Gall-inducing insects and plants: the induction conundrum

Anantanarayanan Raman*
CSIRO (Health and Biosecurity), Underwood Avenue, Floreat Park, WA 6014 & Charles Sturt University, PO Box 883, Orange, NSW 2800, Australia

Galls induced by insects and mites (insects, hereafter) have been a subject of interest to insect ecologists because of the unusual habit of gall induction and for their tightly connected relationships. These specialist insects and mites have been explored to explain the nature of interactions between them and the plants by entomologists, ecologists and plant physiologists over the last two centuries. However, the questions why only certain insect taxa induce galls on specific species of plants and how galls are induced remain challenging. Whereas several efforts made across the world implicate plant-growth regulators (PGRs) in answering the question on how galls are induced, this article emphasizes the establishment of a metaplasied cell at the location where the tip of the chitinous mandible or ovipositor first hits in the plant. In the light of the differentiation of a metaplasied cell, the earliest plant response, it is but critical to evaluate the physiology of that cell and the ‘new’ physiological events triggered around it, heralding gall initiation. PGRs certainly play a role in gall growth, but only during later stages. This article does not answer the question on how galls are induced. However, it brings to light the gaps that need to be addressed in future in the backdrop of the efforts made over the years. Since we need to deal with the physiological changes that occur in a metaplasied cell and a few adjacent cells, the use of sophisticated optical equipment and pertinent software to achieve a structured and articulate explanation impresses as the way to go.

Keywords: Cell-wall debris, chitinous mandible, gall induction, pathogenic fungi, plant-growth regulators.

INSECTS and mites (hereafter ‘insects’) and the galls they induce are known presently far more than before1–4. The discovery of ‘samurai’ aphids and their unique behaviour among the gall-inducing Aphidoidea by Shigéyûki Aôki was a milestone event5,6 that stimulated similar explorations in other gall-inducing insect groups7. The co-evolutionary ecology of gall-inducing insects and their host plants is a widely pursued topic today8–11. Yet, the answer to the question on how galls are induced is in a mess. This article focuses on the above elusive question. It summarizes what is currently known in the induction of galls, simultaneously pointing to the many gaping holes in that knowledge and the areas that need to be focused upon.

Galls induced by insects (hereafter ‘galls’) exemplify defined plant growth12–14. Plant-growth regulators (PGRs) were implicated as the key. Nysterakis15,16 demonstrated auxins in the salivary extracts of gall-inducing Daktulosphaira vitifoliae (Phylloxeridae) on Vitis vinifera (Vitaceae) and leaf curl-inducing Brachycaudus helichrysi (Aphididae) on Prunus domestica var. domestica (Rosaceae) by testing with the oat–coleoptile and vine–tendril–spin tests, popular in the 1940s. The inhibitory and hypertrophic effects on plants with salivary-extract injections were clarified as due to variations in auxin levels17,18. In the D. vitifoliae–V. vinifera gall system, Nysterakis17,19 related the auxins detected in the saliva of D. vitifoliae to auxin precursors in V. vinifera. Boysen–Jensen20 determined that auxins regulate growth in the galls induced by Mikiola fagi ( Cecidomyiidae) on Fagus sylvatica (Fagaceae). In the next two decades, Guiscarfé-Arillaga21, Beck22, Nolte23, Leatherdale24 and Schäller25 – to name a few – obtained ‘swellings’ on plants by injecting measured quantities of indole-acetic acid (IAA). Such artificially induced swellings were true plant growths, but they differed from the naturally induced galls because the former lacked a definite shape and internal tissue differentiation. Bioassay of whole-body extracts (WBEs) of insects was a popular method used in implicating IAA26,27. Hori28,29 confirmed auxin-like compounds in non-gall-inducing, plant-feeding Miridae, Pentatomidae and Coreidae (Hemiptera) using the WBE method in the 1970s.

Although only indirectly relevant, it would be pertinent to recall the ‘tumour-inducing principle’ (TiP) in Agrobacterium (Rhizobiaceae)-induced tumours on plants proposed by Braun30 (Rockefeller University, USA) in the 1950s. The TiP got explained as the ‘Ti-plasmid’ edited by an endonuclease in the 1980s. However, many stark differences differentiate an Agrobacterium-induced tumour from a gall31.

When much was spoken about the role of IAA and other plant hormones in galls, Anders32 detected lysine, histidine and tryptophan in higher levels, and glutamic acid and valine in lower levels in the salivary secretions of D. vitifoliae. He generated knotty swellings (‘nodosities’) on...
V. vinifera roots cultured in solutions with measured quantities of the specified amino acids. Because the generated swellings were morphologically similar to galls induced by D. vitifoliae on the roots of V. vinifera, Anders implicated these amino acids as the gall-inducing chemicals; alternatively, they could be the precursors of the gall-inducing chemical. Between 1958 and 1961, Anders published several articles reinforcing the role of amino acids in gall induction. However, this was challenged a decade later by Miles33 (Adelaide University, Australia).

Galls are more than entomological and botanical novelties

A gall is the product of a natural, tight relationship between specific insects and plants. It is a near-perfect, exquisite expression on a plant in response to insect action34 (Figure 1). The response pattern of plants to gall-inducing insects varies: some plant taxa are susceptible and an equal number are resistant because of the levels and types of proteins they include35,36. An understanding that a gall is the result of insect action is vital, because other plant abnormalities such as witches’ brooms, fascinations, crinkles, folds and puckerings – either vectored by insects or induced by microbes – are not. In a gall, the inducing insect lives as a parasite inflicting minimal alterations to the physiology of the host and not killing it. Küster34 constructively aligned the explanation of galls on the proposition made earlier by Friedrich Thomas37, p. 513: ‘Ein Cecidium nenne … gegen den erfahrenen Reiz.’.

A gall – cecidium – is a developmental deviation of the plant induced by a parasite. The word ‘development’ means an active process. When a leaf is either consumed or mined by a caterpillar, a gall does not manifest. To establish a gall, active growth in the part of a plant occurs in response to the stimulus from the parasitic organism.

A gall is a phenotypic expression that arises because of perturbation in normal plant growth initiated and stimulated by the insect. In the more-evolved insect orders, e.g. Diptera, an individual neonate induces a gall. In the less-evolved orders, e.g. Thysanoptera and Eriophyoida, in contrast, a population arising from a gravid female contributes to gall development, although initiation is by the feeding action of the gravid female38. The group gall-establishment behaviour, as in the Thysanoptera and Eriophyoida, occurs in some of the gall-inducing Sternorrhyncha, e.g. Adelgidae39. What clarifies a gall – irrespective of the inducing insect belonging to a more- or less-evolved order – is the insect’s dependence on the plant for nutrition. Therefore, establishment of the tissue of nutrition in the gall, redirection of different primary metabolites and minerals to that tissue, and completion of most segments of the lifecycle of the inducing insect within the gall are crucial in defining it40.

Development of a gall can be generalized into the following stages: (i) initiation, (ii) triggering of new differentiation pathways, including establishment of a special tissue for nutrition, with a concurrent inhibition of the normal development, (iii) growth and (iv) ageing and senescence41,42. This explanation has been verified in many galls on various dicotyledons induced by different insect taxa43–45. Although each gall is unique in shape, either a radial or bilateral symmetry in external morphology manifests, an aspect that is strongly deficient in microbe-induced tumours46,47. The nematode-induced root knots (e.g. Meloidogyne, Heteroderidae) are amorphous, similar to microbe-induced tumours in external morphology, but differ from the latter because they include ‘giant-nurse cells’ nourishing the nematode48. These cells are structurally and functionally similar to nutritive cells in galls.

Figure 1. Fir cone-like galls induced by Apsylla cistellata (Hemiptera: Psyllidae: Aphyllidae: Rhinocicidae) on the vegetative axillary meristems of Mangifera indica (Anacardiaceae). (Inset) Vertical sectional view of the gall showing nymphal chambers. For the biology of A. cistellata and gall development, see Raman et al.44. For distribution details, see Sharma and Raman46, Burckhardt et al.139.
To comprehensively understand how galls are induced, the critical element will be to examine the physiology of the first two stages: initiation and triggering of new differentiation pathways, which involve the formation of secondary messengers in response to the signals perceived by the plant because of insect action. The PGRs are produced during either the late second or the third stage of gall development. Production of endogenously produced PGRs at a greater intensity than normal – as in galls – requires a trigger, highly likely, a high-molecular weight protein. An interplay of abscisic acid and ethylene along with auxins and cytokinins occurs in senescing galls, similar to the physiology of senescing fruits.

