Analisis ekspresi lncRNA MALAT1 pada penderita periodontitis dengan obesitas = Analysis of lncRNA MALAT1 expression in periodontitis patients with Obesity

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Abstrak

Latar Belakang: Periodontitis adalah penyakit inflamasi kronis yang ditandai oleh pelepasan sitokin proinflamasi seperti TNF- dan IL-6. Obesitas meningkatkan peradangan sistemik melalui sekresi adipokin, yang memperburuk kerusakan jaringan periodontal. Kedua kondisi, periodontitis dan obesitas, menginduksi proses inflamasi dan gangguan stres oksidatif yang dipicu oleh sitokin dan hormon proinflamasi yang dilepaskan oleh jaringan adiposa. Variasi genetik dapat menyebabkan perubahan dalam mekanisme kerja sistem imun, termasuk melalui regulasi epigenetik, seperti peran RNA non-coding (lncRNA). lncRNA yaitu MALAT1 berperan dalam regulasi inflamasi dengan memodulasi ekspresi gen melalui jalur epigenetik, seperti aktivasi NF-B. MALAT1 juga memengaruhi migrasi sel imun, sehingga memperkuat respon inflamasi pada kedua kondisi tersebut. Namun, penelitian tentang hubungan antara lncRNA MALAT1, periodontitis, dan obesitas, khususnya di Indonesia, masih sangat terbatas. Tujuan: Mendapatkan perbedaan ekspresi lncRNA MALAT1 di setiap tingkat keparahan status periodontitis pada individu dengan dan tanpa obesitas. Metode: Sampel usapan mukosa bukal diperoleh dari individu dengan periodontitis-obesitas sebanyak 20 dan individu tanpa obesitas sebanyak 10. Selanjutnya, dilakukan ekstraksi RNA, penghitugan konsentrasi RNA, sintesis cDNA, dan ekspresi lncRNA MALAT1 dan referensi Gen GAPDH diuji menggunakan Real-time PCR. Hasil: Tidak ditemukan perbedaan signifikan ekspresi relatif lncRNA MALAT1 antara individu periodontitis ringan (p=0,950), sedang (p=0,376), berat (p=0,129)-obesitas dibandingkan dengan individu non-obesitas. Kesimpulan: Ekspresi relatif lncRNA MALAT1 lebih tinggi pada individu periodontitis – obesitas dibandingkan dengan individu tanpa obesitas.
.....Background: Periodontitis is a chronic inflammatory disease characterized by the release of pro-inflammatory cytokines such as TNF- and IL-6. Obesity increases systemic inflammation through the secretion of adipokines, which exacerbate periodontal tissue damage. Both conditions, periodontitis and obesity, induce inflammatory processes and oxidative stress disorders triggered by cytokines and pro-inflammatory hormones released by adipose tissue. Genetic variations can cause alterations in immune system mechanisms, including through epigenetic regulation, such as the role of non-coding RNA (lncRNA). The lncRNA MALAT1 plays a role in regulating inflammation by modulating gene expression via epigenetic pathways, such as NF-B activation. MALAT1 also affects immune cell migration, thereby amplifying the inflammatory response in both conditions. However, research on the relationship between lncRNA MALAT1, periodontitis, and obesity, particularly in Indonesia, remains very limited. Objective: To prove the existence of differences in lncRNA MALAT1 expression at each level of periodontitis severity in individuals with and without obesity. Methods: Buccal mucosal swab samples were collected from 20 individuals with periodontitis-obesity and 10 individuals without obesity. RNA extraction, RNA concentration measurement, cDNA synthesis, and the expression of lncRNA MALAT1 and reference GAPDH gene were analyzed using Real-time PCR. Results: No significant differences were found in the relative expression of lncRNA MALAT1 between individuals with mild (p=0.950), moderate (p=0.376), and

severe (p=0.129) periodontitis-obesity compared to non-obese individuals. Conclusion: The relative expression of lncRNA MALAT1 was higher in individuals with periodontitis- obesity compared to non-obese individuals.