (HPA-axis). The HPA axis, as the name implies, is a feedback loop that includes the hypothalamus, pituitary, and adrenal glands. In addition to these structures, the axis gets crucial regulatory input from the hippocampus, bed nucleus of the stria terminalis (BNST), amygdala, and paraventricular nuclei (PVN). When people are faced with mental or physical stress, the HPA axis is activated. The hypothalamus will secrete two hormones, corticotropin-releasing hormone (CRH) and arginine vasopressin (AVP), which work on the pituitary to promote the production of adrenocorticotropin hormone (ACTH) [11].

CRH, also known as corticotropin-releasing factor (CRF), is a 41-amino acid peptide foundation. ACTH then stimulates the adrenal glands to generate and secrete cortisol into the bloodstream [12]. Cortisol is the adrenal glucocorticoid stress hormone that is released in humans and other primates. Cortisol is a hormone that affects how an individual perceives and manages stress. Unmanaged chronic stress impairs a person's ability to produce and use cortisol, resulting in various mental in numerous central nervous system

regions and is the primary regulator of the mammalian stress response [13]. CRH binds to receptors on the pituitary gland that releases ACTH. ACTH is transported to the adrenal cortex via circulatory health problems. As a result, mood disorders such as depression, apathy, or anxiety may develop. Excess cortisol can inhibit the hypothalamus's activity, resulting in chemical messenger imbalances that affect sleep, appetite, sex, cognition, and other elements of one's life, all of which can mimic or contribute to a disease such as depression. Apart from secreting cortisol into the bloodstream, the adrenal glands also regulate the internal stress reaction, which helps regulate the external stress response. Adrenal glands may become dysfunctional when a person is under physical, mental, or any other type of personal stress. Many people deal with stress daily. However, if certain types of stress are endured for an extended period, they might have a negative effect on an individual's mental health [14].

Aside from the adrenal glands, the thyroid gland is also a critical endocrine gland in humans. These endocrine glands are chemical message systems and hormone feedback loops that can be affected in various ways and seriously impacting a person's mental health. The link between thyroid function and psychiatric disorders, particularly mood disorders, has long been established. Thyroid dysfunction can have a significant impact on mental health, including emotion and cognition. Thyroid hormones, both excess and insufficient, can cause mood abnormalities [15].

Multiple factors interact in the hypothalamic-pituitary-thyroid axis (HPT axis), including thyroid hormones, deiodinase enzymes, transporter proteins, and receptors. Thyroid hormone secretion is regulated by pituitary thyrotropin (TSH), which is stimulated by hypothalamic thyrotropin-releasing hormone (TRH) and inhibited by serum thyroid hormones. Twenty percent of triiodothyronine (T3) in the cerebral cortex is secreted directly by the thyroid, while the other

eighty percent is derived from local deiodination of thyroxine (T4). T4 is transported into the brain by transthyretin, a thyroid hormone transport protein synthesized by the choroid plexus and secreted into the CSF. Deiodination occurs in glial cells, and T4 must enter these cells via plasma membrane carrier proteins like OATP1C1 and MCT8 (MCT8). The former transports T4 and rT3, while the latter concentrates on T3. In glial cells, deiodinase enzyme type 2 converts T4 to T3, while deiodinase enzyme type 3 inactivates T4 to 3,3',5'-triiodothyronine (rT3). It also turns T3 into inactive T2. T3 acts by binding to thyroid hormone nuclear receptors (THR), of which THR- accounts for 70–80% of distribution in the adult brain. Thus, the HPT axis includes complex pathways, and some studies have linked impairment in its components to behavioural and mood changes [16]. When a person's thyroid gland is underperforming (producing insufficient T4 and/or T3), thyroid hormone does not reach the brain. This problem has the potential and frequently results in an increased prevalence of depression, anxiety, and other mental health disorders. For example, patients with thyroid diseases such as hyperthyroidism or hypothyroidism may have erratic and unpredictable mood changes [17]. Several thyroid abnormalities have been linked to mood disorders, particularly depression. Thyroid abnormalities are characterized by elevated T4 levels, low T3, elevated rT3, blunted TSH response to TRH, positive antithyroid antibodies, and elevated TRH concentrations in cerebrospinal fluid (CSF) [16]. Furthermore, lower CSF TTR levels have been reported in depressed patients than controls, resulting in "brain hypothyroidism" with normal peripheral thyroid hormones. A study also reported that high aggression scores are associated with a low T3/T4 ratio in suicide attempts [18].