Host–plant relations of gall-inducing insects

Not all plant-feeding insects induce galls, but only species belonging to certain families of the Eriophyoidea (Acari) and Thysanoptera, Hemiptera, Diptera, Lepidoptera, Coleoptera and Hymenoptera (Insects). Among these, the species of Hemiptera, Diptera, Lepidoptera and Coleoptera (Curculionidae) induce galls by the feeding action of their immature stages, whereas in Hymenoptera, gall initiation starts with the ovipositing female inserting her ovipositor into the plant organ, concurrently discharging accessory-gland secretions. Gall induction in certain tribes of the Cecidomyiidae – Asphondylini, Porricondylini and Lasiopterini – occurs via insertion of the ovipositor52 along with the introduction of fungal spores53. Such a gall-inducing behaviour among Cecidomyiidae is odd.

Gall-inducing insects exemplify a sophisticated level of phytophagy. Well-defined galls are induced on specific species of plants by specific species of insects. This specialist behaviour leads to considering gall-inducing insects as ‘excellent plant taxonomists’. The recent resolution reached in the context of Oenothrips cochinchenissis (Phlaeothripidae) that induces large sac-like galls on Getonia floribunda (Calycoperis floribunda, Combretaceae) in peninsular India illustrates this point. In a majority of gall-inducing insects, specialization extends to specific organs and sites. Exceptions exist, however. For instance, Thilakothrips babali (Phlaeothripidae) induces rosette galls on both leaflets and florets of Vachellia leucophloea (Fabaceae: Mimosoideae) in southern India. Quadristicus erythrinae (Eulophidae) is known to induce gall-like abnormalities on the leaves and flowers of more than six species of Erythrina (Fabaceae). Similarly, Leptocybe invasa (Eulophidae) is indicated as the inducer of gall-like abnormalities on the petioles, leaves and flowers of about 30 species and subspecific variants of Eucalyptus (Myrtaceae). Quadristicus erythrinae and L. invasa are confirmed oligophages, and oligophagy is less known among established gall-inducing taxa. Prodiplosis longiflora (Diptera: Cecidomyiidae), recorded as the inducer of rosette galls on the shoot terminals of Jatropha clavuligera (Euphorbiaceae) in South-Central America, is also known to occur on several unrelated plants both as a gall-inducing taxon and not a gall-inducing taxon. However, host-specificity tests of infested and uninfested shoots of the Bolivian populations of J. clavuligera and a few allied and co-occurring species of Jatropha, made in 2017, have clarified that the populations of P. longiflora living on J. clavuligera are a host-specific, cryptic species of the P. longiflora species complex. Therefore, the question is whether the anomalies induced by Q. erythrinae and L. invasa are true galls? That other biological agents, possibly a fungus, induce amorphous growth on Erythrina and Eucalyptus, subsequently infested by the respective Eulophidae, is a strong possibility.

How and why most of the gall-inducing insects remain tied to specific plants is a mystery. Possibly gall induction requires specific molecular signals that can be triggered only by a particular species of insect endowed with specific proteins. One early explanation was that the gall-inducing Taxomyia taxi (Cecidomyiidae) selectively exploits Taxus baccata (Taxaceae) for sterols necessary for the larvae to become adults. Specific mono- and di-glycerides were detected in young, uninfested leaves of Eucalyptus macrorhyncha (Myrtaceae) in Central-West New South Wales, Australia, that hosts an unnamed gall-inducing species of Glycaspis (Synglycaspis) (Aphalaridae). The natural habitat of E. macrorhyncha includes co-occurring populations of Eucalyptus rossii and Eucalyptus dives. Botanists (e.g. ref. 66) treat these three Eucalyptus taxa under the same group: ‘Eucalyptus subgen. Eucalyptus + Primitiva’. The unnamed, pouch gall-inducing species of Glycaspis (Synglycaspis) never occurs on either E. rossii or E. dives. Significant levels of sitosterol, ergosterol and stigmasterol were detected in young leaves of E. macrorhyncha susceptible to gall induction by this species of Glycaspis (Synglycaspis). Moreover, sitosterol and three other undetermined sterols of molecular weights 354, 382 and 440 g mol–1 were present maximally only in young leaves of E. macrorhyncha, but absent in E. dives and E. rossii leaves of comparable age. The unique 440 g mol–1 sterol was clinched as the principal factor in the choice of E. macrorhyncha by the gall-inducing species of G. (Synglycaspis), because of its high levels in the young, gall-susceptible leaves of E. macrorhyncha. This study reinforced the explanation made in the 1980s that gall-inducing insects choose specific plants to meet their sterol needs.

The earliest recognizable element in a gall – the metaplasied cell

Gall initiation becomes apparent in the first 24 h of attack of the plant by the insect (ref. 69, figure 30). In the Fagus
Küster first recognized metaplasied cells in galls attacked by the neonate myiidae gall system, dynamic changes occur at the site of gall initiation. Presently, the terms ‘dedifferentiation’ and ‘trans-differentiation’ have replaced metaplasia in plant and animal physiology respectively. Metaplasia was previously explained in early-stage human cancer by Rudolf Virchow in 1884. According to Küster, first recognized metaplasied cells in galls. Metaplasia was previously explained in early-stage human cancer by Rudolf Virchow in 1884. According to Küster, Metaplasia plays a more serious role in the pathological histology of plants than that of animals and humans.

We see different kinds of metaplasied cells forming the nutritive tissue in Fettgewebe sich umwandelt." Metaplasia plays a more serious role in the pathological histology of plants than that of animals and humans.

We see different kinds of metaplasied cells forming the basis for various important plant-pathological processes. Of course, such a transformation (i.e. from normal cell to metaplasied cell) is possible only among closely related tissue meristems, especially among the nutritive tissue (e.g. parenchyma). Nevertheless, in metaplasia, the original character of the converted cells can be thoroughly unrecognizable – for example, a binding tissue (e.g. cortical parenchyma) transforms into fat (i.e. lipid)-including (i.e. storage) tissue. Virchow explained metaplasia as the replacement of one differentiated cell type by another differentiated cell type, normally absent in that tissue, and cancer arises by the activation of ‘dormant cells’ (stem cells, today) via irritation. Presently, the terms ‘dedifferentiation’ and ‘trans-differentiation’ have replaced metaplasia in plant and animal physiology respectively.

The cell injured by the tip of either the pointed mandibular tooth as in Cecidomyiidae or the pointed, needle-like terebra of the ovipositor as in Cynipidae loses its polarity, turns metaplasied, followed by rapid changes in the quality and quantity of subcellular inclusions. The establishment of a metaplasied cell, however, varies with the nature of the influencing mechanism of the inducing insect and is regulated by the nature of either the mouth parts or the ovipositor of the involved insect, as the case would be. One distinct feature of the metaplasied cell is the asymmetrical distribution of proteins either in or on the plasma membrane. These subcellular modifications enable the host plants to adjust and align their continued growth. Following the establishment of a metaplasied cell, sequel to wounding and irritation caused by insect action, the chemical(s) discharged from the saliva or the accessory glands trigger cell-division activity around it. Details of the involved chemical(s) are presently unknown: high-molecular weight proteins, bruchins, mitogenic lipids. The establishment of metaplasied cells at the prospective gall site is pivotal in gall initiation.

