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Short review: the mechanisms of radiocaesium uptake by Arabidopsis roots

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Abstract Both theoretical models and pharmacological dissection suggest that Cs^+ influx to arabidopsis root cells occurs through voltage-insensitive cation channels (VICCs), encoded by members of the *AtCNGC* and *AtGLR* gene families, and 'high-affinity' K^+/H^+ symporters (KUPs), encoded by members of the *AtKUP/AtHAK* gene family. When arabidopsis have sufficient K, it is observed that VICCs mediate most Cs^+ influx to root cells. However, KUPs contribute more to Cs^+ influx in roots of K-starved plants. This phenomenon has been attributed to an increased expression of *AtHAK5* in roots of K-starved plants. Curiously, although arabidopsis mutants lacking some AtCNGCs show reduced Cs accumulation, mutants lacking other AtCNGCs accumulate more Cs in their shoot than wildtype plants. It is hypothesised, therefore, that the expression of genes encoding diverse K^+-transporters might be altered to compensate for the absence of AtCNGCs that contribute significantly to cellular K homeostasis. Increased Cs^+ influx and accumulation could then be explained if the lack of an AtCNGC caused a physiological K-deficiency that increased the expression of *AtKUP*s. Such observations imply that the consequences of a simple genetic manipulation, such as the mis-expression of a AtCNGC gene, on Cs^+ influx and accumulation might not be predicted *a priori*. Finally, since AtCGNCs, AtGLRs and AtKUPs have contrasting Cs^+:K^+ selectivities, and their relative expression is determined by diverse environmental variables, both the Cs:K ratio in plant tissues and the absolute rates of Cs^+ influx and accumulation will depend critically on environmental conditions. This will impact on strategies for phytoremediation and/or the development of 'safer' crops for radiocaesium-contaminated land.

Key words caesium • channel • Chernobyl • phytoremediation • potassium • root

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Introduction

Harmful radiocaesium isotopes (^{134}Cs and ^{137}Cs) arise from the manufacture and testing of thermonuclear weapons, and from intentional and unintentional discharges from nuclear installations [49]. They enter the terrestrial food chain through plants, and their presence in edible portions impacts on both health and commerce. The physical half-life of ^{134}Cs is 2.06 years, and that of ^{137}Cs is 30.17 years.

Root cells take up Cs^+ from the soil solution [49]. The cation then traverses the root via a symplastic pathway formed by the interconnected cytoplasm of root cells linked by plasmodesmata, and is loaded into the xylem for translocation to the shoot [47]. Only about 20% of the Cs delivered to the shoot via the xylem is retained by the shoot, and most is returned to the root via the phloem for recirculation within the plant [9, 21]. Thus, it is argued that the physiological process impacting most on Cs accumulation by a plant are the uptake of Cs^+ from the rhizosphere and the delivery of Cs^+ to the xylem. These processes are catalysed by transport proteins in the plasma membrane of root cells. This paper reviews our knowledge of the molecular

mechanisms catalysing Cs^+ influx to roots of *Arabidopsis thaliana*, a model plant system, and the consequences of their genetic manipulation on Cs accumulation in the shoot.

Different mechanisms dominate Cs^+ uptake by roots of K-replete and K-starved plants

Caesium is an alkali metal with chemical properties similar to potassium (K). It has been suggested, therefore, that Cs^+ enters plants through the same proteins that catalyse K^+ uptake [47, 49, 51]. In the plasma membrane of root cells, inward-rectifying K channels (KIRCs), voltage-insensitive cation channels (VICCs, which are also called nonspecific cation channels, NSCCs), voltage-dependent Ca^{2+} channels (HACCs and DACCs) and 'high-affinity' K^+/H^+ symporters (KUPs) catalyse Cs^+ influx, whilst outward-rectifying cation channels (KORCs and NORCs) catalyse Cs^+ efflux (Fig. 1; [47, 49, 51]). It is thought that Cs^+ fluxes across the tonoplast might be catalysed by cation channels, such as those encoded by *KCO* genes, K^+/H^+ antiporters (KEAs) and/or other cation/H^+ antiporters such as AtNHX1 [32, 47].