Along with the two primary glands, many studies indicate that sex hormones significantly impact mood changes. The three main classes of sex steroids are 'androgens,' 'es-

trogens,' and 'progestogens,' with the most important human derivatives being 'testosterone' and 'estradiol,' respectively. Androgen, In both male and female rats, testosterone treatment reduces fear and anxiety. Males with low testosterone levels, also known as hypogonadism, can experience anxiety, depression, and irritability [19]. Age-related hypogonadism is a common feature of male ageing. When testosterone levels fall, depression test scores may rise. Exogenous testosterone may improve hypogonadal males' mood, and testosterone itself may be an antidepressant. Over half of circulating testosterone is contributed by the ovaries, either directly or indirectly. Ovarian function, like testosterone and estrogen, declines during menopause. Testosterone supplementation improves overall well-being and mood, indicating that testosterone influences mood in both men and women [20].

Aromatase, The enzyme aromatase, which converts testosterone and other C19 steroids to estradiol, complicates research on the effects of androgens and estrogens on mood and cognition. Animal studies suggest that estradiol may affect the serotonergic pathway in the brain, which is disrupted in depression. Estradiol stimulates gene expression of the 5-HT receptor and the serotonin transporter in the dorsal raphe nucleus of female rats, both of which are targeted by antidepressant medication containing selective serotonin reuptake inhibitors (SSRIs). This increases the density of 5-HT_{2a} receptors and SERTs in the forebrain regions of humans that regulate mood, cognition, and emotion. Castration reduces the expression of the 5-HT_{2a} receptor and the SERT gene in male rats. It cannot, however, be converted to estradiol by aromatase, unlike testosterone [21]. The expression of the 5-HT_{1a} receptor or SERT gene indicates that the effects of testosterone are due to aromatase's conversion of testosterone to estradiol. Many studies are divided on whether testosterone directly affects the CNS or is converted to estradiol and affects CNS function as a result of this conversion.

Further research is needed to determine the effect of testosterone on CNS function. Aromatase appears to affect mood and cognition, according to new research. Female aromatase knockout mice have more depressive-like behaviours. Polymorphisms encode aromatase in the *cyp19* gene are associated with female depression [22]. Psychoactive drugs such as estrogen and steroids such as ecstasy have been shown to alter brain aromatase activity. Animals have been studied for the effects of aromatization on the likelihood of aggression or components of aggression, such as hostility or irritation. As a result, aromatase activity may affect mood. Estrogens, estrogens generally have a positive effect on mood. Estrogen receptors can be found all over the brain. The primary endogenous mammalian estrogen is estradiol. Estradiol regulates neurotransmitter systems involved in depression, including serotonin, norepinephrine, dopamine, acetylcholine, GABA, and glutamate, and immediately affects neuronal excitability [23]. As previously stated, estradiol stimulates the expression of the 5-HT_{2a} receptor and the SERT gene in the dorsal raphe nucleus, both of which are targets of antidepressant SSRIs. This increases the density of 5-HT_{2a} receptors and SERTs in mood-regulating regions of the human forebrain. Estrogen increases dendritic branching and synapse formation in the hypothalamus and hippocampus during the rat estrous cycle, followed by dendritic pruning. Because the estrous cycle lasts only 3–4 days, these neuroarchitecture changes suggest synaptic remodelling in estrogen-induced mood regulation. As a result, estrogens can influence mood via a variety of neural mechanisms [21].

