fectiveness of homozygous val compared to ile isomorph in antioxidant defense and the protective role of val isomorph against respiratory diseases has been evaluated both in vitro and in human studies\(^7\)\(^\text{-}\)\(^9\). As it has been previously examined, in an adult Caucasian population\(^18\), there is a strong correlation between the different polymorphisms of GSTP1 and bronchial hyperresponsiveness (BHR) and asthma, since the homozygous val isomorph was associated with lower incidence of the disease and reduced risk for airway inflammation.

Manar et al recently examined the genetic predisposition to BPD in the GSTP1 polymorphisms in a population of African-American children\(^20\). The main outcome of that study, comparing a group of premature infants with a group of term/near term controls, was a significant association between ile allele and the development of BPD. The presence of homozygous val isomorph was more effective against BPD, as the controls expressed that isomorph in a higher rate compared to neonates with BPD (23.5% vs 5.9%). However, a subsequent study performed by Cooke et al, could not demonstrate any association between GSTP1 polymorphisms and the development of BPD in a group of Caucasian preterm neonates\(^21\). Homozygous val isomorph was equally identified in preterms with BPD and preterms without BPD (43% vs 36%). This discordance between the two studies could potentially be explained by the differences in genotyping methodology between term and preterm controls that were used in each study.

Similarly to the study of Cooke et al\(^21\), our data could not confirm the previously described protective role of the val allele against BPD, or any association between the GSTP1 genotyping and the severity of BPD.

These differences between the present and previous studies could possibly be explained by the small number of all data sets. Moreover, considering that GSTP1 genotyping distribution is known to vary between ethnic groups, differences in the ethnic composition of the study groups, between the present and previous studies could reflect the differences in the genotyping distribution.

**Limitations**

The main limitation of our study was the relatively small number of cases.

**Conclusions**

In summary, our results could not validate any association between the GSTP1 polymorphism and the development of BPD and/or the severity of the disease.

**Conflict of Interest**

The authors have no financial disclosures or conflicts of interest.

**References**


**Table 3:** GST - P1 polymorphisms (n, %) of the study group which consists of preterm infants (< 32 weeks GA) with bronchopulmonary dysplasia and of the control groups which consists of preterm infants (< 32 weeks GA) without bronchopulmonary dysplasia and term infants (37–42 weeks GA) without bronchopulmonary dysplasia.

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Preterms with BPD</th>
<th>Preterms no BPD</th>
<th>Terms</th>
</tr>
</thead>
<tbody>
<tr>
<td>ile/ile</td>
<td>78 (77)</td>
<td>23 (82)</td>
<td>23 (70)</td>
<td>32 (78)</td>
</tr>
<tr>
<td>ile/val</td>
<td>18 (17)</td>
<td>4 (14)</td>
<td>6 (18)</td>
<td>8 (20 )</td>
</tr>
<tr>
<td>val/val</td>
<td>6 (6)</td>
<td>1 (4)</td>
<td>4 (12)</td>
<td>1 (2)</td>
</tr>
</tbody>
</table>

GSTP1: Glutathione S Transferase P1, BPD: Bronchopulmonary Dysplasia.

**Table 4:** GST - P1 polymorphisms (n, %) of the study group which consists of preterm infants (< 32 weeks GA) with bronchopulmonary dysplasia (BPD), according to BPD severity.

<table>
<thead>
<tr>
<th></th>
<th>Overall</th>
<th>Mild</th>
<th>Moderate</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>ile/ile</td>
<td>23 (82)</td>
<td>4 (80)</td>
<td>8 (100)</td>
<td>11 (73)</td>
</tr>
<tr>
<td>ile/val</td>
<td>4 (14)</td>
<td>1 (20)</td>
<td>0 (0)</td>
<td>3 (20)</td>
</tr>
<tr>
<td>val/val</td>
<td>1 (4)</td>
<td>0 (0)</td>
<td>0 (0)</td>
<td>1 (7)</td>
</tr>
</tbody>
</table>

GSTP1: Glutathione S Transferase P1, BPD: Bronchopulmonary Dysplasia.