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Traditional Knowledge Meets Molecular Networks: A Systems-Level Re-evaluation of Medicinal Plant Bioactivity

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ABSTRACT: Traditional medicinal knowledge has played a significant role in the identification of bioactive plants through the many times it has led to the identification of those plants, however, there have been many times when traditional evaluation turned into isolated compounds and single targets. The review revisits the bioactivity of medicinal plants by systematically linking ethnobotanic knowledge, phytochemical diversity, molecular networks, multi-omics information, computational prediction and experimental validation. The relationship among constituents, targets, pathways, diseases, and phenotypes can be mapped using network pharmacology; the high throughput of metabolomics, transcriptomics, and proteomics, molecular docking, and machine learning can provide mechanistic plausible leads for the prioritisation. But network diagrams are models, not realities; authenticated plant material, quantitative chemical standardisation, exposure responsive target assessment, orthogonal assays, causal perturbation, reproducibility, pharmacokinetics and clinically relevant outcomes are all necessary to achieve reliable translation.

Whole-extract effects cannot be inferred from individual compounds, as effects may be additive, synergistic, antagonistic or via bioavailability modification. Technical innovation should be accompanied by ethical governance, protection of indigenous knowledge, benefit sharing, biodiversity conservation and quality standards for regulations. The computational-experimental cycle can largely retain the context of traditional knowledge

whilst transforming complex botanical preparations into clinical, reproducible and testable therapeutic hypotheses.

1. INTRODUCTION

The use of medicinal plants continues to be significant in the areas of medicinal ideas, chemical scaffolds, and culturally embedded health practices. The ethnobotanical observations can inform discovery, and can aid in the identification of species used, preparations used, indications used, that have been selected for humans that have been used repeatedly, but do not prove efficacy or safety (1). Natural products have been directly or indirectly contributing to the current discovery of the majority of modern medicines and there has been a development in analytical chemistry, genomics and computational biology to make them increasingly relevant to the drug discovery process (2,3,24,25). The big problem is the medicinal plants are not monocomponents. They are rich in diverse chemical molecules in concentration that varies according to taxonomy, geography, cultivation, harvest, storage and processing. Traditional studies often test a single constituent in a single target, and then extrapolate findings to the entire preparation. For identification of potent molecules, this reductionistic approach can be helpful, but can fall short in recognizing complementary target engagement, pharmacokinetics interactions, pathway buffering and effects of biological matrix. The view from systems biology and network pharmacology is different: Bioactivity is considered an emergent property of the interactions among the components of the system—compounds, targets, pathways, tissues, phenotypes (7,8). This review merges traditional knowledge with molecular-network analysis, elucidates the experimental steps needed to test the computational hypotheses and the requirements to move complex plant derived interventions into predictable and clinically pliable products. A systems perspective is particularly important for chronic, heterogeneous disorders that involve multifactorial and dynamic interplay between inflammatory, metabolic, vascular, neuronal and behavioural processes. These environments might be more informative if the effects of the several nodes are modest rather than a generalized target maximally inhibited, provided the overall exposure is not harmful and the affected pathway is relevant for the disease. The common language provided in the framework also facilitates communication among ethnobotanists, analytical chemists, pharmacologists, computational chemists, clinicians and regulators.

2. TRADITIONAL KNOWLEDGE AS A SOURCE OF THERAPEUTIC LEADS

Traditional knowledge does not merely consist of a list of medicinal species. Consists of plant part used, preparation method, dose form, route, combinations, perceived indications, contraindications, and social context of treatment. These pieces of information can add to biological plausibility and sampling guidance, but their incorporation needs to be well-documented, botanically authenticated, and based on clear rationale and respect for local knowledge holders. A number of successes, such as the discovery of antimalarial and analgesic compounds, have highlighted the importance of knowledge-driven discovery; and a few failures have illustrated that the traditional reputation of a substance can be misinterpreted for reasons of misidentification, substitution, commercial promotion, or selective reporting (1,28). A systems approach starts with ethnomedical observations, but does not rely on them as unqualified proof, but instead converts them to structured variables. Candidates can be prioritised using consensus measures, frequency of citation, cross-cultural convergence, preparation fidelity and relevance to disease. The selected material should be associated with voucher specimens, accepted botanical names, collection metadata and reproducible processing. In such a traceability, in the absence of any molecular network, noise is reduced before it becomes an issue. Digital ethnobotanical repositories can then link community-based content to phytochemical, pharmacological, toxicological and ecological information. The integration maintains the richness and context of traditional practice whilst creating explicit hypotheses that can be tested in the lab and evaluated clinically. Strength and independence of observations should also be taken into account in prioritisation. If several different communities are using the same text multiple times, it might be convergent experience; if a source from the contemporary period is cited multiple times, it may have been copied between texts. Preparation details need to be kept due to the fact that the chemical composition and bioavailability may change after Boiling or Fermentation and Lipid co-administration or Polyherbal combination. It is then as critical to record uncertainty and variation as it is to record consensus.

