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Evaluation of oxidative stress associated with altered Thiol/Disulphide homeostasis in cats with feline Coronavirus infection

Evaluación del estrés oxidativo asociado a la alteración de la homeostasis Tiol/Disulfuro en gatos con infección por Coronavirus felino

Melek Aydemir Hökelekli**, Selime Kübra Saka, Özcan Örs, Ali Evren Haydardedeoğlu, İlker Çamkerten, Hidayet Metin Erdoğan

Aksaray University, Faculty of Veterinary Medicine, Department of Internal Medicine, Aksaray, Türkiye.
**Corresponding author: melekaydemir09@gmail.com*

ABSTRACT

This study evaluated thiol/disulphide homeostasis and its relationship with oxidative stress in cats diagnosed with feline coronavirus infection. The study population consisted of 18 cats, including 11 feline coronavirus-positive animals and 7 clinically healthy controls. Serum concentrations of total thiol, native thiol, and disulphide were determined using a spectrophotometric assay. Compared with healthy controls, feline coronavirus-positive cats exhibited significantly lower feline coronavirus and native thiol levels, indicating a marked reduction in thiol-based antioxidant capacity. In parallel, a shift in redox balance toward disulphide formation was observed, as reflected by a pronounced decrease in the native thiol /feline coronavirus ratio. These alterations suggest depletion of the thiol pool under conditions of increased oxidative burden. Overall, disruption of thiol/disulphide homeostasis appears to be closely associated with oxidative stress during feline coronavirus infection and may contribute to disease pathogenesis. The present findings provide insight into the biochemical mechanisms underlying oxidative damage in feline coronavirus-infected cats and support the potential utility of thiol/disulphide parameters as early indicators of oxidative stress, with possible implications for future antioxidant-based therapeutic approaches.

Key words: Feline coronavirus; thiol/disulphide homeostasis; oxidative stress; cat

RESUMEN

Este estudio evaluó la homeostasis tiol/disulfuro y su relación con el estrés oxidativo en gatos diagnosticados con infección por coronavirus felino. La población del estudio estuvo compuesta por 18 gatos, incluidos 11 animales positivos para coronavirus felino y 7 controles clínicamente sanos. Las concentraciones séricas de tiol total, tiol nativo y disulfuro se determinaron mediante un método espectrofotométrico. En comparación con los controles sanos, los gatos coronavirus felino-positivos presentaron concentraciones significativamente más bajas de tiol total y tiol nativo, lo que indica una reducción marcada de la capacidad antioxidante basada en tioles. De manera paralela, se observó un desplazamiento del equilibrio redox hacia la formación de disulfuros, reflejado por una disminución pronunciada de la relación tiol nativo/tiol total. Estas alteraciones sugieren una depleción del pool de tioles bajo condiciones de aumento de la carga oxidativa. En conjunto, la alteración de la homeostasis tiol/disulfuro parece estar estrechamente asociada con el estrés oxidativo durante la infección por coronavirus felino y podría contribuir a la patogénesis de la enfermedad. Los hallazgos del presente estudio aportan información relevante sobre los mecanismos bioquímicos subyacentes al daño oxidativo en gatos infectados con coronavirus felino y respaldan la posible utilidad de los parámetros tiol/disulfuro como indicadores tempranos de estrés oxidativo, con potenciales implicaciones para futuras estrategias terapéuticas basadas en antioxidantes.

Palabras clave: Coronavirus felino; homeostasis tiol/disulfuro; estrés oxidativo; gato

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INTRODUCTION

Feline coronavirus (FCoV) is an enveloped RNA virus with a high prevalence among domestic cats and a frequent association with asymptomatic or mild enteric infections. However, in a limited proportion of infected animals, viral mutation may occur, resulting in the development of feline infectious peritonitis (FIP)—a progressive and often fatal disease particularly in multi-cat households, with incidence varying considerably between countries [1, 2, 3].

