HOW LEAD LOSES ITS TOXICITY TO PLANTS

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ABSTRACT

This paper is a brief review of the problem of lead in the environment, particularly constitutional tolerance to lead about which little is known. Taking *Allium* cepa L. roots as a model it has been shown that after an initial phase in which lead is toxic to cells, defence processes appear with the results that lead is no longer poisonous. The lead which penetrates into the root symplast is detoxified in vacuoles, cell walls and dictiosomal vesicles. Initial cells of the meristem (quescent centre) which play a basic role in the root regeneration processes are protected against lead penetration. This surprising result is in agreement with the absence of any symptoms of lead poisoning in plants growing in natural conditions, and suggests that there is a defence mechanism specific only for plant cells.

KEY WORDS: *Allium cepa* L. (onion), lead tolerance, lead detoxification, lead pathways, root tip.

INTRODUCTION

Lead is added to petrol in the form of alkyl compounds. The most prominent effect of these lead compounds results mainly in damage to the mitotic spindle (c-mitosis) and the inhibition of cytokinesis (Ahleberg et al. 1972; Mukherji and Maitra, 1976; Röderer, 1979).

When the environment is polluted inorganic lead compouds are more abundant and arise mainly from exhaust-pipe fumes, as lead halides i.e. bromides and chlorides (Bernhardt et al., 1982). Inorganic lead compounds are also emitted from zinc and lead smelting works.

Lead from exhaust-fume aerosols can either enter the above-ground parts of plants directly through the leaves, or be taken up through the root system from the soil (Zimdahl, 1976; Peterson, 1978). The amount of lead in plants depends on many factors such as: the distance from an emission, wind direction, the season of the year, car traffic intensity and various soil chemical properties. Hence concentrations range from one to several thousand (3000) mg Pb kg^{-1} d.w. (Cannon and Bowles, 1962; Kloke and Leh, 1969; Tesiuk, 1969; Rajns, 1971; Smith, 1972; Kazimierczakowa and Rams, 1974; Laaksovirta et al., 1976; Grodziska, 1979; Wheeler and Rolfe, 1979).

The large amounts of lead which plants accumulate harmlessly may on the one hand be regarded as beneficial, because in this way much lead is withdrawn from circulation in the biosphere (for example it may be locked away in trees growing close to crowded motor-ways). On the other hand, the accumulation of lead in food plants can be dangerous for humans and animals because it is incorporated in their trophic chain (for example through vegetables and fodder plants which grow close to crowded motor-ways) (Wierzbicka and Antosiewicz 1993).

The aim of this paper is present the results of the study which show multipolar effects of lead on plants – and particularly illustrate how lead loses its toxicity to plant cells.

LABORATORY AND ENVIRONMENTAL STUDIES OF THE INFLUENCE OF LEAD ON PLANTS

There is a widely accepted belief among research workers that lead is very toxic to plants. Yet if we look more closely at the problem, we encounter incongruity between the results of laboratory studies and observations out in the field.

Toxic effects of lead on many essential life processes in plants have been shown repeatedly in many laboratory studies. In particular, lead strongly inhibits:

- plant growth (Mukherji and Maitra, 1977; Woźny and Jerczyńska, 1991)
- root elongation (Hasset et al. 1976; Świeboda, 1976; Przymusiński and Woźny, 1985)
- germination of seeds (Woźny et al. 1982)
- development of germinating seeds (Lane and Martin, 1977; Woźny et al. 1982)
- cell division (Levan, 1945; Biesele, 1958; Mukherji and Maitra, 1976; Röderer and Schnepf, 1977; Przymusiński and Woźny, 1985)
- photosynthesis (Bazzaz et al. 1975; Bazzaz and Govindjee, 1974; Poskuta et al. 1987)
- transpiration (Rolfe and Bazzaz, 1975)
- chlorophyll production (Mukherji and Maitra, 1976; Rivkin, 1979; Wrischer and Meglaj, 1980)
- etioplast development (Wrischer and Meglaj, 1980)
- lamellar organization in chloroplasts (Rebechini and Hanzely, 1974).

Also, the presence of lead electron-opaque deposits in very many cellular compartments has been revealed by electron microscopy in studies of lead localization in different plant cells. In particular:

- in cell walls (Gullvåg et al. 1974; Ophus and Gullvåg, 1974; Sharpe and Denny, 1976; Lane and Martin, 1982; Woźny et al., 1982; Książek et al., 1984; Przymusiński and Woźny, 1985)
- in nuclei (Skaar et al., 1973; Gullvåg et al., 1974; Ophus and Gullvåg, 1974; Lane and Martin, 1982)
- in the cytosol (Romanenko and Salyaev, 1978)
- near the plasmalemma (Gullvåg et al., 1974; Malone et al., 1974; Lane and Martin, 1982; Przymusiński and Woźny, 1985)
- in vacuoles (Gullvåg et al., 1974; Ophus and Gullvåg, 1974; Romanenko and Salyaev, 1978; Krupińska, 1981; Lane and Martin, 1982; Woźny et al., 1982; Książek et al., 1984; Przymusiński and Woźny, 1985)
- in lipid bodies (Lane and Martin, 1982)
- in chloroplasts (Sharpe and Denny, 1976)
- in peroxisomes, mitochondria, microbodies (Ophus and Gullvåg, 1974; Krupińska, 1981)
- in dictyosomes (Malone et al., 1974; Woźny et al., 1982; Książek et al., 1984)
- in endoplasmic reticulum and nuclear envelopes (Woźny et al., 1982; Książek et al., 1984).