3. PHYTOCHEMICAL COMPLEXITY AND MULTI-COMPONENT THERAPEUTIC ACTIVITY

A medicinal plant can be a source of hundreds of primary and secondary metabolites, with only a few of them achieving pharmacologically relevant doses in relevant tissues. The observed effects can thus be construed as either a direct interaction of the target, an increase in activity due to interaction with multiple targets, synergy, antagonism, altered absorption or metabolism, or a protection of the target which might be unstable in the extract matrix. An assumption about synergy is likely to be incorrect unless one can use the proper model and concentration ranges to quantitatively compare the mixture with the additive effects expected (5,6). Likewise, if the compound has low in vivo activity because its in vivo bioavailability is low, its in vivo metabolism is rapid, it is unstable, or its in vivo concentration is low, it will be poorly active in vivo despite being highly active in vitro. Although whole extracts can sometimes be more effective, they can also be a dilution of the plant's active principles or toxicity and variable (27). The complex situation can be dealt with at a system level by making a combination of quantitative chemical profile, exposure estimates and target/pathway information which is in line with the multi-target therapeutic concept of phytotherapy (26). It doesn't ask which molecule binds best, it asks which collection of constituents is always available, bioavailable, and can affect a disease-relevant network without unwanted side effects. Reproducible chemical signatures can be identified using chemometric fingerprinting, batch comparison, metabolite annotation and the features associated with activity. These signatures are more helpful for standardisation than a single signature not related to efficacy. Representative plants as multi-component interventions across interconnected pathways instead of one-compound interventions is demonstrated in Table 1. Temporal behaviour adds to the confusion. Exposure of a component can be affected by enzyme inhibition, rapid absorption can trigger a signal and slower absorption can maintain it. Others may be inactive against the disease target, but have increased solubility, intestinal transport, or tissue distribution and/or enhanced chemical stability. On the other hand, beneficial relationships might result in a reduction of the safety margin. Pharmacokinetic and toxicity networks should be therefore included in network models in addition to efficacy networks when available.

Table 1: Representative medicinal plants, major phytochemicals, traditional-use contexts, and systems-level evidence considerations

Medicinal plant	Traditional-use context	Representative constituents	Systems-level targets/pathways and evidence considerations
Curcuma longa L. (turmeric)	Inflammatory conditions and wound-related uses	Curcuminoids; turmerones	NF-kB, COX/LOX, Nrf2, cytokine and redox networks; oral exposure and formulation strongly influence interpretation.
Withania somnifera (L.) Dunal	Stress, weakness, sleep and restorative preparations	Withanolides; sitoindosides	Stress-response, inflammatory, neuroendocrine and proteostasis pathways; extract composition and root/leaf differences require control.
Panax ginseng C.A.Mey.	Fatigue, vitality and metabolic support	Ginsenosides	AMPK, PI3K-AKT, inflammatory and neuroendocrine signalling; metabolites produced by gut microbiota may mediate activity.
Camellia sinensis (L.) Kuntze	Stimulant, cardiometabolic and antioxidant use	Catechins, especially EGCG; caffeine	Redox, AMPK, lipid, vascular and inflammatory networks; dose, beverage matrix and caffeine modify effects.

Medicinal plant	Traditional-use context	Representative constituents	Systems-level targets/pathways and evidence considerations
Artemisia annua L.	Febrile illness and malaria-related traditions	Artemisinin and related sesquiterpenes	Heme-dependent parasite toxicity plus possible partner-compound interactions; therapeutic use requires standardised antimalarial regimens.
Ginkgo biloba L.	Memory and circulatory complaints	Flavonol glycosides; terpene lactones	Platelet-activating factor, vascular, mitochondrial and oxidative-stress pathways; evidence applies to defined standardised extracts, not arbitrary products.
Nigella sativa L.	Respiratory, metabolic and inflammatory complaints	Thymoquinone; volatile and fixed oils	Inflammatory, redox, metabolic and immune pathways; constituent stability and achievable systemic exposure remain key.
Glycyrrhiza glabra L.	Cough, gastric and inflammatory preparations	Glycyrrhizin/glycyrrhetinic acid; flavonoids	Cortisol metabolism, inflammatory and antiviral pathways; mineralocorticoid-like adverse effects illustrate the need for network safety analysis.

