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of P-glycoprotein on the surface of a chronic myeloid leukaemia cell line. P-glycoprotein (P-gp), an ATP-dependent transporter, is expressed by a wide variety of cell types but its function is not fully understood. To investigate the role of P-gp in chronic myeloid leukaemia (CML), we analysed the cell surface expression and function of P-gp on the K562 cell line, a model of chronic myeloid leukaemia.

Using flow cytometry and Western blots, we measured P-gp expression on K562 cells at different stages of the leukaemic process. P-gp was consistently expressed by > 60% of the K562 cells and increased with time in culture. The level of P-gp was highest on the more immature K562 cell line, followed by the more mature K562 cell line. K562 cells were resistant to the cytotoxic effects of vinblastine, whereas inhibition of P-gp using either verapamil or Fumetinib increased drug-induced apoptosis. CML cells may function as a tumour drug pump as the level of P-gp increased with time in culture. P-gp may protect leukaemic cells from apoptosis thereby contributing to the leukaemic process. Helicobacter pylori colonization of the gastric mucosa: a clinical and histological correlation in children and adolescents. Fifty-four children with chronic abdominal pain and an endoscopic finding of chronic gastritis were studied in a prospective way using gastric biopsy specimens. The results revealed that 71% (n = 38) were infected by Helicobacter pylori. This was associated with an older age (P less than .001) and a more advanced histological damage to the gastric mucosa, compared to children without H. pylori infection. In 45% of H. pylori-positive children, the associated histological changes were graded I (i.e., minimal damage), while in the remaining 55% the damage was graded II or III (i.e., mild-to-severe lesions). These findings suggest that children with endoscopic findings of chronic gastritis associated with H. pylori colonization have more severe histological damage than children without H. pylori infection. In some cases, H. pylori-associated inflammation does not necessarily

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