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Supplementary Text:

1. General features of the root microbiota in wild soil.

In line with many recent plant 16S root microbiome census experiments, we found that bacterial EC communities were less diverse than those in bulk soil (Extended Data Fig. 1a, b). We recapitulated previously observed enrichments of Actinobacteria, Firmicutes and Proteobacteria inside the root, and depletions of Acidobacteria, Verrucomicrobia, and Chloroflexi (Extended Data Fig. 1c and Supplementary Table 1). As expected, we found a large difference between root endophytic and bulk soil communities, followed by soil dig (Extended Data Fig. 1d).

2. Control experiments pertinent to figures 2 and 3.

For the design of these experiments, we used as a reference the PSR studied in agar under axenic conditions and long day (where the PSR was originally defined). In this setting, 1 mM - 2 mM Pi is considered full Pi. In our study of the PSR, the optimal Pi concentration in media for microbial growth is typically higher than 2 mM. To avoid excessive stress that could compromise the viability of our SynCom and/or exacerbate production of toxic secondary metabolites that damage the plant, we selected 50 µM Pi (20 times lower than 1 mM). Plants grown at this Pi concentration (in a media free of sucrose and in a short day regime) showed marginal activation of the PSR and a reduced Pi concentration in the shoot (Fig 2a, b and Extended Data Fig. 5d) as compared with plants grown on 1 mM Pi plates. We reasoned that these conditions would facilitate a nutritional competition between plant and the SynCom in the absence of a steady state full induction of PSR, thus providing an excellent scenario for the study of PSR influenced by microbes. The plants showed visible symptoms of PSR only in the presence of the SynCom. We could therefore simultaneously trigger two different stresses (biotic and abiotic) with a single factor (SymCom) and ask how the plants reacted to it.

We performed several sets of control experiments. First, to eliminate the possibility that the SynCom merely mediated sucrose fertilization to restore the PSR transcriptional response, we supplemented Col-0 plants with

a concentration gradient of heat-killed SynCom (see Methods). These treatments did not change either the induction of *IPS1:GUS*[13] or the Pi concentration in the plant shoot (Extended Data Fig. 5e, f). Transcriptional PSR was triggered by our SynCom in low phosphate even without sucrose in the media, a nutritional situation more closely related to growth in wild soil (Fig. 2b). Second, transgenic reporter *IPS1:GUS* plants[13] growing in reduced Pi conditions accumulated reduced shoot Pi concentrations, but the expression of the PSR reporter was not induced even in the absence of supplemental Pi, (Extendend Data Fig. 5c) where seedlings achieved Pi concentrations similar to plants grown in the presence of the SynCom (Extended Data Fig. 5d). However, this marker was induced at low Pi levels in the presence of the SynCom (Extended Data Fig. 5c, d). Therefore, nutritional competition between plant and microbes might explain the reduction in the Pi-concentration in the shoots of plants grown at 50 µM Pi to a level similar to plants grown without Pi supplementation, but it is not enough to explain the induction of the *IPS1:GUS* reporter or the fact that our bacterial SynCom enhanced the PSR (Fig. 2c). Additionally, the finding that PHR1 directly regulates a large proportion of the plant immune system during the PSR triggered by our bacterial SynCom (Fig. 3) argues against Pi exhaustion as the cause of microbial triggering of plant PSR. Third, Phosphite [KH_2PO_3; (Phi)] is a non-metabolizable analog of Pi and its accumulation delays the PSR[56]. Col-0 plants pre-treated with Phi had low Pi content (Extended Data Fig. 6a) but only weakly induced core PSR markers (Extended Data Fig. 6b), even in the presence of our SynCom. We detected similar PHR1 PHL1-dependent induction of the core PSR markers using either replete (1 mM) or low (5 µM) Pi pre-treatments across genotypes, indicating that after 12 days the SynCom induces a long-lasting response to low Pi (Extended Data Fig. 6a, b). Finally, plants colonized by the SynCom also mimicked developmental phenotypes of PSR: a shorter main root (Extended Data Fig. 6c, d) and more lateral roots than non-inoculated plants (Extended Data Fig. 6c, e, f). In sum, the transcriptional PSR responses we observed in the presence of our SynCom were activated by canonical PSR mechanisms and we infer that plants have evolved a mechanism to coordinate defense and PSR.

3. General features of the SynCom colonization experiment in agar.

After SynCom inoculation we also found that agar- and root-associated microbiomes were markedly different from the input and from each other (Fig. 2d and Extended Data Fig. 7e). We also identified eight strains as robust root colonizers regardless of plant genotype or Pi levels (Fig 2d, Supplementary Table 5).

4. Differentially expressed genes in plants growing in the presence of the SynCom.

We identified 3257 differentially expressed genes that responded to either low Pi, presence of the SynCom, or the interaction of both (hereafter PSR-SynCom DEGs) (Extended Data Fig. 8a, b and Supplementary Table 6). In agreement with the fact that PSR is not activated in Col-0 grown in the low Pi conditions we used, only one gene showed a significant change in transcript levels in response to low phosphate availability (Extended Data Fig. 8a, b and Supplementary Table 6). In contrast, 1579 genes, including 164/193 (85 %) of the core PSR marker set, were up-regulated and 958 genes were repressed in response to low phosphate when the SynCom was present (Extended Data Fig. 8a, b).

5. General features of Col-0 and *phr1 phl1* plants exposed to flg22.

To further accentuate the role of PHR1 in the direct regulation of response to microbes, we chose a chronic exposure to flg22. We observed that 251 of the 2690 (9.33 %) genes up-regulated during an acute exposure to flg22 (between 8 and 180 min)[23] were also up-regulated in our experiment (Extended Data Fig. 9a, b; Supplementary Table 11; Supplementary Table 13) and that this gene set contained more PHR1 direct targets than expected by chance (31 observed versus 22 expected, *p*-value = 0.0297).

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