4. FROM REDUCTIONIST PHARMACOLOGY TO SYSTEMS-LEVEL EVALUATION

Reductionist pharmacology simplifies a molecule's activity to demonstrate causal relationships, and it is important to establish molecular mechanisms. It is limiting when an outcome that is the focus of treatment is considered as the whole answer. Network medicine acknowledges that disease can arise from dysregulated modules of interconnected genes, proteins, metabolites, cells and organs, and network pharmacology tries to find interventions that target multiple sites of this system (7,8). In medical research of medicinal plants, it is moving from the rigorous experiments to the descriptive networks, by a passing from isolated observations to an organized framework in which each claim is linked to the chemical identity, to exposure, to the target engagement, to the pathway modulation, and to the phenotype. Convergent targets may be found using compound-target networks, hubs and modules can be uncovered using protein-protein interaction networks, and enrichment in biological pathways can provide a general idea of the biological processes impacted by an extract. However, centrality does not equal therapeutic value and enrichment may be biased by the database or by large collections of genes. Systems-level evaluations should then rank evidence, separate predicted from measured linkages, consider direction and dose if available and include negative results. In re-evaluation (see figure 1), traditional knowledge guides plant selection, phytochemical analysis works out the details of the plant composition that constitutes an intervention, molecular networks can propose hypotheses that can be tested and experimental evidence allows either to determine that the proposed network explains the desired clinical effect, or, if this is not the case, provide insights into alternative networks.

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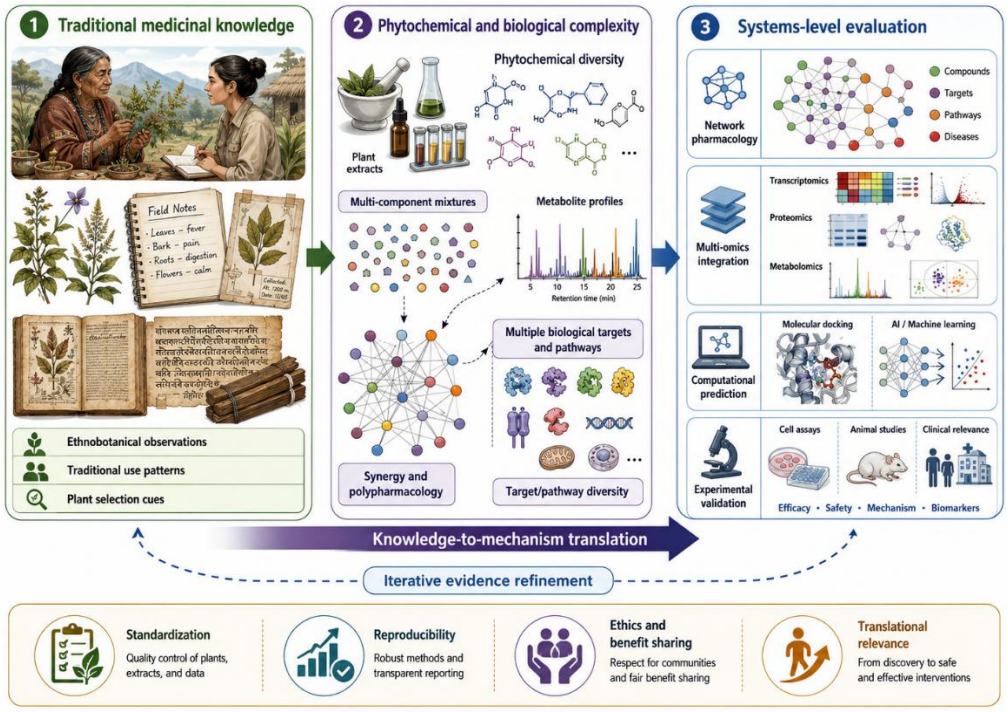

Figure 1: Transition from traditional medicinal knowledge to systems-level evaluation of medicinal plant bioactivity.