In the light of the results given above, what is surprising and seemingly incongruous, are the findings of environmental studies. There are actually no symptoms of lead poisoing observed in plants growing under natural conditions, even near crowded motor-ways. This is true even when such plants are found to contain considerable amounts (some hundred mg Pb kg^{-1}) in their tissues – Zimdahl, 1976; Koeppe, 1981.

Steenken (1973a) provided confirmation by exposing plants experimentally to car exhaust gases containing lead. The plants were not damaged even though they contained 350 mg Pb kg^{-1} d.w. in their tissues. It appeared that the threshold concentration of lead causing the first damage ranged from 500 to 1000 mg Pb kg^{-1} d.w. depending on the species. A considerable inhibition of growth and yield did not appear until 5000 mg Pb kg^{-1} d.w. was exceeded. Steenken's studies explained that plant damage under natural conditions was absent because lead level did not come anywhere near the lowest concentrations which cause initial damage. This suggests that in most experimental studies plants were treated with lead doses which were too high compared to ambient doses, and because of this, it was possible to find some toxic influence of lead on most life processes. The reason for this approach was the pre-assumption that lead acted toxically on plants. If it is not so, we face another important question.

Since there is no apparent plant response to lead under ambient conditions, what allows the plant to tolerante a considerable amount of lead in its tissues?

THE PATHWAYS FOR LEAD IN PLANTS

The plant epidermis is a strong (but not perfect – Lepp, Dollard, 1974) barrier to lead penetration into the tissues; most lead is absorbed within the external cell wall and cuticle (Steenken, 1973b; Arvik and Zimdahl, 1974; Godzik et al., 1979). A considerable part of this lead can be removed by washing (Havre and Underdal, 1976).

Roots have an ability to take up and accumulate large amounts of lead (Raghi-Atri, 1978). Simultaneously they limit lead translocation to the above-ground parts (Jones et al., 1973). This slight translocation (Sieghardt, 1981; 1984) takes place through the root xylem (Lane and Martin, 1977).

DEFENCE AGAINST LEAD IN PLANTS

The tolerance to heavy metals found in plants growing in heavily contaminated areas, such as on metalliferous mine spoils and naturally enriched soils in the vicinity of mineral veins, is a separate branch of research, mainly ecological (Thurman and Hardwick, 1988; Baker et al., 1988). According to Baker (1987), "there are two basic strategies of tolerance: metal exclusion, whereby metal uptake and transport is restricted and metal accumulation, where there is no such restriction and metals are accumulated in a detoxified form. Detoxification may results from cell-wall binding, active pumping of ions into vacuoles, complexing by organic acids and possibly by specific metals-binding proteins. Other, more subtle, properties such as enzymic adaptation and effects on membrane permeability can also be detected".

The problems concerning the tolerance to heavy metals of plants growing in heavily polluted conditions have been covered in several comprehensive reviews (e.g. Baker, 1987; Tomsett and Thurman, 1988; Tukendorf, 1989; Antosiewicz, 1992; Woźny and Krzesowska, 1993 and in the book by Shaw, 1990) and it is unnecessary to cover them here.

What we will deal with is the problem of defence against lead seen in the plants which are not most common in environment. Some authors have until now questioned the presence of constitutional metal tolerance, in other words congenital tolerance and not the one triggered by heavy metals from the environment (Reeves and Baker, 1984; Tomset and Thurman, 1988; Antosiewicz, 1992).

In the remainder of this paper I am going to present a synthesis of several years' research which provides new information about constitutional tolerance to lead in onion (*Allium cepa* L.) roots. This study show that lead loses its toxicity to onion cells after several hours.

The root tip (meristematic zone of root – Fig. 1) is a very good object for this type of study (Grant, 1982; Wierzbicka and Antosiewicz, 1988). The meristematic cells react fairly quickly to the agent since they have high metabolic activity and a short cell cycle (15.5 hours). This reaction is very important for the whole plant because the meristematic cells fulfil a vital role in growth and development.

During all my experiments adventitious roots of onion were treated with aqueous solution of lead salts ($PbCl_2$ or $Pb(NO_3)_2$) in non-lethal doses (usually 2.5 mg dm^{-3} Pb^{2+}) from 5 minutes to 30 hours. Lead localization was examined using the following methods:

- histochemical – with sodium rhodizonate
- autoradiographic – with 210Pb
- transmission electron microscopy
- X-ray microanalysis,
- electron microscopic autoradiography – with 210Pb,
- and Timm's sulphide – silver cytochemical method.

How Pb^{2+} influences root growth, mitotic disturbances, mitotic activity, level of RNA, DNA and protein synthesis (autoradiographically with 3H-thymidine, 3H-leucine and 3H-uridine) was also investigated.