Accumulating evidence indicates that FCoV infection is accompanied by increased production of reactive oxygen species (ROS), largely driven by macrophage activation, pro-inflammatory cytokine release, and mitochondrial dysfunction. These processes collectively promote oxidative stress and contribute to cellular injury. Cats appear to be particularly susceptible to oxidative damage owing to distinctive characteristics of erythrocyte metabolism and splenic function, which render their redox balance relatively fragile compared with that of other species [4, 5, 6].

Oxidative stress is generally defined as a disturbance in the balance between oxidant generation and antioxidant defense mechanisms in favor of oxidants and is widely recognized as a key factor in the pathogenesis of viral infections [7, 8, 9]. In FCoV infection, excessive ROS production has the potential to induce oxidative damage to lipids, proteins, and nucleic acids, thereby compromising cellular integrity. Moreover, inadequate antioxidant responses under these conditions may amplify inflammatory cascades and accelerate disease progression [10, 11, 12].

At the molecular level, thiol groups constitute a major component of endogenous antioxidant defense. The sulfhydryl (–SH) groups present in proteins are capable of directly scavenging free radicals. Under oxidative conditions, thiol groups undergo reversible oxidation to form disulphide (–S-S–) bonds, generating a dynamic redox system that plays a critical role in maintaining cellular homeostasis. This process, referred to as thiol/disulphide homeostasis (TDH), has been proposed as a sensitive indicator of both antioxidant capacity and oxidative load [13, 14].

Physiologically, a tightly controlled equilibrium exists between thiol and disulphide pools. Increased oxidative stress disrupts this balance, leading to depletion of reduced thiols, relative elevation of disulphide forms, and a shift of redox status toward an oxidant-dominant state [14]. Such alterations may result in membrane damage, enzyme inactivation, and impaired intracellular signaling. Consequently, TDH-related indices-including disulphide/total thiol (DsTT), disulphide/native thiol (DsNT), and native thiol/total thiol (NT/TT) ratios-have been suggested as sensitive biochemical markers for assessing oxidative injury [12, 13]. In viral infections associated with heightened oxidative burden, including FCoV, disturbances in TDH may reflect the intensity of inflammatory responses and the extent of tissue damage. Therefore, evaluation of thiol/disulphide balance offers a valuable approach for clarifying the contribution of oxidative stress to the pathogenesis of FCoV infection [15, 16, 17]. To the best of our knowledge, no information with regads to thiol/disulphide homeostasis in cats with FCoV exist in the literature.

The present study therefor aimed to investigate alterations in thiol/disulphide homeostasis as an indicator of inflammation-associated oxidative stress in cats diagnosed with FCoV infection.

MATERIALS AND METHODS

Study design and animals

This study was carried out on domestic cats (*Felis catus*) admitted to the Teaching Hospital of the Faculty of Veterinary Medicine, Aksaray University. The study population comprised of 18 cats, including 11 animals diagnosed as FCoV-positive on rapid test and 7 clinically healthy cats that served as the control group.

The felines included in the patient group exhibited non-specific and non-localized clinical signs suggestive of FCoV infection, including fever, loss of appetite, lethargy, weight loss, vomiting, diarrhea, dehydration, and anemia. A comprehensive physical examination was conducted on all subjects. Although abdominal tenderness was detected in some cases, clinical findings were mostly limited to mild enteric symptoms. These findings are consistent with the non-specific clinical picture reported in FCoV infection [18, 19].

A commercial rapid immunochromatographic antibody test kit (Speed™ Trio FeLV/FIV/Corona, Virbac, France) was used to confirm the presence of FCoV infection. According to the performance characteristics reported by the manufacturer, the test's clinical sensitivity compared to the Reverse Transcription-Polymerase Chain Reaction (RT-PCR) method is stated as 92.86%, clinical specificity as 98.11%, and diagnostic accuracy as 97.01%. Additionally, the test's reported detection limit is 1.97×10^4 $TCID_{50} \cdot mL^{-1}$ [20, 21].

However, given the limitations of rapid diagnostic tests in distinguishing active infection from past infection, it is imperative to interpret test results in conjunction with clinical findings [22]. Consequently, the absence of confirmatory diagnostic methods (e.g., molecular analyses) is regarded as a substantial constraint of the study.