5. NETWORK PHARMACOLOGY IN MEDICINAL PLANT RESEARCH

From a list of literature-based, pharmacologically based or chemically profiled compounds, network pharmacology typically follows a multi-component, multi-target approach (9). Experimentally curated sources, similarity, docking or machine-learning models are then used to assign putative protein targets. Mechanistic hypotheses are generated through the compilation of disease-associated genes, the mapping of overlaps, the construction of protein-protein interaction networks and the analysis of pathway enrichment. Herbal systems research can be aided by databases like TCMSP, chemical information and bioassays are available from PubChem, SwissTargetPrediction suggested possible targets based on molecular similarity, STRING can be used to infer protein associations, and pathway and biological functions information can be obtained from database resources like KEGG and Gene Ontology databases (10-17). Cytoscape is a popular tool to visualize compound-target-pathway relationships (14). While this workflow is efficient it can give a false sense of certainty. Literature-derived compound lists do not necessarily reflect the tested extract; predicted targets are often missing concentration information, metabolism, tissue exposure, stereochemistry, and the context of the assay; disease-gene lists are likely to be heterogenous; and protein-interaction edges are likely to be indirect or come from a different tissue context. High degree nodes like AKT1, TP53, inflammatory mediator etc. are often observed, as they are well studied but do not necessarily have a unique role in the plant effect. Strong studies, then, start by analytical confirmation of constituents, use clear inclusion criteria, include database versions and prediction contexts and differentiate between direct evidence and inferences. Consideration of the directionality, tissue specificity and pharmacokinetics should also be included in networks if data are available. The output is best considered as a prioritised hypothesis map, i.e., a list of combinations of compounds, targets and pathways that are worth testing but may not replace target engagement, causal perturbation or replicated phenotypic evidence. The evidence-weighted construction leads to better interpretability. Evidence for plant compound associations should be separated from entries found in the text; confirmation of binding experimentally in plants should be separated from binding reported in human tissues or in other species or cell lines; and when plant compound binding is measured, it should be clearly separated from predicted binding. Conclusions can be tested for stability after removal of low confidence edges or highly connected generic targets through sensitivity analyses. Overinterpretation can be mitigated by comparing the medicinal-plant network with the network from a random selection or the network with the same number of nodes as the medicinal network and the same average degree. Abundance and exposure can be used to weight compound-target edges if the concentration of the constituents is known, rather than treating all compound-related molecules as equally important.

6. INTEGRATION OF OMICS AND COMPUTATIONAL TECHNOLOGIES

Omics technologies can serve as a backbone for measured biological responses in molecular networks. Metabolomics describes the metabolism of the intervention and the host metabolism, transcriptomics defines the patterns of gene expression that accompany the intervention, proteomics provides information on the changes in functional machinery and microbiome analysis may provide information on microbial biotransformation or host-microbe interactions. Integrated analysis can also compare the identified predicted pathway targets to actual pathways shifts, which can aid in identifying mechanisms that are likely from the database artefacts. HMDB and MetaboAnalyst facilitate metabolite interpretation and pathway analysis; meanwhile, modern natural-product workflows utilize feature-based molecular networking, chemometrics and dereplication to prioritize metabolites of interest for subsequent isolation without exhaustive efforts (18-20). While molecular docking can offer useful suggestions about modes of binding, the scoring depends on the protein structure, protonation and flexibility, and on what assumptions have been made regarding the scoring, and therefore should not be used as a replacement for biochemical validation (23). While machine learning can rank compounds, predict bioactivity or ADMET properties, and discover patterns in high-dimensional data, the quality of the models relies on the quality of training sets, chemical-domain coverage, class balance, and independent validation, and as a result, the models' performance can be poor (21). There is a special need for explainable approaches when predictions play a role in experimental or clinical decisions (22). A comprehensive pipeline that incorporates multiple evidence layers: chemical abundance, predicted and measured exposures, target confidence, multi-omics concordance, pathway relevance, and safety. In addition, it also maintains uncertainty instead of bringing down all evidence to one network score. Table 2 outlines some key resources and who they are suitable for. Smallest coherent set of hypotheses that can be tested by orthogonal methods and that explains desired and adverse phenotypes is the most informative output that does not have to be the largest network. The study design is extremely important, as you can't save shoddy primary data with integration. Enough biological replicates, random processing of samples, QC samples for random processing, rejecting batch-effects, and pre-specified statistical thresholds are required for omics experiments. When the transcript, protein, metabolite and phenotype changes all point to the same direction of action of the mechanism, it is more convincing. Target engagement can be differentiated from later "compensatory" responses by using time-series data. If machine-learning modeling, training and test sets will be separated by chemical scaffold, or by external cohort, to prevent an over-optimistic performance due to near-duplicate compounds or external samples in the set.