1. Lead pathways in Allium cepa roots

Taking account of all the research findings which have been obtained so far in our laboratory (Wierzbicka, 1984 a,b; 1986; 1987 a, b, c; 1988; 1989; 1990; 1994) we can recon-

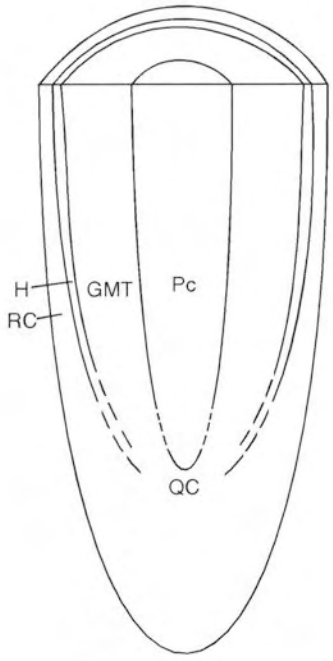


Fig. 1. Schematic diagram of *Allium cepa* root tip. RC – root cap, H – meristematic hypodermis, GMT – ground meristematic tissue, Pc – procambium.

struct the following sequence of events observed in onion roots over the whole period of treatment with lead.

Lead is quickly taken up from the outer solution (Figs 17, 18) with the same intensity along the whole of the root. The longer was the treatment with lead the larger was the accumulation. The process of lead uptake is very intense during the first 4 hours, and is most intense during the first 15 minutes, of incubation:

Radial lead penetration through the meristematic zone (Fig. 1) takes about 80 min.

- The first 5 minutes – lead deposits on the root surfance.
- The first 10-15 minutes – lead penetrates apoplastically (along the cell walls) two layers of the root cap cells radially reaching protoderm cells and meristematic cells of the hypodermis, where it passes into the symplast (lead penetrates into the cells through the plasmalemma).
- The first 60 minutes – lead penetrates through the subsequent ground meristematic tissue layers at the rate of one layer per 5 minutes. Its distribution within the ground meristematic tissue is uneven. In layers 1-6 it spreads evenly, but in the deeper ones (7-10) the lead deposits are first seen in the intercellular spaces and middle lamellae, and later in cell walls above the plasmalemma.
- The first 70-85 minutes – lead is present in procambium. From then on it is present in almost all root tip tissues.
- The first 2-6 hours – the amount of lead in cell walls increases gradually. From the 3rd h of incubation lead is also present in vacuoles. From then on both the number of vacuoles with lead and the amount of lead deposited in each vacuole increases gradually.

Gradually intensifying lead toxicity appears at that time, inhibiting the root growth and mitotic activity (Fig. 2). This effect coincides with the appearance of numerous c-mitoses (Figs 2, 4-6) and binucleate cells (Fig. 3) as there are no spindle microtubules.

In the cells which are in mitosis it is cytokinesis that is damaged at the start of incubation. In the cells which are in the G2 phase at the start of incubation, the mitosis is damaged, because tubulin required for the formation of the mitotic spindle is synthesized during this phase (Olszewska et al., 1990). This type of damage becomes apparent in the form of c-mitoses only after several hours of treatment. The cell cycle of the cells damaged by lead in such a way is three times longer.

- The first 6-12 hours – the strongest action of lead is observed, and is indicated by the lowest mitotic activity (Fig. 2). These symptoms indicate there is by now toxic action of the lead within the root symplast.
- The first 12-24 hours – metabolic activity in roots is observed to return to the control level (Fig. 2), although the total amount of lead in the roots keeps increasing. The root growth is more intensive, the mitotic activity increases (Fig. 2), and cell divisions are not disturbed (Fig. 2). This unexpected response of cells implies a reduction of lead toxicity to meristematic cells during the incubation of roots in solutions of lead salts.

During this period of incubation a large amount of lead is accumulated and inactivated in the cell walls (Figs 7-10), vacuoles (Figs 11-16) and dictiosomal vesicles. A large vacuole system is formed from the endoplasmic reticulum. Digested large parts of the protoplast appear, followed by the formation of large autophagic vacuoles containing lead deposits. Lead is gradually transported from the apoplast to the above-ground parts.

It seems that the reappearance of the metabolic activity in roots, while they are being treated with lead salts (Fig. 2), is caused by lead detoxification processes in the cells (Figs 7-16).

2. The barriers to lead translocation in Allium cepa roots

The root is a barrier which limits lead translocation to the overground parts of a plant, as discussed above. Studies with *Allium cepa* have revealed at least four barriers encountered by lead on its way apoplasically and symplastically (Wierzbicka, 1987a).

The barriers to apoplastic transport are the layers of protoderm and hypodermic meristematic cells, and the layer of endodermis in the mature root zone.

The barriers to apoplastic and symplastic transport are the central zone, which comprises the quiescent centre in the root meristem and the central part of the root cap (Figs 17-20).

The cells of the deepest ground meristematic tissue layers seem to act as a barrier which keeps the lead away from the procambium (Fig. 18).

A very interesting result is an almost complete absence of lead in the middle part of the root cap and in the quiescent centre (Figs 19, 20). It is not known why lead ions penetrate neither through the apoplast nor the symplast of this central zone of the root tip. Absence of lead in the quiescent centre might indicate the existence of a mechanism which protects the root initial cells against damage. It is well known that the initial cells have a significant role in the processes of root regeneration.