In arabidopsis roots, plasma membrane KIRCs are encoded by *AtAKT1*, which appears to be the dominant K^+ channel involved in K^+ nutrition [7, 19, 23, 40], and *AtKC1=AtKAT3=AtAKT4* [17, 33, 34, 52]. The VICCs are encoded by members of the cyclic-nucleotide gated channel (*AtCNGC*) and glutamate receptor (*AtGLR*) gene families [13–15, 48, 49], most of which are expressed in roots [11, 43, 47, 52]. No genes encoding DACCs are known for certain, but HACCs might be encoded by members of the annexin gene family, all of which are expressed in roots [12, 48]. The 'high-affinity' K^+/H^+ symporters are encoded by members of the *AtKUP* gene family, many of which are expressed in root cells and contribute to K^+ nutrition [1, 17, 35, 47, 52]. The KORC AtSKOR is present in the root stele, where it is implicated in loading K^+ into the xylem [18], and AtGORK is present in cells throughout the root, where it is involved in electrical charge compensation [17, 25, 34]. Manipulating the expression, or activity, of these transporters can be used to determine the importance of each to Cs^+ uptake and accumulation by arabidopsis.

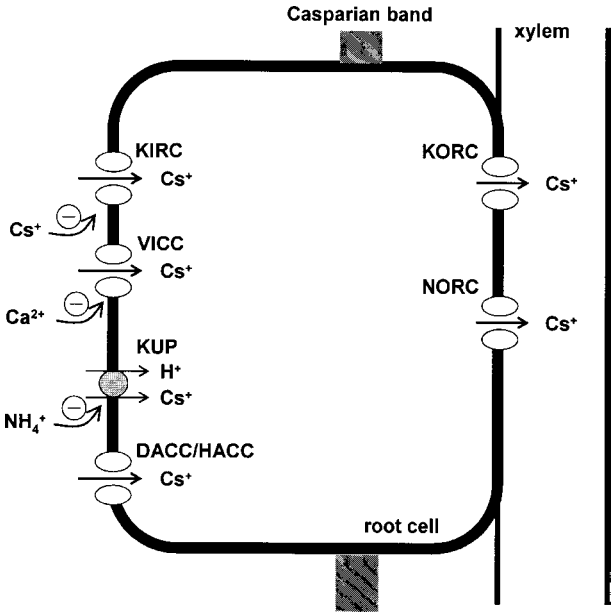

Fig. 1. Cation transport proteins catalysing Cs^+ fluxes across the plasma membrane of root cells [49]. Inward-rectifying K channels (KIRCs) inhibited by extracellular Cs^+, voltage-insensitive cation channels (VICCs) blocked by extracellular Ca^{2+}, 'high-affinity' K^+/H^+ symporters (KUPs) inhibited by ammonium, and voltage-dependent Ca^{2+} channels (HACCs and DACCs) catalyse Cs^+ influx from the apoplast, whilst outward-rectifying cation channels (KORCs and NORKs) catalyse Cs^+ efflux to the xylem. It should be noted that other cations competing for transport might also decrease Cs^+ fluxes catalysed by the proteins illustrated in this figure.

The KIRCs are inhibited by millimolar concentrations of Al^{3+}, La^{3+}, Ba^{2+}, Ca^{2+}, Cs^+ and tetraethylammonium (TEA) in the extracellular medium, but are insensitive to quinine [44, 45]. The VICCs are partially inhibited by millimolar concentrations of La^{3+}, Gd^{3+}, Ba^{2+}, Ca^{2+} and Mg^{2+} in the extracellular medium, some are also inhibited by quinine, but all appear insensitive to TEA, verapamil and nifedipine [14, 44, 45, 48]. There are several types of DACCs with distinct electrical properties and pharmacologies [46, 48]. The most common DACCs in root cells are inhibited by inorganic cations, such as Al^{3+}, Gd^{3+}, La^{3+} and Ni^{2+}, and several organic pharmaceuticals, including TEA, verapamil, diltiazem and ruthenium red, but are insensitive to 1,4-dihydropyridines and bepredil in the extracellular medium [46, 48]. The HACCs are inhibited by submillimolar concentrations of Co^{2+}, Al^{3+}, Gd^{3+}, La^{3+} and verapamil, and weakly sensitive to nifedipine in the extracellular medium [44, 46, 48]. The 'high-affinity' K^+/H^+ symporters are characteristically inhibited by ammonium [5, 30, 37, 40], and monovalent cations compete for transport sites [49]. The KORCs are inhibited by Ba^{2+} and TEA, but not by Ca^{2+}, Cs^+ or quinine at millimolar concentrations in the extracellular medium [44, 46, 48]. The characteristic pharmacology of these transport mechanisms can be used to determine the relative contributions of each to Cs^+ uptake and accumulation by plants [7, 22, 49].