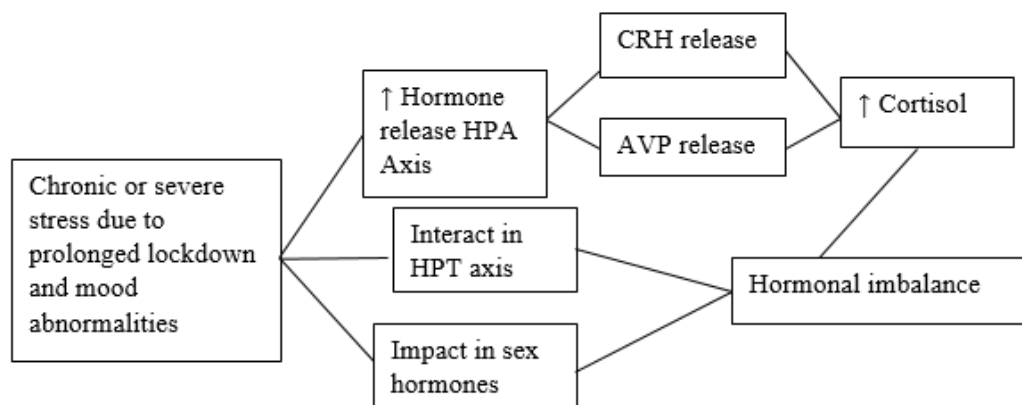
Anxiety is a fear cycle dysregulation. Estradiol has the ability to both help and hinders fear learning. The apparent contradictions may be explained by the antagonistic roles of different estradiol receptors. This relationship is influenced by aromatase activation, estrogen receptor expression and related cofactors, and social experience [24]. Proges-

terone, progesterone can affect mood either directly by interacting with progesterone receptors or indirectly by producing allopregnanolone's progesterone derivative. Allopregnanolone is thought to act in the brain as a positive modulator of the GABA-A receptor rather than the progesterone receptor. The hypothalamic-pituitary-adrenal (HPA) axis and other aspects of emotional arousal are coordinated by the corticotropin-releasing factor (CRF). In female rats, systemic progesterone or allopregnanolone treatment has anxiolytic effects [25].

During the luteal phase, allopregnanolone accumulates in the brain and plasma. Progesterone levels rise during the menstrual cycle's luteal phase, which adds to the mood swings associated with PMD. Premenstrual syndrome necessitates using ovarian steroids to begin, but women with PMS may also have decreased GABA-A receptor sensitivity. A comparable study discovered that postmenopausal women experience more

negative mood symptoms when given progesterone rather than estradiol or placebo, regardless of whether their circulating allopregnanolone levels are higher or lower than mid-luteal levels. These findings suggest that allopregnanolone levels are important for mood regulation [26].

While greater research is needed to understand the relationship between hormones and mental health thoroughly, prior research has established a positive correlation between hormones and well-being. As a result, maintaining homeostasis, or physiological balance is critical for good mental health. The central nervous system and the endocrine system work together to maintain homeostasis and respond to stimuli. A disruption in normal hormonal balance can accompany a variety of psychiatric diseases. An unbalanced hormonal system can impact the entire system, manifesting in both the body and the mind [26].



HPA: Hypothalamus-pituitary-adrenal; CRH: Corticotrophin releasing hormone; AVP: Arginine vasopressin; HPT: Hypothalamic-pituitary thyroid

How to Deal with Cabin Fever

Cabin fever isn't a recognized psychological condition. In this way, there is no standard treatment for them. Cabin fever that occurred during the COVID-19 epidemic was the result of changes in interpersonal relationships caused by self-isolation. Therefore, the main key to dealing with it is the patient himself and their support from the closest people such as family or close friends of the patient. The help and support of professional and so-

cial organizations tend to serve only an additional role. Several things that patients can do to treat cabin fever can be divided into 5 main parts, which are as follows [8]:

- Acceptance and coexistence: reconciliation with oneself

Patients need to reshape their biased, subjective cognition of the epidemic and treat the development of the epidemic reasonably and rationally, so as to avoid thoughts of catastrophe. Through cognitive reconstruction,

patients could try to understand the current tough situation from a position of self-care and self-love, thus giving them a positive meaning that allows them to accept reality calmly [27].

- Coexistence and trust: intimate interactions
In the face of a major crisis, strong family bonds and close social relations are essential sources of one’s security. Therefore, in situations that can cause high stress such as a pandemic, a comprehensive social support system should be established among families, peer groups, and communities [27].

- Stability and empathy: rational emotional participation

Real-time updates about epidemics often cause fear and anxiety in the society. Therefore, patients should limit their online time and avoid possible stressors, but they should also check information about the epidemic periodically and quantitatively and pay proper attention to the current epidemic situation, thus avoiding the anxiety and restlessness caused by excessive intake of negative information. In addition, positive guidance from the media is very important to create a positive and healthy anti-epidemic atmosphere [27].