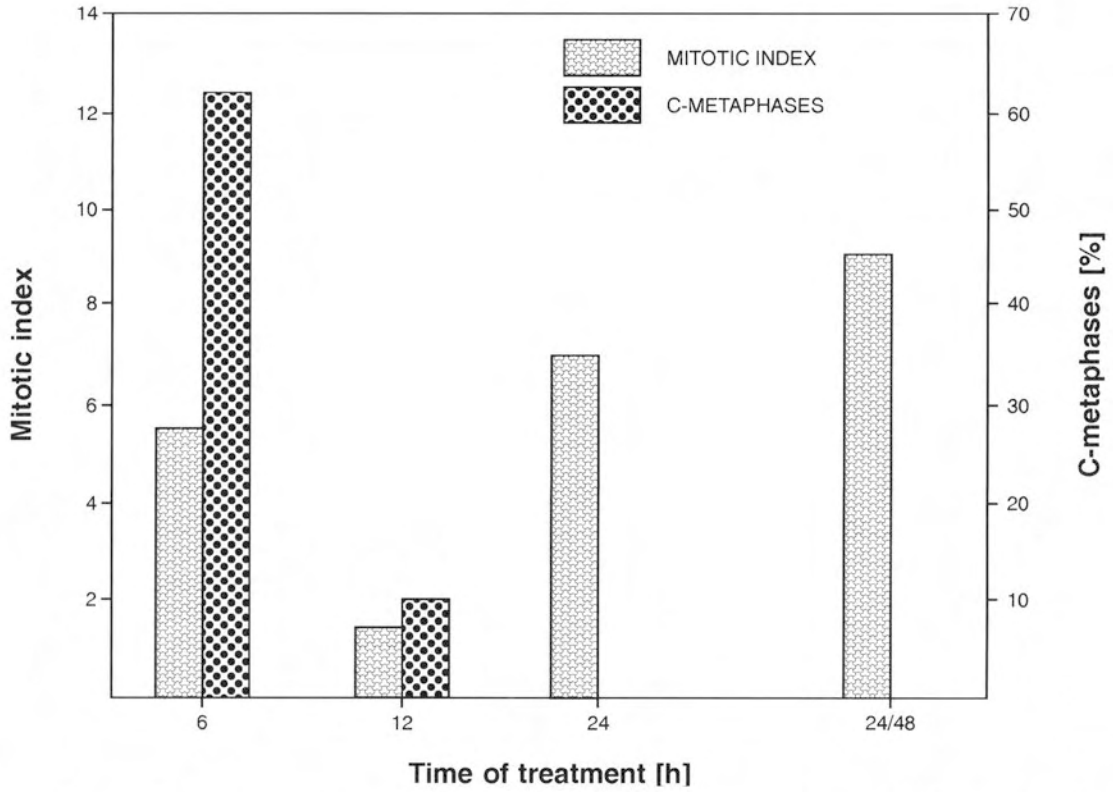


Fig. 2. Mitotic activity and mitotic disturbances (c-metaphases of onion roots during treatment with lead chloride (2.5 mg dm-3 Pb^{2+}) for 24 h and recovery for 48 h. Control mitotic index – average 8.1; control c- methaphases from 0.0 to 0.03%

