Abstract

Lung inflammation resulting from oxidant/antioxidant imbalance is a common feature of many lung diseases. In particular, the role of enzymes regulated by the NF-E2-related factor 2 (Nrf2) transcription factor has recently received increased attention. Among these antioxidant genes, the glutathione S-transferase mu 1 (*GSTM1*) has been most extensively characterized since it has a null polymorphism which is highly prevalent in the population and associated with increased risk of inflammatory lung disease. Present evidence suggests that *GSTM1* acts through interactions with other genes and environmental factors, especially air pollutants. Here, we review *GSTM1* gene expression and regulation and summarize the findings from epidemiological, clinical, animal and *in vitro* studies on the role played by GSTM1 in lung inflammation. We discuss limitations in the existing knowledge base and future perspectives and evaluate the potential of pharmacologic and genetic manipulation of the *GSTM1* gene to modulate pulmonary inflammatory responses.

Key words: *GSTM1* polymorphism; air pollution; lung inflammation; NrF2