Serum biochemical analyses were performed using the FUJIFILM DRI-CHEM NX600 automatic analyzer (Fujifilm Corp., Japan). The albumin/globulin (A/G) ratio, calculated from albumin and globulin concentrations, was considered in the evaluation of systemic inflammation and changes in protein distribution. In this study, cats with an A/G ratio ≤ 0.8 were designated as cases that potentially exhibited biochemical inflammatory changes. However, it was acknowledged that this threshold value did not serve as a diagnostic indicator for a particular disease [23].

The animals included in the control group were determined to be clinically healthy, exhibited no signs of systemic disease during physical examination, had an A/G ratio > 0.8, and tested negative for FCoV in the Speed™ Trio FeLV/FIV/Corona test.

Sample collection and processing

Approximately 0.5 mL of blood was obtained from the cephalic vein of each cat and collected into sterile gel-containing vacuum tubes. Blood samples were centrifuged (LC–04B, China) at 3000 × g

for 10 min, after which serum was separated and stored at −80°C (DF−590, Türkiye) until further analysis.

Determination of thiol/disulphide homeostasis

Serum thiol/disulphide parameters were assessed using a commercially available assay kit (Rel Assay Diagnostics, Relassay, Türkiye) based on a spectrophotometric method, in accordance with the procedure previously described by Erel and Neşelioğlu [12]. Serum concentrations of total thiol (TT), native thiol (NT), and disulphide were determined. Following these measurements, thiol/disulphide-related indices were calculated using established equations [12, 24].

- $Disulphide\ (\mu mol{\cdot}L^{-1}) = (Total\ thiol - Native\ thiol) / 2$
- $Disulphide/Total\ thiol\ (\%) = (Disulphide \times 100) / Total\ thiol$
- $Disulphide/Native\ thiol\ (\%) = (Disulphide \times 100) / Native\ thiol$
- $Native\ thiol/Total\ thiol\ (\%) = (Native\ thiol \times 100) / Total\ thiol$

Statistical analysis

All data are presented as mean ± standard deviation. The distribution of variables was assessed for normality using the Shapiro-Wilk test. Differences between FCoV-positive cats and healthy control animals were evaluated using Student's *t*-test. Associations between measured parameters were examined by Pearson's correlation analysis. Statistical significance was defined as $P<0.05$.

RESULTS AND DISCUSSION

In the healthy control group, mean serum TT concentration was 374.83 μmol·L^{-1}, whereas NT levels averaged 266.17 μmol·L^{-1}, yielding an NT/TT ratio of 70.18%. In cats with confirmed FCoV infection, a marked deterioration in thiol status was evident. Mean TT and NT concentrations declined to 256.40 μmol·L^{-1} and 128.40 μmol·L^{-1}, respectively, and the NT/TT ratio decreased to 49.34% (FIG. 1). Comparative analysis demonstrated that serum TT, NT, and NT/TT ratios were significantly lower in FCoV-positive cats than in healthy controls ($P<0.05$).

All these findings indicate a pronounced impairment of thiol-based antioxidant defense mechanisms during FCoV infection, together with a shift of the redox balance toward an oxidant-dominant state. Similar alterations in redox homeostasis have been reported in various viral infections, in which excessive production of ROS contributes to depletion of endogenous antioxidant reserves [2, 7, 8, 25].

A strong positive correlation was identified between serum TT and NT concentrations in both FCoV-positive and healthy cats ($r = 0.85$, $P<0.05$) (FIG. 1).

This association indicates that total and native thiol pools respond in a parallel manner to oxidative challenges and, when evaluated together, provide a reliable reflection of systemic redox status. Considering the pivotal role of thiols in ROS scavenging and maintenance of redox equilibrium [24], the concurrent reduction of TT and NT further supports the presence of increased oxidative stress and diminished antioxidant capacity in cats affected by FCoV infection.