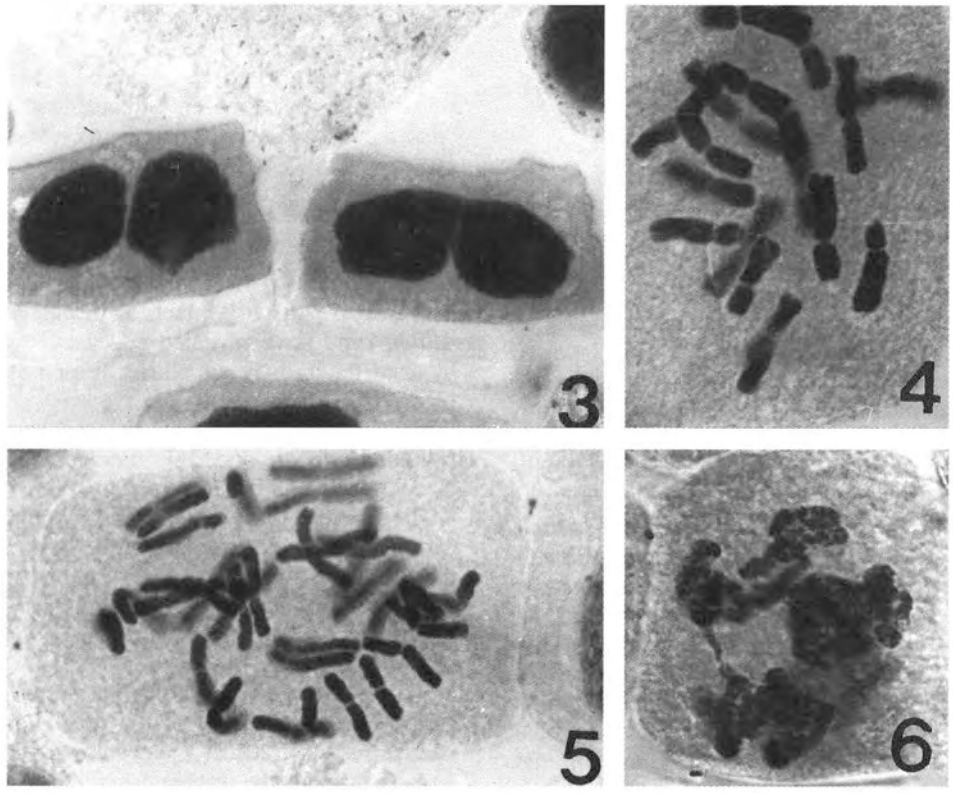
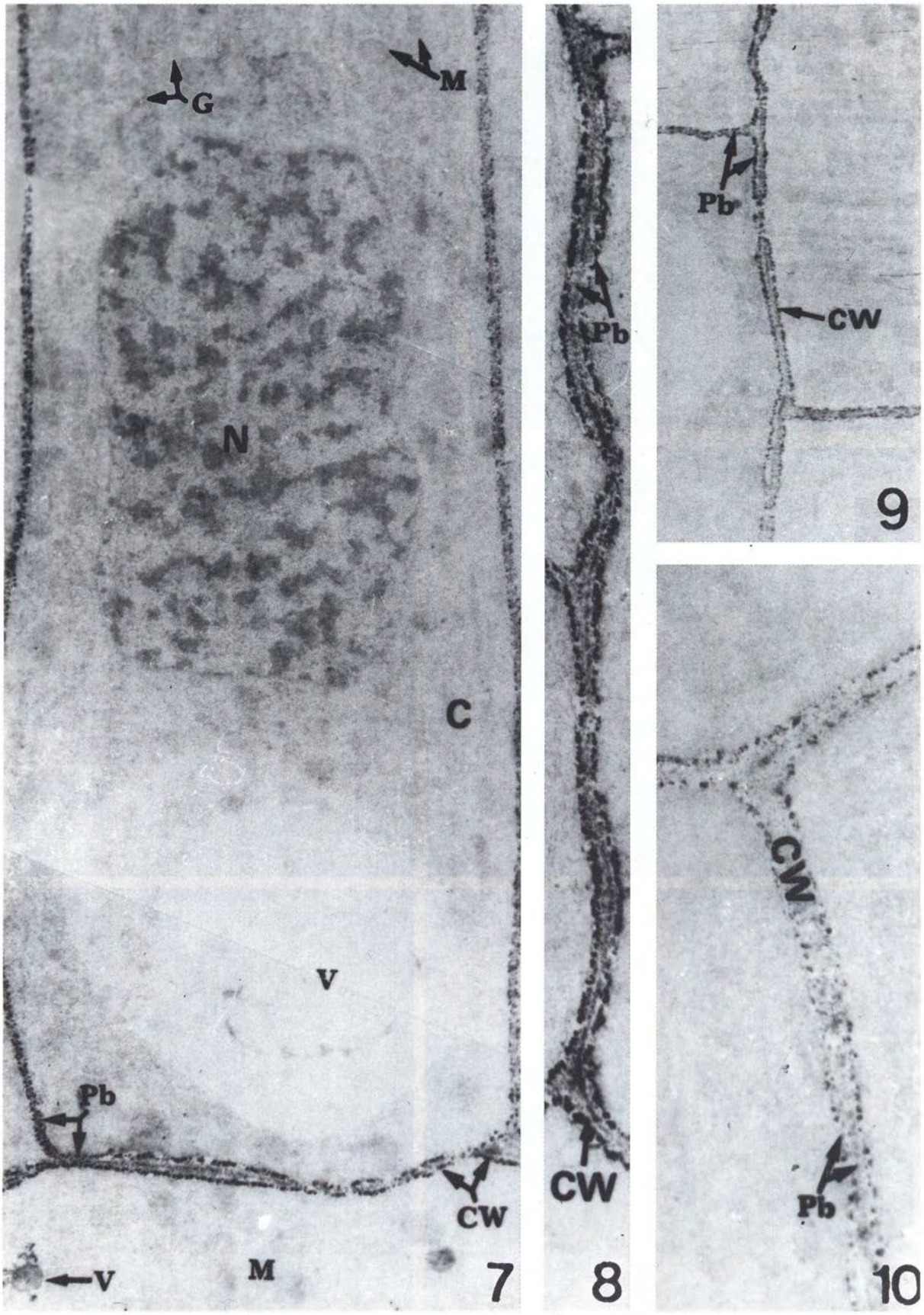
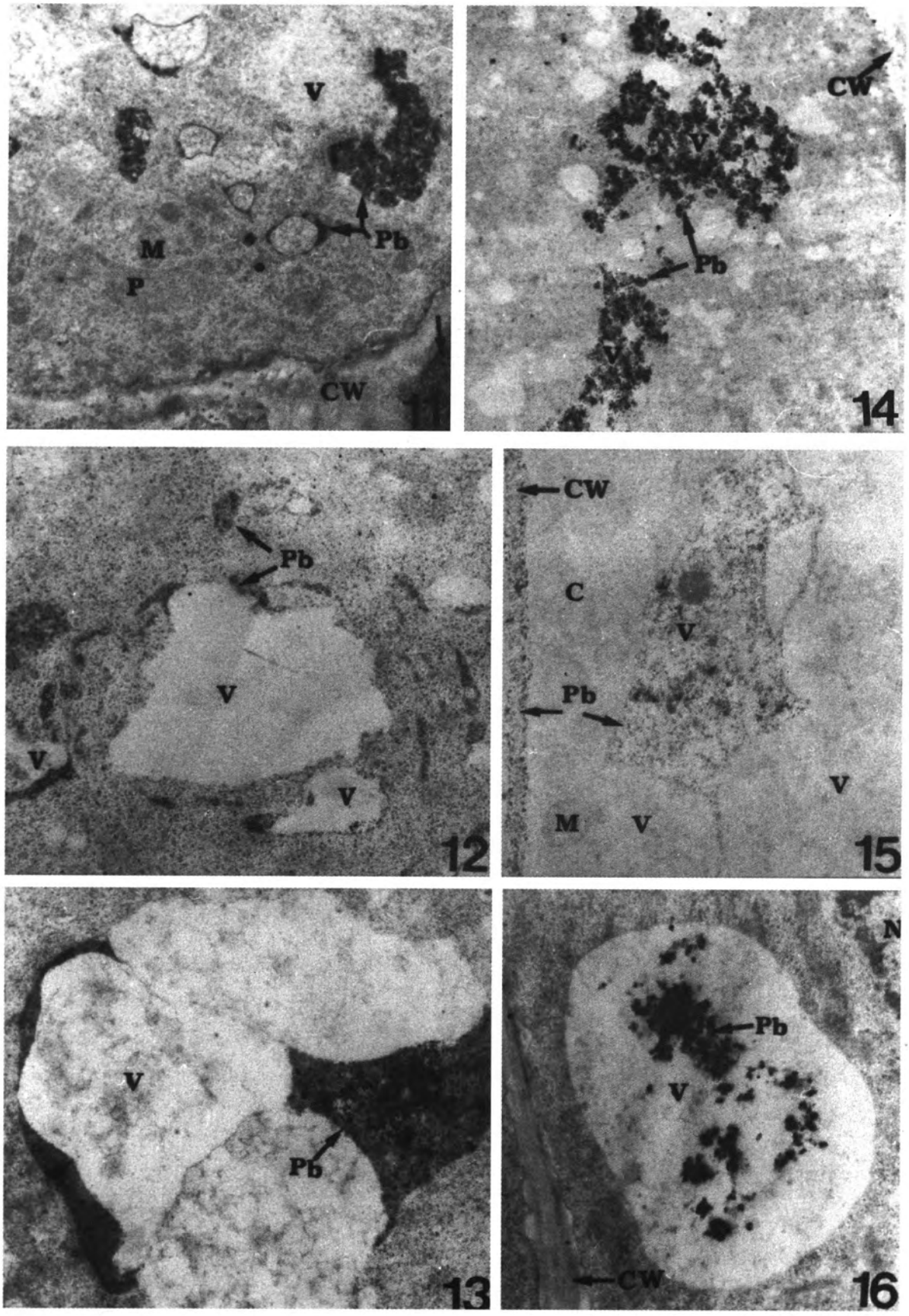