Table 2: Major systems-biology and computational resources used in medicinal plant bioactivity evaluation

Component	Representative resources or methods	Primary role	Key limitation and validation need
Chemical identity and bioassays	PubChem; curated literature; LC-MS/MS and NMR	Confirm structures, activities and measured constituents	Database entries may not match the tested extract; verify analytically.
Herbal systems databases	TCMSP and related curated repositories	Link herbs, compounds, properties and targets	Coverage and curation vary; report database version and inclusion rules.
Target prediction	Swiss Target Prediction; ligand-similarity or ML models	Prioritise likely protein targets	Predictions ignore many exposure and tissue constraints; confirm target engagement.
Protein interaction networks	STRING	Identify interacting proteins, hubs and modules	Edges may be indirect or context-free; use tissue-specific evidence where possible.

Component	Representative resources or methods	Primary role	Key limitation and validation need
Network visualisation	Cytoscape	Build compound-target-pathway and disease networks	Visual centrality is not causal importance; retain evidence weights and uncertainty.
Pathway and function analysis	KEGG; Gene Ontology	Interpret enriched pathways and biological processes	Large or biased gene sets can inflate significance; use corrected statistics and controls.
Metabolomics and integration	HMDB; MetaboAnalyst; chemometrics	Characterise intervention and host-response metabolites	Annotation ambiguity and batch effects require standards, QC samples and orthogonal confirmation.
Structure-based modelling	AutoDock Vina; molecular dynamics	Generate binding hypotheses and rank poses	Scores are approximate; validate using biochemical and biophysical assays.
Machine learning / AI	QSAR, graph models, multimodal integration	Rank bioactivity, ADMET and multi-omic patterns	Domain shift and opacity can mislead; require external validation and explainability.

7. EXPERIMENTAL VALIDATION OF MOLECULAR NETWORKS

Experimental evidence should move from identity of the intervention to the proof of causality of the mechanism. To obtain plant material for extraction, it should undergo authentication and be extracted in a controlled environment and compared via quantitative fingerprints and relevant marker ranges (4,30). Predicted fractions or compounds to induce network activity can then be evaluated in biochemical, cellular, organoid or ex vivo models. Concentration response studies should be conducted using exposures that can be achieved in the biological system and orthogonal assays should be used to minimize the artefacts of the method. Multiple approaches that can be used to examine the target engagement include binding assays, thermal profiling, affinity systems, reporter systems, or downstream biomarker. Causal relevance: perturbation (gene knockdown, pharmacological blockade, rescue experiments, pathway-specific interventions) should affect the predicted effect. While the statistical enrichment may suggest that network-level changes are expected in the correct direction, multi-omics measurements confirm these directions. Without replication and functional validation, however, statistical enrichment alone cannot confirm a networks directionality. Pharmacokinetics, tissue distribution, efficacy, toxicity and interactions between components should be assessed in vivo. Whole extract, reconstituted mixtures and isolated compounds can help identify if the activity of the extract is additive, synergistic, antagonistic, and/or matrix-dependent. Randomisation, blinding, pre-defined outcomes, an appropriate control, and independent replication should be ensured. Figure 2 illustrates an iterative process that puts the experiments at the centre of the process, with the experiments providing data to update the network and candidates with a network structure that meets the chemical, mechanistic, exposure, efficacy, safety, and reproducibility criteria moving forward toward translation. Special attention should be paid to experiments that focus on mixtures. Interactions can be tested in factorial designs, by response-surface methods, isobolographic analysis or combination-index approaches without screening of all possible ratios. The use of stable-isotope tracing may reveal changes in pathway flux as well as metabolite abundance, which may occur as expected. Patient-derived systems (such as cells, organoids, microphysiological systems, and primary tissue) can enhance translation while preserving animal and clinical evidence that is validated, but models should not be used in lieu of validated animal and/or clinical evidence. Having prespecified decision gates ensures that the attractive network narratives do not progress even if there is insufficient causal evidence.