The kinetic parameters of putative Cs^+ transporters have been incorporated into a theoretical model to predict their contributions to Cs^+ influx to root cells [49]. This model predicts that, under K-replete conditions, VICCs mediate most (30 to 90%) Cs^+ influx to root cells, with KUPs mediating the remainder. This prediction is consistent with (a) the identical pharmacology of VICCs and Cs^+ uptake, which are both partially inhibited by extracellular La^{3+}, Gd^{3+}, Ba^{2+} and Ca^{2+} at millimolar concentrations, but not by TEA (Fig. 2; [7, 49]), (b) the phenotypes of some arabidopsis mutants lacking putative VICCs, which show reduced Cs^+ accumulation (Fig. 3; [47]), (c) the phenotypes of arabidopsis mutants lacking AtKUP4 (*trh1*) or with aberrant AtKUP2 activity (*shy3.1*), which accumulate less Cs than wildtype plants when grown on agar containing 20 mM K and 33 mM Cs [47], and (d) the presence of genes encoding VICCs and KUPs at

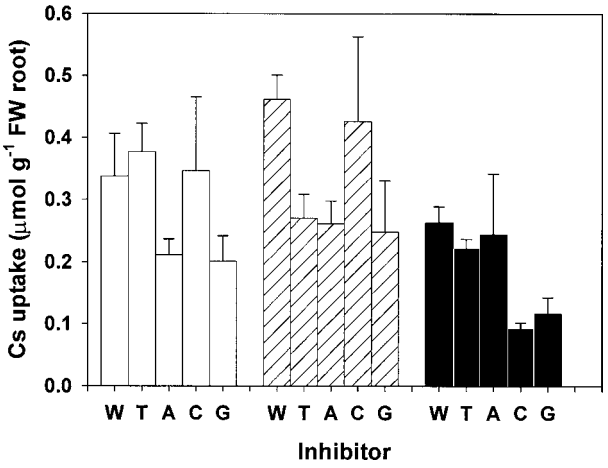

Fig. 2. The effects of 1 mM tetraethylammonium (T), ammonium (A), calcium (C) or gadolinium (G) on Cs^+ influx from a solution containing 50 μM CsCl to 21 d old arabidopsis grown for 14 d in complete nutrient media then transferred to solutions containing 0.5 μM K^+ (white), 100 μM (hatched) or 2 mM (black) prior to the experiment. The K concentrations in roots and shoots of arabidopsis grown with 0.5 μM K^+ were 5.4 ± 1.75 and 1.1 ± 0.27 μmol·g^{-1} DW. The K concentrations in roots and shoots of arabidopsis grown with 100 μM K^+ were 4.8 ± 1.91 and 1.4 ± 0.07 μmol·g^{-1} DW. The K concentrations in roots and shoots of arabidopsis grown with 2 mM K^+ were 9.1 ± 3.37 and 3.0 ± 0.75 μmol·g^{-1} DW. Data were taken from [22] and are expressed as mean ± SE (n = 5 experiments). W = water control.

chromosomal loci (QTL) impacting on shoot Cs accumulation in arabidopsis [32, 51]. The dominant KIRC in the plasma membrane of arabidopsis root cells, AtAKT1, does not appear to mediate significant Cs^+ influx to roots, since Cs accumulation by the *akt1* mutant is often greater than that of wildtype plants [7, 47]. This is not unexpected, since extracellular Cs^+ inhibits KIRCs [45]. It is thought that AtSKOR loads Cs^+ into the xylem, because shoot Cs concentrations are generally lower in the *skor* mutant than wildtype arabidopsis [47].

When arabidopsis lack sufficient K, the complement of K^+ transporters in the root changes. Potassium starvation increases the expression of *AtHAK5* and, occasionally, *AtKUP3* (Fig. 4; [1, 4, 19, 22, 26, 38]), *AtGLR1.2* and *AtGLR1.3* (Fig. 4; [22]). Potassium starvation reduces the expression of *AtSKOR* [29, 33], but rarely affects the expression of genes encoding KIRCs or AtCNGCs in roots (Fig. 1; [22, 29, 33, 38, 47]). The increased expression of *AtKUP*s, and in particular *AtHAK5*, results in an increased capacity for Cs^+ uptake, and changes in the pharmacology of Cs^+ uptake by roots of K-starved plants (Fig. 2; [22]). The fraction of Cs^+ uptake inhibited by ammonium is greater in K-starved arabidopsis (34%) than in K-replete arabidopsis (7%), which is consistent with the hypothesis that AtKUPs mediate more Cs^+ influx to roots of K-starved plants [22].