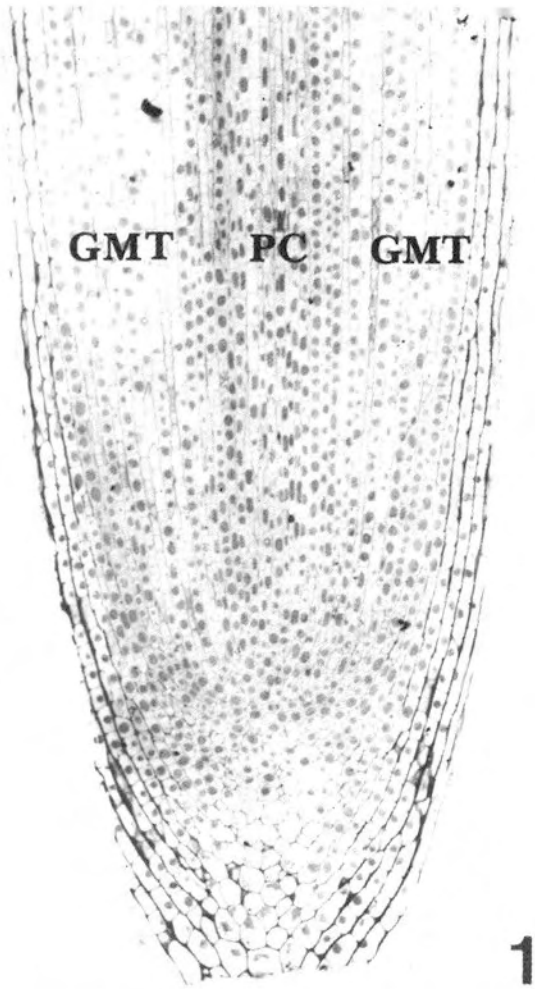
Fig. 3. Binucleate cell. Incubation for 24 h at 3 mg dm^{-3} Pb^{2+} from $PbCl_2$. Micrograph from a squash of onion root tip cells stained with acetic orcein.
Figs 4-6. C-mitoses leading to poliploid cells. Micrographs from squashes of onion root cells stained with acetic orcein.
Fig. 4. C-metaphase and Fig. 5. C-anaphase, incubation for 6 h at 2.5 mg dm^{-3} Pb^{2+} from $Pb(NO_3)_2$.
Fig. 6. Poliploid cell. Incubation for 12 h at 3.0 mg dm-3 Pb^{2+} from $Pb(NO_3)_2$.