The nutritive tissue

Several subcellular modifications, reflecting the physiology of the involved cell, follow immediately. I will use examples of D. vitifoliae–Vitis cv. 3309 Couderc (V. riparia × V. rupestris cv. C–3309) and Aceria lycopersici (Eriophyidae)–Solunum dulcamara (Solanaeae) to illustrate the less than 24 h changes in the susceptible plant organ, a leaf. The tips of 60–65 μm long stylets of the neonate nympha of D. vitifoliae can only reach the fifth–sixth layer mesophyll cells. In 3–6 h, the cell including the styel tip enlarges at least twice its normal size, concurrently presenting a modified subcellular structure (Figure 3a), similar to the changes that occur in early-stage human cancer cells, except cell-wall modifications. In the next 24 h, the mesophyll–parenchyma cells lining the styel path present modified subcellular profiles (Figure 3b). Vitis cv. 3309 Couderc leaf under the feeding pressure of D. vitifoliae develops a nutritive tissue in 48 h.
Figure 2. Gall initiation (<24 h) on the leaves of Fagus sylvatica by neonate larvae of Hartigiola annulipes. a, Neonate H. annulipes larva (el) settling on a leaf of F. sylvatica. Note the edges around the larva showing early signs of overarching growth (scale bar – 100 μm). b, Paired feeding punctures inflicted by H. annulipes (unfilled arrow), stomata (filled arrow; scale bar – 100 μm). c, Sectional view of a feeding puncture (EM) (arrow – callose; scale bar – 0.5 μm). (Source: Rohfritsch70, with permission from O. Rohfritsch.)

Figure 3. Less than 24 h in Daktulosphaira vitifoliae interaction with Vitis vinifera cv. 3309 Couderc leaf. a, Neonate nymph of D. vitifoliae on Vitis leaf (1 h; scale bar – 100 μm). b, Target parenchyma cell including the stylet tip (st) activated and characterized by intense cytoplasm, enlarged nuclei and numerous small vacuoles; the parenchyma cells along the stylet path are also activated, developing into the nutritive tissue which includes abundant chloroplasts (3–6 h) (ue, upper epidermis; Scale bar – 100 μm). c, Parenchyma cells, slightly away from the developing nutritive tissue, include enlarged nuclei (n), hyaline cytoplasm, and from one to a few centrally placed large vacuoles with many small and large exhausted multi-vesicular bodies (mb) and subcellular debris (scd); the multivesicular bodies in the developing nutritive cells appear normal and active (arrow; 3–6 h; scale bar – 100 μm). d, A nutritive cell with enlarged and irregularly-shaped nuclei (n) and enlarged nucleoli; the nuclei include electron-dense chromatin material and interchromatin granules, whereas the nucleoli are vacuolated (nv); membranes of the nuclear envelope are separated and bear patches of electron-dense condensations; the plasma membrane is unevenly retracted (arrows) from the cell wall; especially at the retraction points, lomasomes occur in the periplasmic space; cytoplasm is dense and includes many small, but scattered vacuoles (v); strands of rough endoplasmic reticulum (rer) occur scattered throughout the cytoplasm; the mitochondria (m) distributed along the plasma membrane appear normal and are numerous, whereas those either between the nucleus and vacuoles or between two adjacent chloroplasts include vesiculated cristae and empty central spaces; chloroplasts (chl) are not hypertrophied, but the granal stacks are numerous and compressed; the thylakoids are condensed with their membranes dilated, sequel to granal compression; chloroplasts include neither plastoglobuli nor starch (24 h response; scale bar – 1 μm). (Source: Raman et al.71.)
tissue includes hypertrophied cells and nuclei, more-than-usual numbers of mitochondria, and other modified cell organelles. This tissue includes high levels of starch and lipids, but low levels of phenolic inclusions (Figure 3c and d).

At this earliest ‘recognizable’ stage of gall induction, the nucleus in the metaplasied cell remains spherical and unlobed, but will be strikingly different from the nuclei in normal cells in the same organ by its large size, greatly dispersed heterochromatin and large nucleolus. Many subcellular changes occur concurrently. The endoplasmic reticulum expresses as myelin figures indicating oxidative stress.\(^{89}\) The Golgi bodies get intensely modified reflecting alterations in the pathways in lipid–protein metabolism and subcellular transport. Mitochondria mostly remain unaltered, indicating no major alteration in the respiratory activity. Various modified plastids occur reflecting stress and altered photosynthesis.\(^{90}\) This modified tissue will include elevated levels of primary metabolites, further to an intense phosphatase activity reinforcing greater inorganic phosphate utilization.\(^{91}\) Starch usually occurs in non-hydrolysable form in cells away from the site of feeding by the insect; lipids, on the other hand, occur in cells close to the insect.\(^ {92}\) In Cynipidae-induced galls, lipids occur as di- and triacylglycerides.\(^ {14}\) The contrasting distribution patterns of carbohydrates and lipids in the nutritive tissue are an established expression of stress-neutralization effort by the plant,\(^ {93}\) because of the production of superoxide radicals affecting various cell and tissue functions.\(^ {94}\)

The less than 24 h changes that occur in the leaf cells of susceptible varieties of\( S. \) dulcamara punctured by the chelicerae of\( A. \) lycopersici include vacuolar alkalinization followed by alteration in DNA levels associated with chitosan build-up,\(^ {98,95}\) illustrating the changes influenced by signal perception and transduction. This action triggers the host plant cell to turn metaplasied, communicating with the neighbouring cells via signal transduction. A nutritious tissue gets established in the next 3–4 h, on which individuals of\( A. \) lycopersici feed. These changes never manifest in the varieties of\( S. \) dulcamara resistant to\( A. \) lycopersici. In the incompatible (resistant) reactions between\( A. \) lycopersici and\( S. \) dulcamara, a rapid spread of subcellular damage from the punctured cell to those in the neighbourhood manifests as cell necrosis, expressing externally as tissue lesions. This hypersensitive reaction in resistant varieties of\( S. \) dulcamara impedes further feeding by\( A. \) lycopersici, followed by their death.\(^ {96}\)

Establishment of the nutritive tissue in galls reinforces the nutrition hypothesis, underpinning its ecological relevance in gall induction.\(^ {76,97,98}\) The structure and design of nutritive tissue in galls induced by different insect groups is generalizable, although the specific nature of location, distribution and orientation varies with insect groups.\(^ {93}\) Such a variation arises because of the nature of the mouth parts of the inducing insect(s) and their respective feeding behaviour(s). For example, in galls induced by hemipteroids with sucking behaviour, nutritive tissue differentiates at varied depths on the same plant organ (e.g. a leaf). In galls induced by Phlaeothripidae, the nutritive tissue differentiates immediately below the epidermis, because of the short length and asymmetry of mouth parts.\(^ {99}\) In contrast, in galls induced by Sternorrhyncha that bear relatively long and slender stylets, the nutritive tissue develops at 5–10 layers depth in the mesophyll.\(^ {100}\) The location of the nutritive tissue in the plant organ is directly related to the lengths of stylets of the feeding Sternorrhyncha.\(^ {45,101}\)

Feeding action – physical injury and irritation, chemical action by the salivary secretions – of the inducing insect ensures the active status of the nutritive tissue. When the larva stops feeding, the nutritive tissue loses its dynamic profile and gets replaced by inactive parenchyma (e.g. sclereids)\(^ {14,45,102}\). When the larva is either removed or killed, the distribution of carbohydrates and lipids in the non-functional nutritive tissue rapidly reverses.\(^ {93}\) Accumulation of other metabolic products, such as minerals, is known,\(^ {103,104}\) but those details would be irrelevant here.

That a gall is a nutrient sink was first shown in the\( M. \) fagi – \( F. \) sylvatica gall system by Kirst and Rapp\(^ {105}\) (Darmstadt, Germany) in the 1970s. Total non-structural carbohydrates (TNCs) and carbon–nitrogen isotope ratios were measured in tissues of galls, gall-proximal, gall-distal and non-gall-bearing stems of identical age of\( P. \) hysterophorus (Asteraceae) induced by\( E. \) strenuana (Curculionidae).\(^ {106}\) The\( E. \) strenuana larvae drain nutrients and energy, stress the shoot tissues of\( P. \) hysterophrous by intercepting normal-nutrient transport. The\( \delta^{13}\)C and\( \delta^{15}\)N values in galls were significantly different from those in stem segments proximal and distal to the galls, although the TNC levels were insignificant regardless of plant age (Figure 4 and Table 2). The stem distal to the gall functioned more efficiently as a nodal channel than the stem proximal to the gall, especially in the translocation of nitrogenous nutrients, affirming that a gall is a nutrient sink. This is due to the injury inflicted by feeding action of the insect, and the plant responds by pumping nutrients to the gall site primarily to repair the injury. The insect utilizes the redirected nutrients to its advantage.