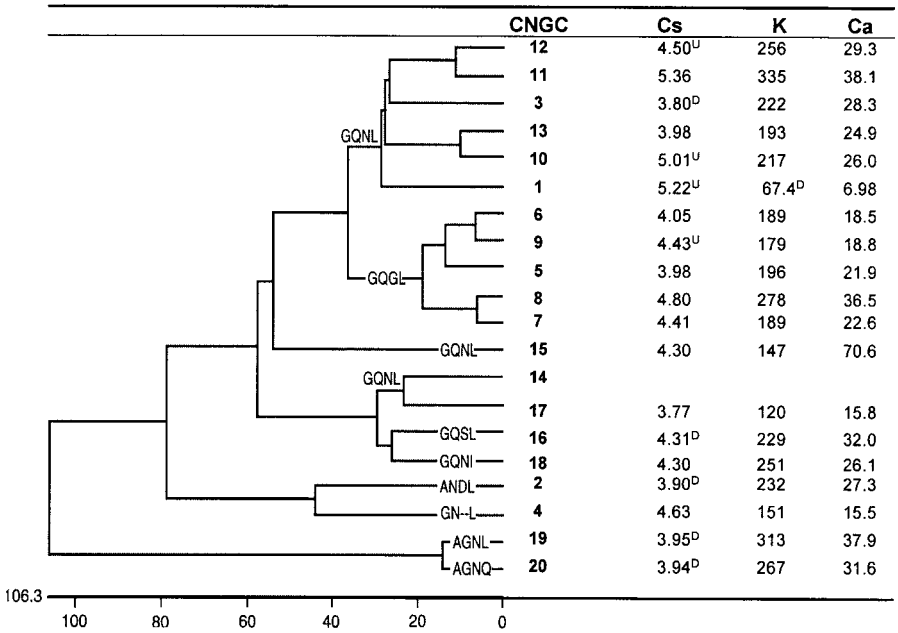

Fig. 3. Dendrogram showing sequence relationships between the twenty AtCNGC proteins identified in *Arabidopsis thaliana* [48] together with the shoot Cs, K and Ca concentrations (μmol·g^{-1} FW) of mutants lacking individual AtCNGCs. The quartet of amino acids in the pore loop implicated in cation selectivity is indicated. The mutants AtCNGC1 [42], AtCNGC4 [51] were assayed in a Wassilewskija (Ws2) wildtype background. The mutants AtCNGC2 (N518387), AtCNGC3 (N556832), AtCNGC5 (N514991), AtCNGC6 (N542207), AtCNGC7, AtCNGC8 (N560397), AtCNGC9, AtCNGC10 (N571112), AtCNGC11 (N548183), AtCNGC12, AtCNGC13 (N560826), AtCNGC15, AtCNGC16, AtCNGC17 (N541923), AtCNGC18 (N581403), AtCNGC19 (N507105) and AtCNGC20 were assayed in a Columbia wildtype background [21]. Codes refer to the Nottingham Arabidopsis Stock Centre (NASC) accession number. Plants were grown on 0.8% w/v agar containing 1% (w/v) sucrose and full-strength MS salts [31] in the absence (for K and Ca) or presence of 1 mM CsCl. Element concentrations given as Residual Maximum Likelihood (REML) means from 3 to 5 replicated experiments. Shoots of the Ws wildtype had 4.15 μmol Cs g^{-1} FW, 423 μmol K g^{-1} FW and 64.3 μmol Ca g^{-1} FW. Shoots of the Columbia wildtype had 4.39 μmol Cs g^{-1} FW, 188 μmol K g^{-1} FW and 25.9 μmol Ca g^{-1} FW. U = significantly greater than the wildtype ($P < 0.05$). D = significantly lower than the wildtype ($P < 0.05$).