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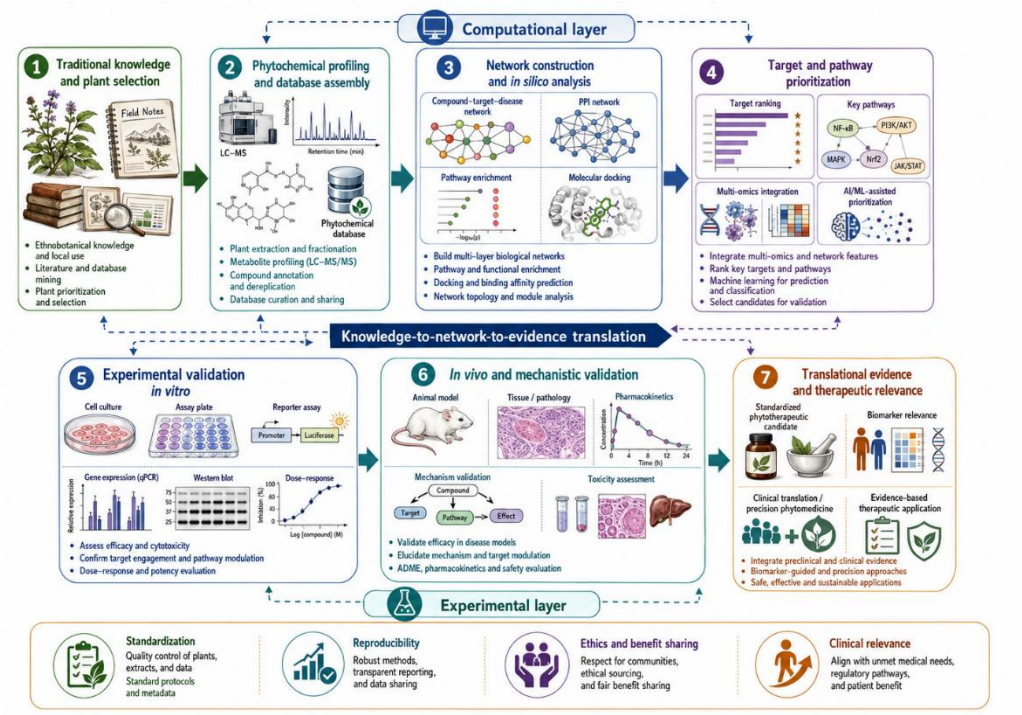

Figure 2: Integrated computational-experimental workflow for validating medicinal plant molecular networks.

8. CLINICAL AND TRANSLATIONAL SIGNIFICANCE

The product that is tested must be chemically and biologically identical to the one that is used in the clinic. Stability, batch-to-batch chemical fingerprints, contaminant limits and batch-to-batch controls, representative raw-material specifications and authenticated compound composition should be established for a botanical. Even if there are multiple constituents, pharmacokinetic studies are required as the exposure dictates whether targets can be achieved if proposed. Systems analysis can yield biomarkers that can be used to select patients, validate pathway modulation, or detect early toxicity, but these must be analytically validated and show meaningful association with patients' outcomes. Trial design must be in line with the product intended; the results of a trial with a standard extract cannot be used for another cultivar, solvent, dose or formulation. The following are clinically relevant comparators, sufficient sample size, preregistration requirements, reporting, and evaluation of herb-drug interactions. Network pharmacology could help to inform indication selection, biomarker development, and therapeutic claims can be substantiated only by appropriate human studies. The regulatory guidance for botanical drugs focuses on the control of identity, quality, consistency and clinical evidence as a way to ensure that a plant-based drug product may contain several active ingredients (29). The utility of systems methods is in their translation rather than in reducing evidentiary standards of systems interventions. In multi-component products, the selection of dose should be related to chemical fingerprint and biological responses. Three factors, namely safety, constituent pharmacokinetics, and metabolite profiling together with pathway biomarkers can be utilized during the early phase studies to decide if the proposed network is engaged in humans. If a patient population responsive to trial interventions is identified through a mechanistic or enrichment biomarker, then an adaptive or enrichment trial design might be appropriate; but exploratory biomarkers should not be interpreted as validated surrogate endpoints. Post-marketing surveillance should be emphasized since patients can use more than one botanical product at the same time with prescription medicines or botanical products may differ from the trial product.