**Gall growth**

Bioassays of galls induced at the base of the young needles of\( P. \) edulis (Pinaceae) by\( J. \) coloradensis (Cecidomyiidae) revealed that the galls included about 20 times more of auxin activity and gibberellin-like substance activity. The highest levels of both auxin and gibberellin-like substance were apparent during the rapid gall growth stage.\(^ {107}\) Curiously, only traces of substances
Figure 4. Mean values (+ SE of mean) of $\delta^{13}C$ and $\delta^{15}N$, and levels of total non-structural carbohydrate (TNC) from the leaf (L), proximal stem (PS), gall (G), distal stem (DS) and root (R) in different developmental stages of Parthenium hysterophorus. Same letters indicate that means are not statistically different (Tukey’s HSD test, $P < 0.05$). (a–c) Carbon values ($\delta^{13}C$, ‰) from rosette (a), preflowering (b) and flowering (c) stages. (d–f) Nitrogen values ($\delta^{15}N$, ‰) from rosette (d), preflowering (e) and flowering (f) stages. (g–i) TNC levels from rosette (g), preflowering (h) and flowering (i) stages. Source: Raman et al. 106.

with gibberellin-like activity were detected and there were no auxins at detectable levels in the extracts of J. coloradensis107. Byers et al.107 extrapolated this finding that the auxins are of plant source and insect action stimulates their activation. In contrast, in an unnamed species of Pontania (Tenthredinidae) – Salix japonica (Salicaceae) gall system108, IAA has been detected in the larval saliva, metabolized from tryptophan via deamination and decarboxylation. Transcript levels of auxin- and cytokinin-responsive genes were higher in gall-bearing than in non-gall-bearing plant organs, indicating that the insect action activates the genes responsible for this action. Abnormally high levels of t-zeatin riboside in Pontania galls on S. japonica indicate that Pontania could synthesize cytokinins as well as IAA. Gene profiles indicate high levels of auxin and cytokinin activity in galls108. Yamaguchi et al.108 clarify that the two undetermined adenine derivatives identified by McCalla et al.109 in the 1960s are in fact the ‘t-zeatin riboside’ and ‘isopentenyladenosine’, signal molecules of cytokinin biosynthesis in plants. This is an elegant explanation of stage-3 in gall development. Using gas chromatography coupled with mass spectrometry (GC–MS), high levels of IAA have been demonstrated in the larvae of Eurosta solidaginis (Tephritidae)110 and Gnorimoschema gallae solidaginis (Gelechiidae)111 respectively, both inducing galls on the stems of Solidago altissima. Collectively, the studies made in recent years, using sophisticated analytical equipment and those made between the 1940s and 1980s using less sophisticated methods suggest the possibility that the inducing insect larvae include precursors of IAA and cytokinins, which get introduced into the plant tissues through their salivary or accessory-gland secretions.

Senescing galls

The physiology of galls on maturation, i.e. when the inducing insect ceases to feed and refrains from stimulating the gall to grow is broadly similar to the physiology of normally ripening and senescing fruits112. However, what needs to be factored here is that the proportions of production, transport and storage of various primary and secondary metabolites vary with the insect and plant.
species involved. These responses depend on the nature of physical and chemical stresses exerted by the inducing insect. Photosynthesis, for instance, is intensely altered in galls because of structural and functional modifications in chloroplasts. Yet, sugar transport from other parts of the same plant occurs mostly, via symplast. Dehiscence of galls and fruits involves similar physiological processes. The dehiscing fruits and galls include newly differentiated tissue and their source are critical factors.

Chitin, a glucosamine polymer, is a key constituent of body parts of insects (e.g. mandibles, ovipositor). Plants include chitinases and are capable of recognizing chito-oligosaccharides from pathogenic fungi during the early infection stage. Introduction of chitin also stimulates mitogen-activated protein kinase (MAPK) cascades and a network of transcription factors. Similar to the action of chitin in plant-pathogenic fungi, the chitin discharged by gall-inducing insects into plant cells during gall induction acts as an elicitor. With the discharged chitin from the feeding insect, the host plant cell recruits a downstream pathway negotiating either a susceptible or a resistant response.

Wounding of plant cells by the insect results in rapid modification of the subcellular environment, accompanied by chemical shock triggered by chitin discharged by the attacking insect. Physiological steps characterized in Arabidopsis thaliana (Brassicaceae) wounded by Spodoptera littoralis (Noctuidae) – a non-gall-inducing taxon – provide a credible explanation. When wounded by S. littoralis, A. thaliana tissue instantaneously activates two MAPKs: MAPK3 and MAPK6. But this activation is dependent on the upstream MAPKs: MAPK4 and MAPK5, but independent of jasmonic acid. Extending the interpretations made in the S. littoralis – A. thaliana system to a gall, during inception, activation of specific MAPKs

**Table 2.** Sterols (mol%) in uninfested and gall-bearing leaves of Eucalyptus macrorhyncha and comparable leaves of Eucalyptus rossii and Eucalyptus dives*

| Sterol molecular weight | E. macrorhyncha – Synglycaspis sp. system (sterols in mol%) | E. rossii (sterols in mol%) | E. dives (sterols in mol%) |
|-------------------------|-------------------------------------------------------------|-----------------------------|--------------------------|
|                         | Y               | M       | 1'     | 2'     | 3'     | 4'     | 5'     | Y     | M       | Y     | M       | 0     | 0     |
| 326.4                   | 0.02            | 0.03    | 0.02   | 0.02   | 0.03   | 0.03   | 0.03   | 0.02  | 0.01    | 0.03  | 0.02    | 0     | 0     |
| 354.1                   | 0.08            | 0.11    | 0.07   | 0.07   | 0.08   | 0.07   | 0.06   | 0.14  | 0.08    | 0.08  | 0.13    | 0     | 0     |
| 382.1                   | 0.13            | 0.2     | 0.16   | 0.16   | 0.17   | 0.18   | 0.12   | 0.40  | 0.32    | 0.27  | 0.37    | 0     | 0     |
| 396.3                   | 0.01            | 0.02    | 0.01   | 0.01   | 0.01   | 0.01   | 0.01   | 0.01  | 0.01    | 0.00  | 0.01    | 0     | 0     |
| 410.2                   | 0.05            | 0.25    | 0.12   | 0.12   | 0.18   | 0.13   | 0.13   | 0.08  | 0.13    | 0.02  | 0.02    | 0     | 0     |
| 412.4                   | 0.05            | 0.05    | 0.05   | 0.05   | 0.05   | 0.05   | 0.05   | 0.02  | 0.02    | 0.03  | 0.02    | 0     | 0     |
| 414.3                   | 0.11            | 0.12    | 0.16   | 0.16   | 0.16   | 0.16   | 0.19   | 0.21  | 0.24    | 0.20  | 0.18    | 0     | 0     |
| 424.4                   | 0.00            | 0.015   | 0.01   | 0.01   | 0.01   | 0.01   | 0.01   | 0.04  | 0.01    | 0.01  | 0.01    | 0     | 0     |
| 426.2                   | 0.06            | 0.05    | 0.08   | 0.07   | 0.07   | 0.07   | 0.10   | 0.04  | 0.05    | 0.06  | 0.05    | 0     | 0     |
| 440.3                   | 0.38            | 0.060   | 0.34   | 0.23   | 0.17   | 0.14   | 0.17   | 0.02  | 0.02    | 0.14  | 0.11    | 0     | 0     |
| 454.2                   | 0.02            | 0.03    | 0.02   | 0.03   | 0.03   | 0.03   | 0.05   | 0.00  | 0.00    | 0.02  | 0.04    | 0     | 0     |
| 534.3                   | 0.02            | 0.00    | 0.03   | 0.01   | 0.02   | 0.00   | 0.00   | 0.00  | 0.00    | 0.00  | 0.00    | 0     | 0     |

0, Uninfested leaves; I, Infested leaves; Y, Young uninfested leaves; M, Mature uninfested leaves; 1’, 2’, 3’, 4’, 5’, Galls harbouring populations of the first, second, third, fourth, and fifth instars (n = 50 each category).

*Source: Sharma et al.*
possibly occurs and they in turn provoke osmotic changes in the proplast of the attacked cell, resulting in the earliest recognizable stage in gall induction.