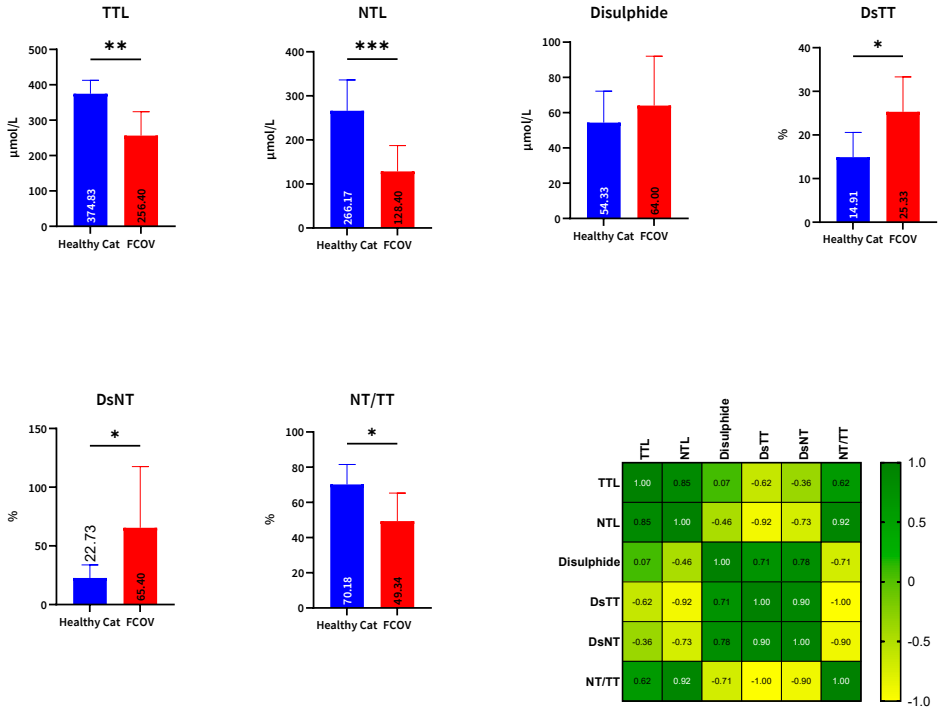

FIGURE 1. Thiol/disulphide parameters in healthy and FCoV-positive cats: total thiol (TT), native thiol (NT), and disulphide levels; ratios of disulphide/total thiol (DsTT, %), disulphide/native thiol (DsNT, %), and native thiol/total thiol (NT/TT, %). *P*<0.05

Comparable relationships between disturbed thiol/disulphide balance and systemic oxidative stress have also been documented in human inflammatory and autoimmune disorders [26, 27].

Mean serum disulphide concentrations were 54.33 µmol/L in the healthy control group and 64.00 µmol/L in cats with FCoV infection, and no statistically significant difference was observed between groups. In contrast, evaluation of disulphide-related ratios revealed pronounced alterations. In healthy cats, the DsTT and DsNT ratios were 14.91 and 22.73%, respectively, whereas markedly higher values were detected in FCoV-positive cats, reaching 25.33 and 65.40%. Both DsTT and DsNT ratios were significantly increased in the FCoV-positive group compared with controls ($P<0.05$, FIG. 1). Similar elevations in disulphide-related indices have been reported in infectious and inflammatory conditions in both human and veterinary medicine, underscoring the sensitivity of thiol/disulphide parameters in reflecting oxidative imbalance [25, 27, 28].

The observation that disulphide-related ratios increased despite the absence of a statistically significant elevation in absolute disulphide concentrations indicates that thiol consumption predominates during the acute phase of FCoV infection. Under conditions of pronounced oxidative stress, thiol groups are rapidly oxidized to form disulphide bonds; however, this process appears to be driven primarily by depletion of native thiol reserves rather than by proportional accumulation of disulphide products [12, 13].

Thiols play a central role in maintaining cellular integrity through direct interaction with free radicals, yet excessive generation of ROS leads to a rapid shift in redox equilibrium toward disulphide formation, a process characterized by limited reversibility [13, 29]. Accordingly, alterations in TDH provide particularly informative insight into the direction and severity of redox imbalance during the acute stage of infection, as demonstrated in both human and veterinary disease models [26, 30].