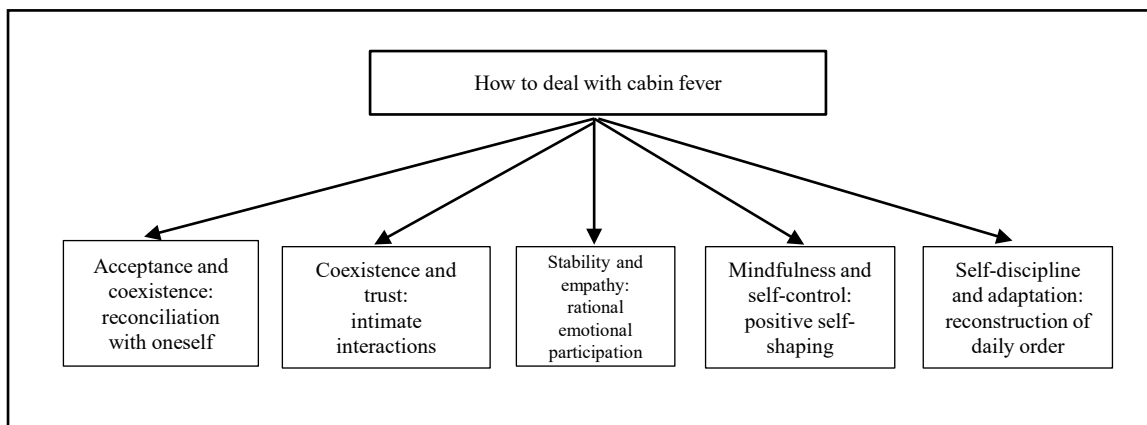
- Mindfulness and self-control: positive self-shaping

Patients must realize that COVID-19 is only a temporary risk in the long course of human history. They must believe that “everything will be fine” and must cultivate a sense of

hope for the future through a positive outlook and a sense of optimism. Through self-reflection, a person can discover the new values and meaning of life revealed by the epidemic, and they can use their rare alone time to complete their wish list of life goals that was previously unfinished due to the pressures of study and work [8].

Self-discipline and adaptation: reconstruction of daily order

During home quarantine, one’s work efficiency is greatly reduced, and the daily anxiety of aimless activity creates a sense of emptiness and self-deprecating identity. Due to a lack of self-control and a poor sense of boundaries, their daily life is disrupted and gradually spirals out of control. Therefore, reshaping an individual’s sense of boundaries, including space and time constraints, which supports the reconstruction of patients’ daily lives, will help them to maintain an orderly, healthy, and stable lifestyle [27]. In situations such as the COVID-19 outbreak, in addition to emphasizing on the psychological adaptation of patients, it is advisable for health policy makers to pay attention to psychological problems during social restrictions by creating programs or counseling services that can prevent cabin fever. Collaboration with universities and other institutions is also needed to actively publish information related to the disease and its treatment [1].



CONCLUSION

The stay-at-home order that is being implemented as one of the preventive measures to control the current COVID-19 pandemic has proved to be effective to lower the rates of the virus' transmission, but it might become a double-edged sword as it has the potential to inflict psychological disorders in people. One of those is cabin fever, a combination of psychological symptoms caused by prolonged confinement at home. Anybody who has to isolate themselves at home due to this pandemic is at risk of developing cabin fever. The risk factors for cabin fever are lack of physical contact with other people, inability to do one's favorite activities, burn out, decreased workload that may induce the feeling of being unmotivated, and financial problems that can cause anxiety.

Social isolation that is experienced by people with cabin fever may result in hormonal imbalances that will eventually affect their mood. This is due to the stress that comes from prolonged stay-at-home order which is thought to disturb the balance of the hormones in a person's body. Hormones with the ability to cause mood changes are cortisol, thyroid, testosterone, estradiol, and progesterone.

As of now, a standard treatment for cabin fever has yet to be found because it is considered as a group of symptoms, instead of as an actual disease. Nonetheless, there are still several methods that can be done to alleviate the suffering of people with cabin fever. The most important part of treating cabin fever is the patient themselves and the support that they receive from their loved ones. The help and support of professionals and social organizations might aid the patient's symptoms, but in the end, it all comes back to the patient and their loved ones because cabin fever is caused by the changes in interpersonal relationships that occurred due to the isolation.