The wounded cell gets activated and turns metaplasied because of the effecter proteins discharged from the insect: either from the saliva (e.g. Cecidomyiidae) or from the accessory glands (e.g. Cynipidae). Among the different suggestions explaining the possible chemical triggering the gall, the 58 kDa protein shown in S. altissima – E. sолодагинис галл impresses as the most credible. Highly likely, a high-molecular weight protein discharged simultaneously with the chitin triggers the formation of the metaplasied cell, followed by a new morphogenetic pathway to establish the specialized nutritive tissue around it.

Similar to pathogenic fungi, gall-inducing insects can overcome the innate immunity and establish a susceptible response of plants. Here a question whether the gall is overcame the innate immunity and establish a susceptible cell, followed by a new morphogenetic pathway to establish the specialized nutritive tissue around it.

The wounded cell gets activated and turns metaplasied because of the effecter proteins discharged from the insect: either from the saliva (e.g. Cecidomyiidae) or from the accessory glands (e.g. Cynipidae). Among the different suggestions explaining the possible chemical triggering the gall, the 58 kDa protein shown in S. altissima – E. sолодагинис галл impresses as the most credible. Highly likely, a high-molecular weight protein discharged simultaneously with the chitin triggers the formation of the metaplasied cell, followed by a new morphogenetic pathway to establish the specialized nutritive tissue around it.

The resulting metabolic changes stimulated by alterations in the vacuolar pH – presently referred as ‘novel’ chemicals of unknown details – diffuse from these dedifferentiated cell(s) into the immediate neighbourhood, but are localized because of their obvious weak nature. This means that the effect does not spread throughout either the involved organ or the plant, explaining why galls and their effects are localized. Relevant here will be to remind us that the term ‘toxin’ was liberally used in the 1960s to refer to the secretions (the gall-inducing factors) of gall-inducing insects (e.g. ref. 127). This usage was incorrect, since the physical action of the insect and chemical secretions stimulate growth in the affected tissue and do not kill them, although an insignificant level of necrosis may manifest during early stages in interactions, especially in some species of the less-evolved groups such as the Thysanoptera and Eriophyroidea. Osmotic change-related metabolic pressure builds up when gall-inducing insects attack plant cells, activating a train of events in the immediate environment of those plant cells delicately punctured by the feeding or ovipositing insect. The latter sequence of events includes alterations in gas exchange and synthesis of growth promoters. Gall induction involves the vigorous uptake of oxygen, stimulating auxin activity. Osmotic stress alters electrical properties of the plasma membrane and impacts on IAA synthesis and activity, which, in turn, alters H+ transport. From what we know thus far, it is possible to infer that the plant actively mobilizes energy and nutrients to mitigate the stress and repair the wound from the time of attack by the insect. The insect incidentally utilizes the energy and nutrients mobilized at this site to its advantage.

Carango et al. in the 1980s and Schönrogge et al. and Hearn et al. in recent times clarify that the initial-most stage in galls (the metaplasied cell?) is triggered by proteins. No third-party organisms such as virus-like particles have been detected in Biorhiza pallida (Cynipidae)-induced galls on F. sylvatica. However, Hearn et al. show many differentially and highly expressed genes in young B. pallida larvae encoding secretory peptides – the possible effecter proteins – transmitted into F. sylvatica. The arabino-galactan proteins of F. sylvatica and chitin from B. pallida interact in young galls arising on F. sylvatica. The B. pallida larvae express genes encoding multiple plant cell-wall degrading enzymes.

Plants hosting gall-inducing insects employ varied strategies to neutralize the stress that arises during gall induction and growth-and-differentiation phases. These stress-neutralizing strategies are necessarily dictated by the genetic constitution of plants, but their responses are mediated by molecular changes, varying with the kind of the involved insect. The variation in the strategies could be due to the physiology of action and the nature of chemicals acting in the process. Yet, to generalize, the feeding biology of Cecidomyiidae and the oviposition biology of Cynipidae are useful models. In the context of gall induction, susceptible plants use a flexible, short-term strategy responding to stress inflicted by the insect. That short-term strategy involves mobilization of energy and other metabolites to the wounded site as a reparative effort to heal the wound, which the inducing insect exploits for its nourishment. This point is fully clear when we realize that the plant returns to its normal physiology the moment the insect ceases to feed. Genetic factors play a role in controlling the shape of the gall, coordinated by the innate correlating morphogenetic factors that operate normally in the plant.

Conclusion

In spite of unveiling details of several galls and inducing insects of different groups, our understanding of the physiology of gall induction is a conundrum. We are in a state similar to that which existed between the times of Hooke (1660s) on the one hand, and Schleiden and Schwann (1830s) on the other, in explaining the cell. Every biologist interested in explaining gall induction has broached it in the way he/she considered the best using insects of various groups that display varied feeding and oviposition behaviours. Curiously, each of these investigators found an answer and unhesitatingly suggested what they found was ‘the’ answer to the nagging question.

In short, a generalizable answer to how galls are induced is still elusive. First, we lack a precise definition of a gall. Any abnormality with the involvement of an insect is conveniently, but incorrectly, referred as a gall. Second, from what we know today, a gall induced by a less-evolved insect follows a distinctly different
developmental process from that induced by a better-evolved insect. Such varied biologies make them ambivalent. A thorough understanding of the basic biological processes occurring during early stages of interaction between the insect and the plant—clarifying the role of chitin supported by carefully designed biochemical and molecular studies—is the immediate need. There are difficulties, of course. Subjecting a metaplasied cell to biochemical quantification using sophisticated instrumentation is hard. A smart combination of biochemistry and sophisticated microscopy combined with various omics tools should offer insights into the molecular physiology of the metaplasied cell and the events that follow during the early phase of gall development, answering the long-pending question on how galls are induced. Nevertheless, looks-like we still have a long way to go.