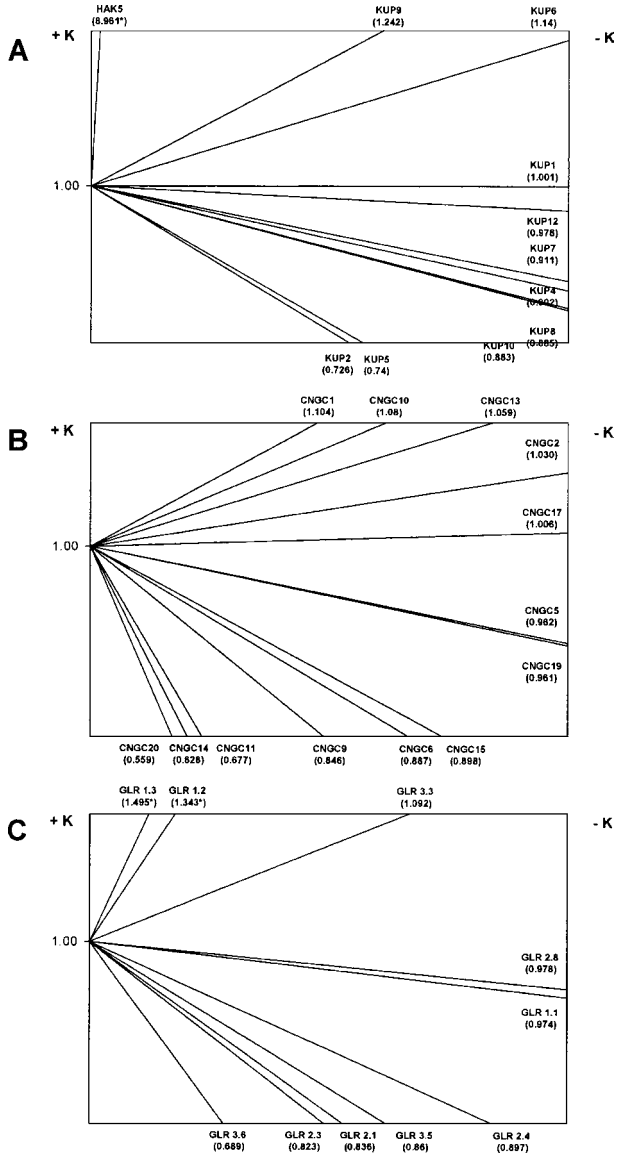

Fig. 4. Relative expression of genes encoding (A) AtKUPs, (B) AtCNGCs and (C) AtGLRs in roots of K-replete (+K) and K-starved (–K) *Arabidopsis thaliana* (Accession Ws2). Plants were grown on 0.8% w/v agar containing 1% (w/v) sucrose and 10% MS salts [31] for 14 d, before being transferred to solutions containing 10% MS salts (K-replete plants) or a modified 10% MS medium containing only 0.5 μM K, in which K salts were replaced by Ca salts (K-starved plants). Data are mean values from 3 experiments. * = significant difference in gene expression between +K and –K treatments ($P < 0.05$).

The mineralogy of arabidopsis lacking individual Cs^+ transporters

It has been suggested that arabidopsis VICCs are encoded by members of the *AtCNGC* and/or *AtGLR* gene families [13, 14, 48, 49]. Based on the rationale that mutants lacking dominant Cs^+ transporters in the root plasma membrane would accumulate less Cs in their shoot, the contribution of specific AtCNGCs to Cs^+ uptake was assessed using mutants lacking each AtCNGC (Fig. 3). Curiously, although mutants lacking some AtCNGCs had reduced shoot Cs concentrations, mutants lacking other AtCNGCs accumulated more Cs in their shoots than the wildtype (Fig. 3; [47]). Specifically, mutants lacking AtCNGCs thought to form Ca^{2+}-permeable cation channels that might contribute to cytosolic Ca^{2+} signals and/or cytosolic Ca^{2+} homeostasis (e.g. AtCNGC2; [24, 48, 50]) often had low shoot Cs concentrations, whilst mutants lacking AtCNGCs thought to form cation channels principally permeable to monovalent cations (e.g. AtCNGC1; [28, 44]) often had high shoot Cs concentrations. Since K starvation has been shown to increase Cs^+ influx and accumulation (Fig. 2; [22]), it is possible that the physiological responses providing K^+ homeostasis in root cells might result in increased Cs accumulation in mutants lacking AtCNGCs that catalyse significant K^+ fluxes. Thus, it can be hypothesised that mutants lacking AtCNGCs affecting K^+ nutrition should show increased Cs accumulation, increased expression of genes encoding AtKUPs, and ammonium-sensitive Cs^+ influx. The correlation between the transcriptional profiles of the *cngc1* and *cngc4* mutants and those of K-deficient arabidopsis [21], and the greater inhibition of Cs^+ influx to *cngc1* and *cngc4* mutants by ammonium (C.R. Hampton, unpublished data) is consistent with this hypothesis. Interestingly, only the lack of AtCNGC1 decreased shoot K concentration significantly (Fig. 3), which may attest to functional compensation by other K^+ transport proteins to maintain K^+ homeostasis in mutants lacking other AtCNGCs. Shoot Ca concentration was unaffected by the absence of AtCNGCs (Fig. 3), which is consistent with previous data. For example, tobacco mutants overexpressing *NtCBP4* (an ortholog of *AtCNGC1*) or a truncated version of *NtCBP4* lacking its C-terminal regulatory domains had the same shoot Ca concentrations as wildtype plants [42] and arabidopsis mutants lacking AtCNGC2 had the same shoot Ca concentrations as their wildtype [10]. No comparable studies have been performed with mutants lacking AtGLRs, and it is not known whether Cs or K accumulation is affected in these mutants. However, arabidopsis overexpressing *AtGLR3.2* had the same shoot Ca concentration as wildtype plants [27].