ACKNOWLEDGEMENTS

None

CONFLICT OF INTEREST

The author declares that there is no conflict of interest

FUNDING

There are no financial support and sponsorship related to this work.

REFERENCES

- [1] M. Christo, L. D. Saraswati, A. Udiyono, and D. Sutiningsih, "Mixed Methods Systematic Review : Kejadian Cabin Fever Selama Pembatasan Sosial Saat Penyebaran Penyakit Sars, Mers Dan Covid-19," *J. Epidemiol. Kesehat. Komunitas*, vol. 6, no. 2, pp. 307–316, 2021.
- [2] R. D. Estacio, D. D. Lumibao, E. A. S. Reyes, and M. O. Avila, "Gender Difference in Self-reported Symptoms of Cabin Fever among Quezon City University Students during the Covid19 Pandemic," *Int. J. Sci. Res. Publ.*, vol. 10, no. 9, pp. 848–860, 2020, doi: [10.29322/ijsrp.10.09.2020.p105102](https://doi.org/10.29322/ijsrp.10.09.2020.p105102).
- [3] Z. Yan, "Unprecedented pandemic, unprecedented shift, and unprecedented opportunity," *Hum. Behav. Emerg. Technol.*, vol. 2, no. 2, pp. 110–112, 2020, doi: [10.1002/hbe2.192](https://doi.org/10.1002/hbe2.192).
- [4] P. C. Rosenblatt, R. M. Anderson, and P. A. Johnson, "The Meaning of 'Cabin Fever,'" *J. Soc. Psychol.*, vol. 123, no. 1, pp. 43–53, Jun. 1984, doi: [10.1080/00224545.1984.9924512](https://doi.org/10.1080/00224545.1984.9924512).
- [5] P. Crawford Jamie and O. Crawford, *Cabin Fever: Surviving Lockdown in the Coronavirus Pandemic*. Warrington: Emerald Publishing Limited, 2021.
- [6] A. O.- Guizzoa, A. Fogel, and N. E. R. Ho, "Effects of COVID-19-related stay-at-home order on neuropsychophysiological response to urban spaces: Beneficial role of exposure to nature?," *J. Environ. Psychol.*, vol. 75, 2021.
- [7] H. Su, L. Wang, Y. Li, H. Yu, and J. Zhang, "The correlation between mental health status, sleep quality, and inflammatory markers, virus negative conversion time among patients confirmed with 2019-nCoV