1. Skuhrová, M. and Skuhrový, V., Gall midges (Diptera: Cecidomyiidae) of Italy. *Entomologica*, 1994, 28, 45–76.
2. Yukawa, J. and Partomihardjo, T., Insect and mite galls collected from Peucang, Panaltan, and the Krakatau Islands, Indonesia. *Tropics*, 1997, 7, 141–152.
3. Blanche, R., *Life in A Gall*. CSIRO, Collingwood, Australia, 2012, p. 71.
4. Saleem, U. K. A. and Nasser, M., Insect-induced galls of the Malabar region, Southern India. *Orient. Insects*, 2015, 49, 165–197.
5. Wool, D., Gall-inducing aphids: biology, ecology, and evolution. *In Biology, Ecology, and Evolution of Gall-inducing Arthropods* (eds Raman, A. et al.), Science Publishers, New Hampshire, USA, 2005, pp. 73–132.
6. Costa, J. T., *The Other Insect Societies*. Harvard University Press, Harvard, 2006, pp. 215–244.
7. Chapman, T. W., Crespi, B. J. and Perry, S. P., The evolutionary ecology of eusociality in Australian gall thrips: a ‘model clades’ approach. In *Ecology of Social Evolution* (eds Korb, J. and Heinze, J.), Springer, Heidelberg, 2008, pp. 57–83.
8. Stone, G. N. and Schönroegge, K., The adaptive significance of insect gall morphology. *Trends Ecol. Evol.*, 2003, 18, 512–522.
9. Joy, J. B. and Crespi, B. J., Adaptive radiation of gall-inducing insects within a single host-plant species. *Evolution*, 2007, 61, 784–795.
10. Hardy, N. B. and Cook, L. B., Gall induction in insects: evolutionary dead-end or speciation driver? *BMC Evol. Biol.*, 2010, 10, 1257; http://www.biomedcentral.com/1471-2148/10/257 (accessed on 20 February 2020).
11. Raman, A., Adaptive radiation and diversification in gall-inducing insects: search for a pattern. *Dtsch. Entomol. Z.*, 2012, 59, 177–187.
12. Mani, M. S., *Ecology of Plant Galls*. W. Junk, The Hague, The Netherlands, 1964, p. 434.
13. Rohfritsch, O., Développement cécidien et rôle du parasite dans quelques galles d’arthropodes. *Marcellia*, 1971, 37, 233–339.
14. Harper, L. J., Schönroegge, K., Lim, K. Y., Francis, P. and Lichtenstein, C. P., Cynipid galls: insect-induced modifications of plant development create novel plant organs. *Plant, Cell Environ.*, 2004, 27, 327–335.
15. Nystyrakis, F., Nouvelle interprétation de la formation des cécidies. *C. R. Acad. Sci.*, 1946, 222, 1133–1134.
16. Nystyrakis, F., Zooécidies et substances de croissance. *C. R. Soc. Biol.*, 1947, 141, 1218–1219.
17. Nystyrakis, F., Phytohormones and inhibition of the croissance des organes vegetaux attaqués par des aphides. *C. R. Acad. Sci.*, 1948, 226, 746–747.
18. Nystyrakis, F., Nouvelles observations sur l’inhibition de la croissance des organes du prunier attaqués par *Anuraphis helichrysi*. *C. R. Acad. Sci.*, 1948, 226, 831–832.
19. Nystyrakis, F., Autres preuves sur la sécrétion d’auxine par certains insectes: un nouveau test, très sensible, pour le dosage des substances de croissance. *C. R. Acad. Sci.*, 1948, 226, 1917–1919.
20. Boysen-Jensen, P., Formation de galls by Mikroola fagi. *Physiol. Plant.*, 1948, 1, 95–108.
21. Guiscaře-Arillaga, J., Formation of galls in stems and leaves of sugar cane in response to injections of growth regulating substances. *Physiopathology*, 1949, 39, 489–493.
22. Beck, E. G., The nature of the stimulus in the *Solidago* gall induced by the larva of *Gnorimoschema galleasoldagiinis*. *Brookhaven Symp. Biol.*, 1954, 6, 235–251.
23. Nolte, H. W., Untersuchungen über die stofflichen Grundlagen der Gallenbildung. *Verh. Dtsch. Ges. Angew. Entomol.*, 1954, 12, 124–128.
24. Leatherdale, D., Plant hyperplasia induced with a cell-free insect extract. *Nature*, 1955, 176, 553.
25. Schäller, G., Untersuchungen zur Erzeugung künstlicher Pflanzengallen. *Marcellia*, 1968, 25, 131–153.
26. Hori, K. and Endo, M., Metabolism of ingested auxins in the bug *Lygus disponsi*: conversion of indole-3-acetic acid and gibberel- lin. *J. Insect. Physiol.*, 1977, 23, 1075–1080.
27. Hori, K., Insect secretions and their effect on plant growth, with special reference to hemipterans. In *Biologie of Insect-induced Galls* (eds Shorthouse, J. D. and Rohfritsch, O.), Oxford University Press, New York, USA, 1992, pp. 157–170.
28. Hori, K., Plant growth-regulating factor in the salivary gland of several heteropterous insects. *Comp. Biochem. Physiol.*, B, *Biochem.*, 1976, 53, 435–438.
29. Hori, K., Possible causes of disease symptoms resulting from the feeding of phytophagous Heteroptera. In *Heteroptera of Economic Importance* (eds Schaefer, C. W. and Panizzi, A. R.), CRC Press, Boca Raton, FL, USA, 2000, pp. 11–35.
30. Braun, A. C., A physiological basis for autonomous growth of the crown-gall tumor cell. *Proc. Natl Acad. Sci. USA*, 1958, 44, 344–349.
31. Rohfritsch, O. and Shorthouse, J. D., Insect galls. In *Molecular Biology of Plant Tumors* (eds Kahl, G. and Schell, J. S.), Academic Press, New York, 1982, pp. 131–152.
32. Anders, F., Aminosäuren als gallenerregende Stoffe der Reblaus *Viteus [Phylloxera] vitifoli Shimer*. *Experientia*, 1958, 14, 62–63.
33. Miles, P. W., Insect secretions in plants. *Annu. Rev. Phytopathol.*, 1968, 6, 137–164.
34. Küster, E., *Die Gallen der Pflanzen*, Hirzel, Leipzig, Germany, 1911, p. 437.
35. Sharma, A. et al., Salivary proteins of plant-feeding hemipteroids – implication in phytophagy. *Bull. Entomol. Res.*, 2014, 104, 117–136.
36. Oates, C. N. et al., Insect gallers and their plant hosts: from omics data to systems biology. *Int. J. Mol. Sci.*, 2016, 17(11), 1891; doi:10.3390/ijms17111891 (accessed on 8 March 2020).
37. Thomas, F., Beiträge zur Kenntnis der Milbengallen und Gallmilben. *Zeit. Gesam. Wissens.*, 1873 (1874), 42, 513–537.
38. Meyer, J., *Plant Galls and Gall Inducers*, Gebrüder Bornträger, Berlin, Germany, 1987, p. 283.
39. Wool, D., Gall ing aphids: specialization, biological complexity, and variation. *Annu. Rev. Entomol.*, 2004, 49, 175–192.
40. Sharma, A. et al., How do the free-living, lerp-forming, and gall-inducing Aphalaridae (Hemiptera: Pyllioidea) affect the nutritional quality of *Eucalyptus* leaves? *Ann. Entomol. Soc. Am.*, 2016, 109, 127–135.
41. Maresquelle, H.-J. and Meyer, J., Physiologie et morphogénese des galls d’origine animale (Zooécidées), Handb. Pflanzenphysiol., 1965, 15(2), 280–329.

42. Meyer, J. and Maresquelle, H.-J., Anatomie des Galles, Gräbrüder Bornträger, Berlin, 1983, p. 662.

43. Rohfritsch, O., Morphogenesis und Pflanzenkäfer. Ber. Deutsch. Bot. Ges., 1977, 90, 339–350.

44. Rohfritsch, O., Patterns in gall development. In Biology of Insect-induced Galls (eds Shorthouse, J. D. and Rohfritsch, O.), Oxford University Press, New York, USA, 1992, pp. 60–80.

45. Meyer, J., Problèmes actuels de cécidologie. Bull. Soc. Bot. Fr., 1969, 116, 445–481.

46. Raman, A., Cecidogenetic behavior of some gall-inducing thrips, psyllids, coccids, and gall midges, and morphogenesis of their galls. Orient. Insects, 2003, 37, 359–413.

47. Raman, A., Morphogenesis of insect-induced plant galls: facts and questions. Flora, 2011, 206, 517–533.

48. Wyss, U., Root parasitic nematodes: an overview. In Cellular and Molecular Aspects of Plant–Nematode Interactions: Developments in Plant Pathology (eds Fennoll, C. et al.), Springer, Dordrecht, 1997, vol. 10, pp. 5–22.

49. McAttee, P. et al., A dynamic interplay between phytohormones is required for fruit development, maturation, and ripening. Front. Plant Sci., 2013, 4, Article 79; doi:https://doi.org/10.3389/fpls.2013.00079 (accessed 15 April 2020).

50. Bronner, R., Propriétés lytiques des œufs de Diplolepis rosae (Cynipidae, Hymenoptera). J. Protozool., 2013, 60(4), 465–470.

51. Bronner, R., Anatomy of the ovipositor and ovipositional behavior of the gall wasp Diplolepis rosae (Cynipidae, Hymenoptera). Can. Entomol., 1985, 117, 849–858.

52. Yokawa, J. and Rohfritsch, O., Biology and ecology of gall-inducing Cecidomyiidae (Diptera). In Biology, Ecology, and Evolution of Gall-inducing Arthropods (eds Raman, A. et al.), Science Publishers, New Hampshire, USA, 2005, pp. 273–304.

53. Rohfritsch, O., Plants, gall midges, and fungi: a three-component system. Entomol. Exp. Appl., 2008, 128, 208–216.

54. Mani, M. S., Plant Galls of India, Science Publishers, New Hampshire, 2000, p. 477.

55. Gagné, R. J. and Jaschhof, M., Evolution of gall-inducing arthropods: an overview of their biology, ecology, and evolution. In Biology, Ecology, and Evolution of Gall-inducing Arthropods (eds Raman, A. et al.), Science Publishers, New Hampshire, USA, 2010, p. 430.

56. Sylvén, E., Gall midges (Diptera, Cecidomyiidae) as plant taxonomists. Symb. Bot. Upsal., 1979, XXII, 63–69.

57. Mound, L. A., Taxonomic confusion among gall thrips and host plants, with three new combinations from the genus Auctrothrips (Thysanoptera, Phlaeothripidae). Zootaxa, 2020, 4755(3), 587–592.

