lings, catalase activity peaked at 50 μM $HgCl_2$. On the other hand, 15-day-old seedlings showed the highest level of catalase activity when grown at 250 μM $HgCl_2$ (Fig. 3B). At the concentrations of 50 and 500 μM $HgCl_2$, catalase activity of 15-day-old seedlings was, respectively, 30% and 51% lower than that of the control.

Ascorbate peroxidase activity varied only in accordance with Hg concentration in the substrate (Fig. 3C). A higher inhibition was observed at concentrations of 250 and 500 μM $HgCl_2$, both for 10 or 15 days.

4. Discussion

Mercury is inadvertently added to soils in fertilizer, limestone, natural gypsum, phosphogypsum, manure (especially of marine origin), sewage sludge, etc., and intentionally added in fungicides containing Hg (Andersson, 1979). Mercury concentrations in limestone are generally <20 μg kg^{-1}, whereas animal manures may have concentrations of the order of 100 μg kg^{-1}. Occasionally, values of up to 100 mg kg^{-1} are reported (Steinnes, 1990).

The changes observed in the growth of cucumber seedlings were consistent with the results obtained at low Hg concentrations in tomatoes (Cho and Park, 2000). Suszcynsky and Shann (1995) showed that inhibition of root and shoot growth occurred at 1.0 μg mL^{-1} Hg and above, with very limited tissue damage at higher levels of treatment. Also, Hg-induced root damage may have serious consequences for nutrient and water supply to above ground plant parts (Godbold and Huttermann, 1986).

Our results indicated that higher concentrations of Hg increased the production of root dry weight (Fig. 2E). This may be explained by mercury-induced formation of gathering in the vegetable tissue. These changes are consistent with the hypothesis that Hg induces an abnormal proliferation of root cells. This also has been observed in studies with cadmium in plants (Arduini et al., 2004).

On the other hand, higher concentrations of Hg dramatically reduced shoot biomass (Fig. 2F). The increase in root fresh weight at lower Hg-concentrations (50 μM $HgCl_2$) might be caused by the hormetic effect. Calabrese (1999) observed a similar effect in *Mentha piperita* to the synthetic plant growth inhibitor phosfon. Growth hormesis represents an overcompensation due to a disruption in homeostasis that has been described in relation to different factors, such as several organic and inorganic chemicals, Al, and the amelioration of a latent deficiency of an essential element or stimulation of defense reactions leading to a general activation of metabolism (Barceló and Poschenrieder, 2002; Calabrese and Blain, 2005).

Results of the present study indicate a continuous increase in the content of Hg in the roots and cotyledons of cucumber seedlings with the increase of the external concentration of Hg. Seedlings of cucumber accumulated a significantly higher Hg content in the roots when compared to the cotyledons, which is in agreement with the findings of other authors (Greger et al., 2005). Hg accumulation in the root system indicates that roots serve as a partial barrier to the transport of Hg to shoots (Cavallini et al., 1999). In this study, a portion of Hg could have been simply sequestered away by epidermal cell walls or cuticles, though in response to the effects of Hg on seedlings, we can suggest that Hg was, in fact, taken by tissue cells.

Zang and Tyerman (1999) reports that Hg is known to inhibit water uptake via aquaporins on plasma membranes in higher plants, which could explain the detrimental effect of higher concentrations of Hg on the fresh weight of seedlings. It is interesting to note that, contrarily, root dry weight significantly increased.

The decreased chlorophyll content observed in our study corroborates with other reports (Cho and Park, 2000). $HgCl_2$ (0.5–500 μM) caused a time-dependent and concentration-dependent decline in chlorophyll content (Fig. 2A) in the cotyledons. In plants, Hg ions may substitute metal ions in photosynthetic pigments, causing a decrease in photosynthesis rates (Xylander et al., 1996). Exposure to Hg was reported to induce a loss of K, Mg, Mn and an accumulation of Fe (Doening, 2000). Several studies have shown that Hg in the substrate decreased the levels of photosynthetic pigment chlorophylls and carotenoids at a prolonged duration of exposure. It also strongly inhibits the photosynthetic electron transport chain, where photosystem II (PS II) is the most sensitive target (Bernier et al., 1993; Bernier and Carpentier, 1995). Assche and Clijsters (1990) reported that lipid peroxidation causes membrane impairment and leakage, and suggested that the reduction in chlorophyll content in the presence of metals is caused by an inhibition of chlorophyll biosynthesis.

Heavy metal toxicity is believed to induce the production of reactive oxygen species (ROS) and may result in significant damage to cellular constituents. Membrane lipids and proteins are especially prone to attack by free radicals, considered to be reliable indicators of oxidative stress in plants (Halliwell and Gutteridge, 1993). It is known that high concentrations of metals in plants can interfere with physiologically important functions, can cause an imbalance of nutrients and have detrimental effects on the synthesis and functioning of biologically important compounds, such as enzymes, vitamins, hormones, etc. (Vangronsveld and Clijsters, 1994).

The peroxidation of lipids probably starts with the hydroxyl radical. Scavengers of $OH^{\cdot}$ do not inhibit the process, and Fe^{2+} bound to the membrane and exposed to the attack of H_2O_2 generates $OH^{\cdot}$ formed will react locally and immediately with the lipids in the membrane (Halliwell and Gutteridge, 1999). Therefore, $O_2^{\cdot}$, H_2O_2 and other ROS such as the hydroxyl radical ($OH^{\cdot}$) could be responsible for Hg-induced membrane damage. Active oxygen species bring about the peroxidation of membrane lipids, which leads to membrane damage (Scandalios, 1993). Since lipid peroxidation is the symptom most easily ascribed to oxidative damage (Zhang and Kirkam, 1996), it is often used as an indicator of increased oxidative damage (Halliwell, 1987).