- during the COVID-19 outbreak in China,” *Med.*, vol. 27, no. June, pp. 1–7, 2018, doi: [10.1097/MD.00000000000026520](https://doi.org/10.1097/MD.00000000000026520).
- [8] M. S. John C. Smulian Sonja A. Rasmussen MD, “Multidisciplinary research priorities for the COVID-19 pandemic,” *Lancet Psychiatry*, vol. 7, no. 7, pp. 19–21, 2020, doi: [10.1192/bjp.2019.191.Andy](https://doi.org/10.1192/bjp.2019.191.Andy).
- [9] T. Field, S. Mines, S. Poling, M. Diego, D. Bendell, and C. Veazey, “Young, Alone, and Young Alone During a COVID-19 Lockdown,” *J. Ment. Heal. Clin. Psychol.*, vol. 4, no. 4, pp. 31–38, 2020, doi: [10.29245/2578-2959/2020/4.1219](https://doi.org/10.29245/2578-2959/2020/4.1219).
- [10] E. L. Knight, C. B. Christian, P. J. Morales, W. T. Harbaugh, U. Mayr, and P. H. Mehta, “Exogenous testosterone enhances cortisol and affective responses to social-evaluative stress in dominant men,” *Psychoneuroendocrinology*, vol. 85, pp. 151–157, 2017, doi: [10.1016/j.psyneuen.2017.08.014.Exogenous](https://doi.org/10.1016/j.psyneuen.2017.08.014.Exogenous).
- [11] J. P. Herman et al., “Regulation of the hypothalamic-pituitary-adrenocortical stress response,” vol. 6, no. 2, pp. 603–621, 2016, doi: [10.1002/cphy.c150015.Regulation](https://doi.org/10.1002/cphy.c150015.Regulation).
- [12] “Adrenal Glands,” The Johns Hopkins University, The Johns Hopkins Hospital, and Johns Hopkins Health System, 2021.
- [13] D. Richard and C. Lopez, “CRH,” *Handb. Biol. Act. Pept.*, pp. 1084–1088, 2013.
- [14] C. Tsigos, L. Kyrou, E. Kassi, and G. P. Chrousos, “Stress: Endocrine Physiology and Pathophysiology No Title,” MDText.com, Inc., 2000.
- [15] H. H. Loh, L. L. Lim, A. Yee, and H. S. Loh, “Association between subclinical hypothyroidism and depression: an updated systematic review and meta-analysis,” *BMC Psychiatry*, vol. 19, no. 1, pp. 1–10, 2019, doi: [10.1186/s12888-018-2006-2](https://doi.org/10.1186/s12888-018-2006-2).
- [16] M. P. Hage and S. T. Azar, “The link between thyroid function and depression,” *J. Thyroid Res.*, vol. 2012, 2012, doi: [10.1155/2012/590648](https://doi.org/10.1155/2012/590648).
- [17] M. Bathla, M. Singh, and P. Relan, “Prevalence of anxiety and depressive symptoms among patients with hypothyroidism,” *Indian J. Endocrinol. Metab.*, vol. 20, no. 4, pp. 468–474, 2016, doi: [10.4103/2230-8210.183476](https://doi.org/10.4103/2230-8210.183476).
- [18] G. M. Sullivan, J. J. Mann, M. A. Oquendo, E. S. Lo, T. B. Cooper, and J. M. Gorman, “Low Cerebrospinal Fluid Transthyretin Levels in Depression: Correlations with Suicidal Ideation and Low Serotonin Function,” *Biol. Psychiatry*, vol. 60, no. 5, pp. 500–506, 2006.
- [19] Brian Furman, “Testosterone,” Elsevier, 2018.
- [20] C. A. Shively, “Sex Hormones, Mood, and Cognition,” *Encycl. Behav. Neurosci.*, no. December, pp. 196–200, 2010, doi: [10.1016/B978-0-08-045396-5.00198-6](https://doi.org/10.1016/B978-0-08-045396-5.00198-6).
- [21] Sergio Della Sala, *Encyclopedia of Behavioral Neuroscience*, 2nd ed. United Kingdom: Elsevier Science, 2021.
- [22] S. R. Hammes and E. R. Levin, “Impact of estrogens in males and androgens in females,” *J. Clin. Invest.*, vol. 129, no. 5, pp. 1818–1826, 2019, doi: [10.1172/JCI125755](https://doi.org/10.1172/JCI125755).
- [23] J. P. Del Río, M. I. Allende, N. Molina, F. G. Serrano, S. Molina, and P. Vigil, “Steroid Hormones and Their Action in Women’s Brains: The Importance of Hormonal Balance,” *Front. Public Heal.*, vol. 6, no. May, pp. 1–15, 2018, doi: [10.3389/fpubh.2018.00141](https://doi.org/10.3389/fpubh.2018.00141).
- [24] A. P. Borrow and R.J. Handa, “Estrogen Receptors Modulation of Anxiety-Like Behavior,” *Vitam Horm.*, vol. 103, pp. 27–52, 2017, doi: [10.1016/bs.vh.2016.08.004](https://doi.org/10.1016/bs.vh.2016.08.004).
- [25] K. Tsutsui and S. Haraguchi, *Handbook of Hormones*. Academic Press, 2015.
- [26] C. Woodyard, “Exploring the therapeutic effects of yoga and its ability to increase quality of life,” *Int. J. Yoga*, vol. 4, no. 2, p. 49, 2011, doi: [10.4103/0973-6131.85485](https://doi.org/10.4103/0973-6131.85485).
- [27] R. Chen, Y. Bao, and Z. Li, “From being trapped to breaking through: manifestations of cabin fever in young people in response to COVID-19 and suggestions for adaptation,” *China J. Soc. Work*, vol. 14, no. 2, pp. 133–152, May 2021, doi: [10.1080/17525098.2021.1932542](https://doi.org/10.1080/17525098.2021.1932542).