58. Miller, D. G. and Raman, A., Host-plant relations of gall-inducing insects. Ann. Entomol. Soc. Am., 2019, 112, 1–19.

59. Raman, A. and Ananthakrishnan, T. N., On the developmental morphology of the rosette galls of Acacia leucophloea Willd. (Mimosaceae) induced by Thilakothrips babuli Ramk. (Thysanoptera: Insecta). Proc. Indian Acad. Sci. Sect. B, 1983, 92, 343–350.

60. Yang, M. M. et al., Outbreak of erythrina gall wasp on Erythrina spp. (Fabaceae) in Taiwan. Plant Prot. Bull., 2004, 46, 391–396.

61. Kim, I.-K., Evolution of gall-inducing Euolophidae (Hymenoptera: Chalcidoidea) on Myrtaceae in Australia, Ph.D thesis, Australian National University, Canberra, 2008, p. 149, https://openresearch-repository.anu.edu.au/handle/1885/110000 (accessed on 8 December 2019).

62. Gagné, R. J., Revision of Prodiplossis (Diptera: Cecidomyiidae) with description of three new species. Ann. Entomol. Soc. Am., 1986, 79, 235–245.

63. Dhileepan, K. et al., Host associations of gall-inducing Prodiplossis longifilia (Diptera: Cecidomyiidae) from Bolivia: implications for its use as a biological control agent for Jatropha gossypifolia (Euphorbiaceae). Fl. Entomol., 2017, 100, 777–786.

64. Lovett, T. J., Some phytochemical changes in Taxus baccata L. (yew) shoots associated with stages in the life cycle of Taxomyia taxif. Bull. Soc. Bot. Fr. (Actual. Bot.), 1980–1981, 127, 129–136.

65. Sharma, A. et al., Feeding and oviposition behaviour of a gall-inducing species of Glycaspis (Synglycaspis) (Hemiptera: Psyli- loidea: Aphiidae) and development of galls on the leaves of Eucalyptus macrocarpha (Myrtaceae) in central western New South Wales, Australia. Eur. J. Entomol., 2015, 112, 75–90.

66. Brooker, M. I. H., A new classification of the genus Eucalyptus L’Hér. (Myrtaceae). Aust. Syst. Bot., 2000, 13, 79–148.

67. Sharma, A. et al., Complex lipids and sterols in the leaves of Eucalyptus macrocarpha (Myrtaceae) in the context of feeding by an unnamed gall-inducing species of Glycaspis (Synglycaspis) (Hemiptera: Psylioloidea: Aphiidae). Ann. Entomol. Soc. Am., 2016, 109, 890–898.

68. Sharma, A. and Raman, A., Feeding biology and nutritional physiology of Psylioloidea (Insecta: Hemiptera): implications in host-plant relations. Curr. Sci., 2017, 113, 1543–1552.

69. Ananthakrishnan, T. N. and Raman, A., Thrips and Gall Dyna- mics, E. J. Brill, Leiden, The Netherlands, 1989, p. 120.

70. Rohfritsch, O., Relations hôte – parasite au début de la cécidogènèse du Hartigioila annulipes Hartig sur le hêtre. Bull. Soc. Bot. Fr. (Actual. Bot.), 1980–1981, 127, 199–207.

71. Raman, A., Beiderbeck, R. and Herth, W., Early subcellular re- sponses of susceptible and resistant Pitia taxa to feeding by grape phyllloxera Daktulosphaira vitifoliae. Bot. Helv., 2009, 119, 31–39.

72. Rey, L., Les premiers stades de développement de la galle de Pontania proxima Lep. Bull. Soc. Bot. Fr., 1967, 115, 413–424.

73. Rey, L., Les premiers stades du développement de la galle de Biorzicha pallida Ol. Marcella, 1969, 36, 119–135.

74. Shorthouse, J. D., Galls induced by cynipid wasps of the genus Diplolepis (Hymenoptera: Cynipidae) on the roses of Canada’s grasslands. In Arthropods of Canadian Grasslands: Ecology and Interactions in Grassland Habitats (eds Shorthouse, J. D. and Floate, K. D.), Biological Survey of Canada, Ontario, 2010, 1, 251–279.

75. Albersheim, P. et al., Plant Cell Walls, Taylor & Francis Group, New York, USA, 2010, p. 430.

76. Raman, A., Schaefer, C. W. and Withers, T. M., Galls and gall-inducing arthropods: an overview of their biology, ecology, and evolution. In Biology, Ecology, and Evolution of Gall-inducing Arthropods (eds Raman, A. et al.), Science Publishers, New Hampshire, 2005, pp. 1–33.

77. Jiang, F., Zhu, J. and Liu, H.-L., Protoplasts: a useful research system for plant cell biology, especially dedifferentiation. Protoplasma, 2013, 250, 1231–1238.

78. Küster, E., Pathologische Pflanzenanatomie, Gustav Fischer, Jena, Germany, 1903, p. 312.

79. Goldthwaite Jr. C. A., Are stem cells involved in cancer? In Regenerative Medicine, National Institutes of Health, Department of Health and Human Services, USA, August 2006; https://stemcells.nih.gov/info/Regenerative_Medicine.htm (accessed on 8 February 2020).

80. Stewart, F. C., Ammirato, P. V. and Mapes, M. O., Growth and development of totipotent cells. Ann. Bot., 1970, 34, 761–787.

81. Graf, T., Historical origins of transdifferentiation and reprogram- ming. Cell Stem Cell., 2011, 9, 504–516.

82. Hatchett, J. H., Kretner, G. L. and Elzinga, R. J., Larval mouthparts and feeding mechanism of the Hessian fly (Diptera: Cecidomyiidae). Ann. Entomol. Soc. Am., 1990, 83, 1137–1147.
83. Le Relac, A., Rabasse, J. M. and Wajnberg, E., Comparative morphology of the ovipositor of some parasitic Hymenoptera in relation to characteristics of their hosts. Can. Entomol., 1996, 128, 413–433.

84. Dettmer, J. and Frimmel, J., Cell polarity in plants: when two do the same, it is not the same. Curr. Opin. Cell Biol., 2011, 23, 686–696.

85. Carango, P. et al., Induction of a 58,000 Dalton protein during goldenrod gall formation. Biochem. Biophys. Res. Comm., 1988, 152, 1348–1358.

86. Doss, R. P. et al., Bruchins: insect-derived plant regulators that stimulate neoplasmat formation. Proc. Natl. Acad. Sci. USA, 2000, 97, 6218–6223.

87. Farmer, E. E., Potential mitogenic lipids from gall-inducing insects. Trends Plant Sci., 2000, 5, 359–360.

88. Westphal, E., Bronner, R. and Le Rot, M., Changes in leaves of susceptible and resistant Solanum dulcamara infested by the gall mite Eriophyes cladophthirus (Acaria, Eriophyidae). Can. J. Bot., 1981, 59, 875–882.

89. Breusegem, F. V. and Dat, J. F., Reactive oxygen species in plant cell death. Plant Physiol., 2006, 141, 384–390.

90. Zechmann, B., Ultrastructure of plasidts serves as reliable abiotic and biotic stress marker. PLoS ONE, 2019, 14(4), e0214811; https://doi.org/10.1371/journal.pone.0214811 (accessed on 20 July 2020).

91. Gilbert, G. A., Knight, J. D., Vance, C. P. and Allan D. L., Acid phosphatase activity in phosphorus-deficient white lupin roots. Plant Cell Environ., 1999, 22, 801–810.

92. Bronner, R., Contribution à l’étude histochimique des tissus nourriciers des zoécidées. Marcellia, 1977, 40, 1–134.

93. Bronner, R., The role of nutritive cells in the nutrition of cynipids. Phytopathologia, 2009, 98, 263–270.

94. Price, P. W., Fernandes, G. W. and Waring, G. L., Oxidative stress, and signal transduction. York, 1992, pp. 117–192.

95. Dettmer, J. and Friml, J., Cell polarity in plants: when two do the relation to characteristics of their hosts. Trends Plant Sci., 2016, 21, 198–206.