Prospects for phytoremediation and/or the development of 'safer' crops

The deposition of ^{137}Cs to soils, and its subsequent transfer to the terrestrial food chain through plants, poses a major radiological hazard [20]. Several million people in Belarus, Russia and Ukraine may be affected by ^{137}Cs that still contaminates vast areas of agricultural land following the Chernobyl accident of 1986 [6, 39]. To return this land to safe agricultural production, there appears to be two options. The first is to cleanse the soil of radioisotopes. The second is to grow crops that do not accumulate radioisotopes in their edible portions. Since Cs accumulation by plants is a heritable trait [32], plants that rapidly accumulate large amounts of radiocaesium could be developed in breeding programs. These would accelerate the cleansing of radiocaesium-contaminated soil [16, 51]. Alternatively, or additionally, 'safer' crops could be developed that accumulate little

radiocaesium in their edible tissues [51]. These would complement other agricultural countermeasures to reduce the radiation dose to populations inhabiting areas contaminated by radiocaesium [2, 6].

Root cells take up Cs^+ from the soil solution and deliver it to the xylem via a symplastic pathway [47]. Thus, Cs accumulation by plants is likely to be controlled by the abundance and activity of transport proteins catalysing Cs^+ fluxes across the plasma membranes of root cells. Since these transport proteins also transport K^+ to varying degrees [49], the relative fluxes of Cs^+ and K^+ to the shoot will also be determined by the complement and activity of the transport proteins present in the plasma membranes of root cells. Since the expression of genes encoding transport proteins differs between plant species and species accessions, and is also regulated by plant K status, the relative accumulation of Cs and K in the shoot will depend both on plant genotype and the absolute and relative Cs^+ and K^+ concentrations in the soil solution [47]. Indeed, plant species and species accessions can differ greatly not only in their ability to accumulate radiocaesium (e.g. [8, 32, 41, 51]) but also in their tissue Cs:K concentration ratios (e.g. [3, 25]) when grown under identical conditions. This indicates considerable genetic potential to select and/or breed for plants that might be used for phytoremediation of radiocaesium contaminated soils and/or as 'safer' crops for contaminated land.

Phytoremediation of soil contaminated by radiocaesium is possible, provided total Cs does not reach toxic tissue concentrations [16, 51]. Caesium toxicity is thought to result from competition between Cs^+ and K^+ for binding sites in essential proteins, but rarely occurs in nature [22, 36]. In addition, radiocaesium is a small fraction of the total Cs in the environment [49]. Hence, it should be possible to increase plant uptake and accumulation of ^{134}Cs and ^{137}Cs without affecting cellular biochemistry or K homeostasis. Indeed, this is the phenotype of arabidopsis mutants lacking certain AtCNGCs (Fig. 3). Transcriptional profiling of such mutants suggests that the accumulation of radiocaesium by plants might be increased through constitutively high expression of genes encoding KUPs. This knowledge can be used to develop functional molecular markers for use in conventional breeding programs, or to inform transgenic phytoremediation strategies.

The development of 'safer' crops must be a priority if radiocaesium-contaminated land is to be used for agricultural purposes [22]. However, since the expression of genes encoding proteins that catalyse Cs^+ influx to roots depends critically on plant K status, it has been argued that the success of genetic strategies to produce 'safer' crops might be confounded by the variability in both Cs contamination and K fertilisation of agricultural soils [22, 47]. Specifically, it is argued that, in well K-fertilised soils, reducing the activity of certain CNGCs might lower radiocaesium concentrations in edible tissues, whereas, in K-deficient soils, the activity of KUPs would have to be reduced [22]. In this case, a clearer understanding of the molecular mechanisms dominating radiocaesium influx to roots of arabidopsis grown in contrasting environments will assist the development of 'safer' crops for agriculture.

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