Figs 7-16. Electron micrographs illustrating lead electron-opaque deposit distribution in ground meristematic cells, of onion root tip; treated for 6-24 h at 2.5 mg $dm^{-3}Pb_{2+}$ from $Pb(NO_3)_2$. Tissue fixed in glutaraldehyde followed by OsO_4. Figs 7, 8, 11, 12, 13, 16 – sections stained with uranyl acetate and Reynolds' reagent; Figs 9, 10, 14, 15 – no stain sections.
Figs 7-10 Lead (Pb) in cell wall (CW).



Figs 11-16 Lead (Pb) in vacuoles (V); CW – cell wall, D – dictyosome, M – mitochondria, C – cytoplasm, V – vacuole, N – nucleus, P – proplastid, Pb – electron-opaque deposist of lead (Fig. 16 from Wierzbicka M., 1987a. Plant Cell Environ. 10: 17-26, published by permission of Blackwell Science LTD).



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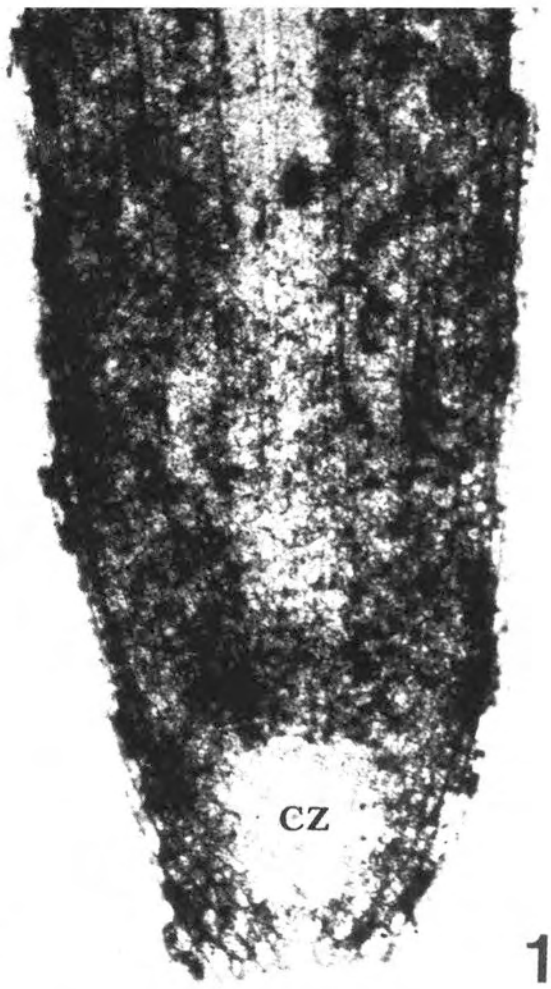

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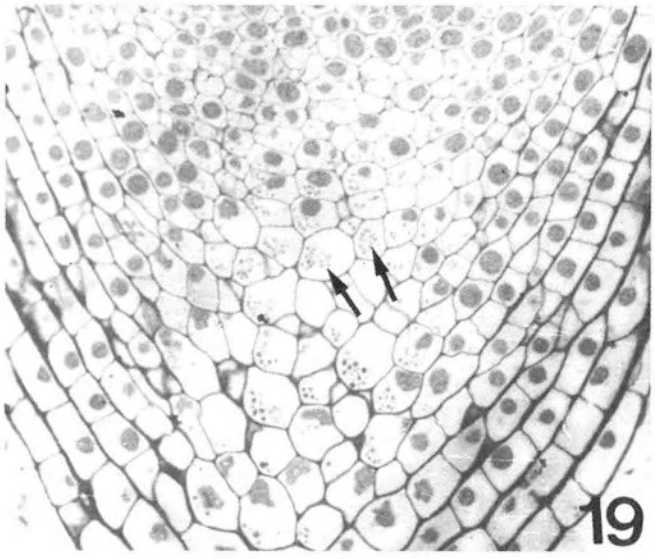

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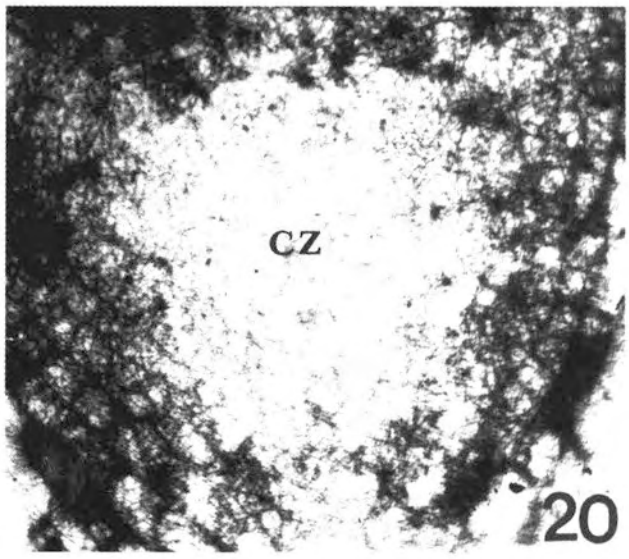

