Pengaruh waktu pemaparan hipoksia sistemik kontinu terhadap kadar glutation tereduksi (GSH) paru pada tikus = The influence of exposure time to continuous systemic hypoxia on lung reduced glutathione (GSH) level in rats

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Abstrak

ABSTRAK
Latar Belakang: Glutation tereduksi (GSH) adalah antioksidan endogen nonenzimatik utama di paru dan saluran pernapasan. GSH mengoksidasi spesi oksigen reaktif (ROS) untuk mencegah terjadinya kerusakan oksidatif, sehingga GSH menjadi salah satu parameter pengukuran derajat stres oksidatif. Hipoksia sistemik kontinu telah diketahui menyebabkan pembentukan ROS dan kerusakan oksidatif. Oleh karena itu, penelitian ini bertujuan mengetahui pengaruh waktu paparan hipoksia sistemik kontinu terhadap pembentukan ROS di jaringan paru, yang direpresentasikan melalui kadar GSH. Metode: Sampel paru didapat dari tikus Sprague-Dawley jantan berusia 6-8 minggu dengan berat badan 150-200 g, yang telah terpapar kondisi normoxia (kontrol) atau hipoksia sistemik kontinuselama hari. Kemudian, kadar GSH diukur dari ekstrak jaringan paru. Hasil: Data analisis dengan ANOVA mengindikasikan adanya perbedaan bermakna antara kadar GSH paru terhadap perbedaan waktu pemaparan hipoksia sistemik kontinu Perbandingan post hoc LSD memperlihatkan bahwa dibutuhkan pemaparan hipoksia setidaknya 5 hari untuk menimbulkan efek, ditunjukkan dengan adanya penurunan bermakna kadar GSH pada kelompok hipoksia 5 hari dan 7 hari Namun, paparan hipoksia selama kurang dari atau sama dengan 3 hari tidak berpengaruh signifikan terhadap kadar GSH. Kemudian, uji korelasi Pearson menunjukkan adanya hubungan berbanding terbalik yang sangat kuat antara waktu pemaparan hipoksia terhadap kadar GSH paru Kesimpulan: Waktu pemaparan hipoksia sistemik kontinu mempengaruhi kadar GSH paru secara berbanding terbalik, di mana kadar GSH paru semakin menurun seiring dengan semakin bertambahnya waktu paparan hipoksia
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ABSTRACT
Background: Reduced-glutathione (GSH) is a major endogenous nonenzymatic antioxidant in the lung and airway system. GSH oxidizes reactive oxygen species (ROS) to prevent oxidative damage. Hence, GSH is considered one of the parameters for measuring the degree of ROS-induced oxidative stress. Continuous systemic hypoxia has been known to cause ROS formation and oxidative damage. Consequently, this research attempted to see the effect of exposure time to continuous systemic hypoxia to ROS formation in the lung as reflected by GSH level. Methods: Lung samples were collected from 6-8 weeks old male Sprague-Dawley rats weighing 150-200g, previously exposed to normoxic environment (control) or continuous systemic hypoxia (days. Afterwards, GSH level was measured from lung extracts. Results: Data analysis using ANOVA indicated a significant difference in lung GSH level upon different exposure times to continuous systemic hypoxia Post hoc LSD comparisons revealed that hypoxic exposure should be of at least 5 days to yield an effect, as shown by significantly reduced GSH level in hypoxic groups of 5 days . Meanwhile, hypoxic exposure for 3 days or less did not significantly affect GSH level. Further Pearsons correlation analysis demonstrated a very strong negative relationship between hypoxic exposure times and

lung GSH level Conclusion: The exposure times to continuous systemic hypoxia were inversely proportional to lung GSH level, in which lung GSH level decreased as the exposure time was increased.