9. CHALLENGES AND LIMITATIONS

Mismatch is the largest methodological issue, as the compounds used to create a network are not necessarily those present in the extract and predicted targets are not necessarily attainable at realistic exposures. The topology and enrichment distortions in databases arise from database incompleteness, publication bias, inconsistent identifiers, promiscuous-assay artefacts, and over-representation of well-studied proteins. Add to that batch effects, multiple testing problems, tissue variability, and an

unknown biological direction in omics studies. Additionally, networks can include nonlinear dose-response, toxicity and antagonism. The lack of plant provenance, extraction parameters, analytical thresholds, software versions, and code creates a weaker reproducibility. There are also conceptual restrictions since traditional use may not straightforwardly translate into contemporary diagnostic categories, and by forcing an ethnomedical use into a contemporary disease-based label, context and validation targets can be lost. Prospective protocols, FAIR data practices, openly reported workflows, chemical and biological quality controls, external validation and explicit uncertainty are all necessary for progress. Negative Results should be kept with models as they enhance the validity of models and decrease the need to repeat testing of unlikely mechanisms. A second difficulty is the size of the networks—the ones with hundreds of compounds and thousands of targets are visually impressive, but not easily falsified. Helpful models should simplify by narrowing down the number of evidence-based models, competing models and experiments that decide the model. Different analysis plans and computational pipelines would be comparable if benchmark data sets were used along with preregistered analysis plans. If this discipline is not followed then the same extract could result in many different possible mechanisms depending on the database used and the lists used to filter.

10. ETHICAL, REGULATORY, AND SUSTAINABILITY CONSIDERATIONS

There are obligations to traditional knowledge besides scientific citation. Communities should be involved via culturally appropriate consent, control, recognition and equitable benefit sharing mechanisms, particularly if knowledge is used to create commercial products. IP strategies should not transform collectively created knowledge into "claims to exclusion" without the right to participate. Where additional demand comes into play, the principles of sustainable harvesting, cultivation, traceable supply-chain and conservation planning are paramount. Species substitution and contamination, and lack of uniformity in manufacture should be regulated, as should be pharmacovigilance to record adverse effects and interactions during use. International and national frameworks vary, but there is a need to view translations as a system—ethical provenance, ecological impact, product quality and scientific evidence as linked components—rather than as administrative priorities (29).

11. FUTURE PERSPECTIVES

Future research will shift from static compound-target diagrams to dynamic models, dependent on exposure and specific to tissue. Spatial transcriptomics, single cell profiling, organoids, organ-on-chip systems and quantitative systems pharmacology could explain where and when botanical components act on the disease network. Digital ethnobotany can archive preparation information, the surrounding community and focus it, connect it with voucher specimens, chemical fingerprints, and results. Explainable artificial intelligence could use mixtures as a priority, predict interactions, and specify smaller experimental sets as long as models report the level of uncertainty and are interpretable to scientists and clinicians. The possibility of personalised phytotherapy is also possible if patient genomics and microbiome composition, metabolism and co-medications, and disease subtype, are combined but will require robust clinical evidence and appropriate equity protection. Advances would be made more rapidly through the use of shared reference materials, open spectral libraries, standardised reporting, and multicentre replication. The aim is not to displace traditional knowledge with computation but to develop a 'clear' evidence cycle, whereby cultural observation and analytical chemistry feed in, analytical chemistry and network inference feed in, network inference and causal experiments feed in, causal experiment and clinical outcomes feed in, and clinical outcomes and culture follow through. Prospective living networks could be revised with the acquisition of additional chemical, mechanistic, safety and clinical data. These models should incorporate provenance for each edge, include confidence and direction, and provide a way to follow up a conclusion to the experiment or community observation in question. This would enable auditing of systems pharmacology, and allow regulators and clinicians to know the difference between exploratory prediction and validated evidence.

12. CONCLUSION

The power of traditional knowledge and the power of molecular networks are most powerful when neither is taken as self valid. The ethnobotanic process can select relevant prospects and the preparation contexts; the systems approach can structure the chemical and biological complexity; and a sound experiment can resolve which of the interactions predicted by the ethnobotanic process is causal, reproducible, safe, and clinically relevant. The field is ready to shift from decorative networks to models that are based on exposure and uncertainty, and experimented. In addition to standardisation, ethical governance, benefit sharing, sustainability, and properly designed clinical trials, this can turn medicinal plant research from the fragmented observations to evidence-based therapeutic translation.

Declarations

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Conflict of Interest: The authors declare that they have no conflict of interest.

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