96. Carango, P. et al., Induction of a 58,000 Dalton protein during goldenrod gall formation. Biochem. Biophys. Res. Comm., 1988, 152, 1348–1358.

97. Doss, R. P. et al., Bruchins: insect-derived plant regulators that stimulate neoplasmat formation. Proc. Natl. Acad. Sci. USA, 2000, 97, 6218–6223.

98. Farmer, E. E., Potential mitogenic lipids from gall-inducing insects. Trends Plant Sci., 2000, 5, 359–360.

99. Westphal, E., Bronner, R. and Le Rot, M., Changes in leaves of susceptible and resistant Solanum dulcamara infested by the gall mite Eriophyes cladophthirus (Acaria, Eriophyidae). Can. J. Bot., 1981, 59, 875–882.

100. Breusegem, F. V. and Dat, J. F., Reactive oxygen species in plant cell death. Plant Physiol., 2006, 141, 384–390.

101. Zechmann, B., Ultrastructure of plastidts serves as reliable abiotic and biotic stress marker. PLoS ONE, 2019, 14(4), e0214811; https://doi.org/10.1371/journal.pone.0214811 (accessed on 20 July 2020).

102. Gilbert, G. A., Knight, J. D., Vance, C. P. and Allan D. L., Acid phosphatase activity in phosphorus-deficient white lupin roots. Plant Cell Environ., 1999, 22, 801–810.

103. Apel, K. and Hirt, H., Reactive oxygen species: metabolism, oxidative stress, and signal transduction. Annu. Rev. Plant Biol., 2004, 55, 373–399.

104. Westphal, E., Modification du pH vacuaire des cellules épidermiques foliaires de Solanum dulcamara soumises à l’action d’un acarian cécidogène. Can. J. Bot., 1982, 60, 2882–2888.

105. Westphal, E., Dreger, F. and Bronner, R., The gall mite Aceria cladophthirus (Nalepa). I. Life cycle, survival outside the galls and symptoms expression on susceptible and resistant Solanum dulcamara L. plants. Exp. Appl. Acarol., 1990, 9, 183–200.

106. Price, P. W., Fernandez, G. W. and Waring, G. L., Adaptive nature of insect galls. Environ. Entomol., 1987, 16, 15–24.

107. Diamond, S. E., Blair, C. P. and Abrahamson, W. G., Testing the physiology of gall induction by Sternorrhyncha, ract. Raman, A., Gall induction by hemipteroid insects.

108. Raman, A. et al., Metabolite mobilization in the stem galls of Parthenium hysterophorus induced by Epiblema strenuana inferred from the signatures of isotopic carbon and nitrogen and concentrations of total non-structural carbohydrates. Entomol. Exp. Appl., 2006, 119, 101–107.

109. Byers, J. A., Brewer, J. W. and Denna, D. W., Plant growth hormones in pinyon insect galls. Marcelia, 1976, 39, 125–134.

110. Yamaguchi, H. et al., Phytohormones and willow gall induction by a gall-inducing sawfly. New PhytoL, 2012, 196, 586–595.

111. McCalla, D. R., Genthe, M. K. and Hovavitz, W., Chemical nature of an insect gall growth-factor. Plant Physiol., 1962, 37, 98–103.

112. Tooker, J. F. and De Moraes, C. M., A gall-inducing caterpillar species increases essential fatty acid content of its host plant without concomitant increases in phytohormone levels. Mol. Plant–Microbe Interact., 2009, 22, 551–559.

113. Palm, J. M. et al., Fruit ripening: from present knowledge to future development. Front. Plant Sci., 2019; https://doi.org/10.3389/fpls.2019.00545 (accessed on 25 July 2020).

114. Arenas, Y. A. et al., Functional analysis and mode of action of phytoxic Nep1-like proteins of Botrytis cinerea. Physiol. Plant Pathol., 2010, 74, 376–386.

115. Siewers, V. et al., Functional analysis of the cytochrome P_{450} monooxygenase gene behotl of Botrytis cinerea indicates that botrytid is a strain-specific virulence factor. Mol. Plant–Microbe Interact., 2005, 18, 602–612.

116. Newman, M.-A. et al., MAMP (microbe-associated molecular pattern) triggered immunity in plants. Front. Plant Sci., doi:10.3389/fpls.2013.00139 (accessed on 10 March 2020).

117. Agrawal, G. K. et al., Plant organelle proteomics: collaborating for optimal cell function. Mass Spectrom. Rev., 2011, 30, 772–853.

118. Gozzo, F. and Faoro, F., Systemic acquired resistance (50 years after del capo della larva matura de Diplolepis triforma (Hymenoptera: Cynipidae)). In: Arthropods and Fungi in Animal Agriculture, Environmental Impact and Management, 2003, 813–828.

119. Solinas, M., Morfologia, anatomia e organizzazione funzionale del capo della larva matura de Phaenobrevia aphidimyza (Ronanni). Entomologica, 1968, 4, 7–44.

120. Collinge, D. B. et al., Plant chitinases. Plant J., 1993, 3, 31–40.

121. Wojcik, A. et al.,Conservation of the chitinase gene family in rice (Oryza sativa L.). Plant J., 2001, 26, 561–570.

122. Cerkvenik, U. et al., Softness gradients facilitate ovipositor bending and spatial probing control in a parasitic wasp. J. Exp. Biol., 2019, 222, jeb195628, doi:10.1242/jeb.195628 (accessed on 20 April 2020).

123. Szulczewski, L. et al., The role of the chitinase gene family in the biology of rice. Plant J., 2001, 26, 561–570.
125. Raman, A., Visionary words and realistic achievements: one hundred years of cecidology. *Formos. Entomol.*, 2018, **38**, 5–24.
126. Harris, M. O. and Pitzschke, A., Plants make galls to accommodate foreigners: some are friends, most are foes. *New Phytol.*, 2020, **225**, 1852–1872.
127. Carter, W., *Insects in Relation to Plant Disease*, Interscience (Wiley), New York, USA, 1962, p. 705.
128. Westphal, E. and Manson, D. C. M., Feeding effects on host plants: gall formation and other distortions. *World Crop Pests*, 1996, **6**, 231–242.
129. Miles, P. W., Aphid saliva. *Biol. Rev.*, 1999, **74**, 41–85.
130. León, J., Rojo, E. and Sánchez-Serrano, J. J., Wound signalling in plants. *J. Exp. Bot.*, 2001, **52**, 1–9.
131. Schönrogge, K. *et al.*, Reprogramming plant development: two approaches to study the molecular mechanism of gall formation. In *The Biology of Gall-inducing Arthropods*, General Technical Report NC 199 (eds Csóka, W. *et al.*), USDA Forest Service, St. Paul, Minnesota, USA, 1998, pp. 153–160.
132. Hearn, J. *et al.*, Genomic dissection of an extended phenotype: oak galling by a cynipid gall wasp, *PLoS Genet.*, 2019, **15**(11), e1008398; https://doi.org/10.1371/journal.pgen.1008398 (accessed on 22 May 2020).
133. Wolpert, L., Evolution of the cell theory. *Philos. Trans. Biol. Sci.*, 1995, **349**, 227–233.
134. Raman, A., Burckhardt, D. and Harris, K. M., Biology and adaptive radiation in the gall-inducing Cecidomyiidae (Insecta Diptera) and Calophyidae (Insecta Hemiptera) on *Mangifera indica* (Anacardiaceae) in the Indian subcontinent. *Trop. Zool.*, 2009, **22**, 27–56.
135. Burckhardt, D., Sharma, A. and Raman, A., Checklist and comments on the jumping plant-lice (Hemiptera: Psylloidea) from the Indian subcontinent. *Zootaxa*, 2018, **4457**(1), 1–38.

**ACKNOWLEDGEMENTS.** I thank Laurence Mound (Australian National Insect Collection, CSIRO, Canberra, Australia), Soundararajan Madhavan (University of Nebraska, Lincoln, USA), and Krishnapappa Chandrashekar (University of Agricultural Sciences, Bengaluru) for reviewing the final draft of this manuscript and Anamika Sharma (IPM Innovation Lab, Virginia Tech, Blacksburg, USA) for help in organizing the images.

Received 16 October 2020; accepted 2 November 2020
doi: 10.18520/cs/v120/i1/66-78