As oxidative burden intensifies, the buffering capacity of thiol pools becomes progressively exhausted, thereby facilitating tissue injury and amplification of inflammatory damage [31, 32, 33]. In this context, Yeşilırmak *et al.* [34] reported a strong association between disruption of thiol/disulphide balance and disease severity in inflammatory skin disorders.

These findings support the interpretation that depletion of thiol reserves similarly occurs during FCoV infection. Consistent with this view, reduced thiol concentrations have been shown to reflect sustained inflammatory stress in post-COVID–19 patients [35]. Moreover, increased ischemia-modified albumin levels together with a shift of thiol/disulphide balance toward oxidative stress have been described in ovine toxoplasmosis, further underscoring the relevance of these parameters across species and disease conditions [36].

Feline coronavirus infection is known to induce pronounced oxidative responses, largely mediated by macrophage activation and subsequent cytokine release [2]. Enhanced production of tumor necrosis factor–α, nitric oxide (NO), and hydrogen peroxide during infection augments ROS generation, thereby accelerating oxidation of thiol groups and contributing to depletion of antioxidant reserves [37, 38].

Experimental studies of feline viral infections have consistently demonstrated suppression of major antioxidant enzymes, including superoxide dismutase, catalase, and glutathione peroxidase, in parallel with increased malondialdehyde (MDA) concentrations, a well-established marker of lipid peroxidation [39, 40, 41]. In agreement with these observations, Kayar *et al*. [11] reported elevated MDA and NO levels in FCoV-positive cats. By focusing on TDH, the present study highlights a redox parameter that appears to be more sensitive and responsive at earlier stages of oxidative stress than conventional oxidative biomarkers [10, 11].

The pronounced reduction in the NT/TT ratio observed in FCoV-positive cats reflects a clear shift of the redox environment toward an oxidant-favored state. This ratio represents the proportion of reduced thiols within the TT pool; under conditions of elevated ROS production, reduced thiols are rapidly converted into disulphide forms, resulting in diminished antioxidant capacity and early predominance of disulphide formation within the thiol/disulphide system [12, 13].

The strong positive correlation between NT and TT levels ($r = 0.85$) further indicates that these parameters function in concert as reliable indicators of systemic oxidative stress (FIG. 1). Similar shifts toward disulphide dominance have been associated with increased oxidative burden in both veterinary and human studies [26, 27, 29, 30, 38]. Therefore, the NT/TT ratio may be considered a sensitive and early biomarker of oxidative stress, with potential clinical relevance for monitoring disease progression and prognosis in cats with FCoV infection.

CONCLUSION

The present study has demonstrated that there is a significant disruption in thiol/disulphide homeostasis in cats infected with FCoV. These changes may be associated with increased oxidative stress. The findings, which included a decrease in total and native thiol levels, along with a redox balance shifted towards disulphide formation, suggest a possible reduction in antioxidant defense capacity and an increase in oxidative load during infection. The findings suggest that thiol/disulphide parameters may serve as sensitive biochemical markers for evaluating oxidative changes occurring during FCoV infection.

However, the cases were not classified according to clinical severity, long-term follow-up was not performed, and the different clinical forms of FCoV infection were not evaluated. Consequently, it is not feasible to make direct inferences about the relationship between TDH and disease severity or potential clinical progression. This should be regarded as a substantial constraint of the study. The results of this study suggest that larger sample groups and prospective studies will contribute to a clearer understanding of the clinical significance of the findings.

Implications

The results of the present study demonstrate that TDH is substantially disrupted in cats with FCoV infection and is closely linked to elevated oxidative stress. Thiol/disulphide-related parameters may therefore serve as practical biochemical indicators for the assessment and clinical monitoring of oxidative stress in affected animals. In addition, these findings offer a biochemical rationale for future studies exploring antioxidant-based supportive

therapeutic approaches aimed at correcting redox imbalance in FCoV infections.

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Conflict of interests

The authors of this study declare that there is no conflict of interest with the publication of this manuscript.

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