The statistical analyses revealed insignificant difference regarding the distribution of the GSTP1 polymorphisms between the case group and the control subgroups.

The correlation of the BPD severity and the GSTP1 polymorphisms was also examined in the group of the preterms that developed BPD (Table 4). The homozygous ile isomorph was predominantly identified: in 4 neonates with mild BPD, 8 with moderate and 11 with severe BPD. However, no correlation between the distribution of the GSTP1 polymorphisms and the BPD severity was found.

**Discussion**

In the present study, we examined a potential association of the GSTP1 polymorphisms and the development of BPD, comparing a group of preterms with BPD with a group of preterms without BPD and a group of healthy terms. Our analyses, however, revealed significant difference neither between cases and controls, nor between the group of premature infants with or without BPD.

As demonstrated in previous studies, the molecular basis of BPD is well established and has been identified in surfactant proteins (SP), immune system proteins and antioxidant defenses. The polymorphisms in the intron 4 of the SP-B can modify the severity of the BPD while SP-A1 polymorphism 6A6 can be also an independent risk factor for the disease. Furthermore, the severity of BPD has been associated with the variability of genes encoding Toll-like receptor’s (TLR), especially TLR5 and TLR4.

Regarding the GSTP1 polymorphic alleles, the ef-