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Figs 17-20. Longitudinal sections of root tip (Figs 17, 19) and corresponding autoradigraphs (Figs 18, 20) illustrating lead isotope distribution in onion root tip cells; treated for 24 h at 2.5 mg dm^{-3} Pb^{2+} from $Pb(NO_3)_2$ + 3.77 k Bq cm^{-3} 210Pb. The largest amount of lead is in ground meristematic cells (GMT). In procambium (Pc) the amount of lead is limited. Evident absence of lead in the central zone (cz) of root – it comprises the quiescent centre (QC) and the central part of the root cap (RC) – Figs 19, 20. Arrows indicate statolith starch in root cap cells. (Figs 17 and 18 from Wierzbicka M., 1987a, plant Cell Environ. 10: 17-26, published by permission of Blackwell Science LTD).

3. Accumulation and detoxification of lead

One of the reasons for the high tolerance of lead in plants is its accumulation only in the cell wall without its penetrating into the protoplast. Such is the case in *Cladonia rangiformis* (Brown and Slingsby, 1972), *Hylocomium splendens* (Gullvåg et al.,1974) and a lead tolerant clone of *Anthoxanthum odoratum* (Quresthi et al., 1986).

In the case of onion roots, lead accumulation in cell walls (Figs 7-10) undoubtedly increases the lead tolerance level of the plant. Lane et al., (1978) indicated in their studies on *Triticum aestivum* coleoptiles that the largest amount of lead was bound to the pectinic acid fraction, pectin substances and hemicellulose.

Therefore, also in *Allium cepa* roots there are possibilities for lead ions to be bound in the apoplast, that is, for their inactivation outside the cell protoplast. Significant quantities of lead, however, migrate from the *Allium cepa* root apoplast into the symplast. It is the lead in the symplast that causes toxic action within the root. Not until later is this lead detoxification.

The main process of lead detoxification, and till now the most often cited, was described by Malone et al. (1974). Studying lead distribution in *Zea mays* roots, they found the occurrence of lead deposits in the dictyosomal vesicles transporting cell wall material with such deposits towards the plasmalemma. Further dictyosomal vesicles fused with plasmalemma, and at the same time the cell wall material containing lead was excreted out of the cell. Hübner et al. (1985) question the direction of process described above, and argue that the processes occuring in these cells are not exocytosis but rather endocytosis.

My studies were carried out under similar conditions as the experiments on *Zea mays* roots.Yet, I have not found the existence of such a process in *Allium cepa* L., although the occurrence of lead deposits in the dictyosomal vesicles was observed. It seems that in the case of *Allium cepa* roots, the only important fact for detoxification was enclosing lead deposits by the membrane in the dictyosomal vesicles and storing them in this harmless form within the cytoplasm. It must be noted that in *Allium cepa* it is the accumulation in vacuoles which is of essential importance (Figs 11-16). The vacuoles are the typical place where cells store their metabolic by products. As *Allium cepa* studies indicate, lead accumulation in vacuoles of ground meristematic cells results from several different processes, and is accompanied by an increase in the number and size of vacuoles.

The findings summarised here suggest that lead loses its toxicity to cells of onion roots. After the initial phase in which lead is toxic to cells, defence processes appear which result in lead no longer being poisonous. Similar defence processes were also found for other plant species (Wierzbicka and Antosiewicz, 1993). It indicates that constitutional lead tolerance is present in plants, and that it is a tolerance for an element totally unnecessary for plant metabolism. Even those plants which have no prior opportunity to create special tolerance mechanisms (Tomsett and Thurman, 1988) have the ability (after several hours) to adapt to a contaminated environment once they are in it.

The findings of this paper allow to draw a futher, biological generalization. Animal organisms have smaller possibilities to defend against lead than plants do. It is due to the different excretory system. Animal cells have neither cell walls nor vacuoles, and just these compartments, present only in a plant cell, play an essential role in effective defence against lead.

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DLACZEGO OŁÓW JEST MAŁO TOKSYCZNY DLA ROŚLIN

STRESZCZENIE

Ta praca jest syntetycznym przeglądem badań problemu ołowiu w środowisku, w szczególności mało poznanego zagadnienia jakim jest tolerancja konstytucjonalna na działanie ołowiu. Na przykładzie korzeni *Allium cepa* wykazano, że po początkowym okresie toksycznego działania ołowiu na komórki włączają się procesy obronne przed ołowiem, w wyniku czego ołów przestaje być dla komórek toksyczny. Ołów, który przedostał się do symplastu korzenia jest unieczynniany na terenie ścian, wakuol i pęcherzyków diktiosomalnych. Przed wnikaniem ołowiu chronione są komórki inicjalne merystemu /strefa QC/ odgrywające podstawową rolę w regeneracji korzenia. Powyższe dane dobrze korespondują z faktem braku objawów zatrucia ołowiem roślin w warunkach naturalnych i wskazują na istnienie mechanizmów obronnych właściwych tylko komórkom roślinnym.

SŁOWA KLUCZOWE: *Allium cepa* L., tolerancja na ołów, detoksykacja ołowiu, translokacja ołowiu w korzeniu, wierzchołek korzenia.