Research Article

The Anticancer Mechanisms of Scutellaria barbata against Lung Squamous Cell Carcinoma

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1. Introduction

Lung squamous cell carcinoma (LUSC) is one of the most frequent malignancies in the world. The incidence and mortality of lung cancer were the highest among all tumors, as shown by the global cancer statistics in 2018 [1]. Non-small-cell lung cancer (NSCLC) consists of 85% of all lung cancer cases and is therefore the most common pathological type of lung cancer [2]. LUSC, which is now the second most frequent type of NSCLCs, accounts for 20%-30% of NSCLCs, showing more than 400,000 new cases each year [3]. Although the treatment of LUSC has improved, the 5-year overall survival chance of patients with clinical stage III and IV LUSC patients is normally lower than 5% [4]. Hence,
identifying novel potential therapeutic drugs and targets is of great significance for treating LUSC in clinical practice. Based on the cumulative effect of multigene and multisite mutations in tumors, multitarget drugs, as an important direction of tumor-targeted therapy, provide new ideas for tumor treatment [5, 6]. Slight side effects, good therapeutic effects, and multiple targets are the major characteristics of traditional Chinese medicine (TCM). In the current clinical practice, TCM could prolong patients’ survival time and improve their life quality [7]. Recently, studies have shown that *Hedyotis diffusa* plus *S. barbata* is capable of inducing apoptosis and alleviating cell proliferation and colony formation during tumor growth *in vivo* and *in vitro* [8]. Network pharmacology is a systematic analysis of the “disease-gene-target-drug interaction network based on computer fusion and systems biology technology and has become a potent tool for in-depth understanding of drug mechanisms [9]. According to the “lock and key principle” of the interaction between ligands and receptors, molecular docking could simulate the interaction between receptor biological macromolecules and small ligand molecules [10]. Using molecular docking technology to virtually screen the active ingredients in Chinese medicines and compound prescriptions not only saves costs but also greatly accelerates the research cycle of Chinese medicines. To clarify the effective molecular structure, reasonable compatibility, and molecular mechanism of *S. barbata* for treatment of LUSC, we did the relevant research.

2. Material and Methods


2.2. Screening of Action Targets and Chemical Components of *S. barbata*. The action targets and chemical components of *S. barbata* were identified using the online database TCMSP. Under the oral bioavailability criteria (OB) ≥ 30% and drug-likeness (DL) ≥ 0.18 [13], related target proteins and the monomer chemical components were screened out and then converted to their corresponding UniProt IDs.

2.3. Screening of Target Genes in LUSC. The target genes related to lung squamous cell carcinoma (LUSC) were screened by searching the DisGeNET, GeneCards, OMIM, DrugBank and TTD databases, and the LUSC target dataset was established.

2.4. Gene Ontology (GO) Function Enrichment Analysis. Common target genes were identified from the intersected *S. barbata* and LUSC target genes. These predicted target genes were involved in the anticancer effect of *S. barbata* against LUSC. The GO function enrichment analysis was carried out via the “Functional Annotation” module of the online tool DAVID. The predictive target genes of *S. barbata* against LUSC were introduced into the online DAVID database, based on these parameters: species, *Homo sapiens*; identifier, official gene symbol; gene list, list type; and remaining parameters, default values. Then, the results of GO function enrichment analysis were obtained.

2.5. KEGG Pathway Enrichment Analysis. To identify the associated pathways of these predicted target genes of *S. barbata* against LUSC, the KOBASE databases were used to perform pathway enrichment analysis. The pathway enrichment was considered significant if *P* < 0.05.

2.6. Integrative Gene Network Construction and Analysis. Cytoscape 3.8.2 was employed to establish the relationship network of *S. barbata* genes encoding functional monomers and target genes in LUSC. The PPI network of target proteins of *S. barbata* in LUSC was constructed by the online tool STRING. The PPI was constructed by setting the protein type to “Human”, minimum interaction threshold to more than 0.9, and other parameters to default settings. It was then imported into Cytoscape, and the interaction PPI network was scored using the CytoNCA plug-in to filter hub genes in accordance with degree centrality (DC) value.

2.7. The Expression of Hub Genes in LUSC Analysis. The hub gene expression in LUSC was analyzed by the GEPIA database.

2.8. Verification of Molecular Docking between the Functional Monomer Component and Target Proteins of Hub Genes. The key target protein’s 3D structure and the mol2 file of the functional monomer component were downloaded from the PDB database and the TCMSP platform, respectively. Then, we use PyMOL software to carry out water removal, hydrogenation, and other operations on the key target protein, conduct molecular docking through AutoDockTools, and record the binding energy.

3. Results

3.1. Screening of Functional Monomer Components and Action Targets of *S. barbata*. A total of 29 functional monomer components of *S. barbata* and 592 corresponding active target proteins were gotten from the TCMSP database, under the criteria of OB ≥ 30% and DL ≥ 0.18. After the removal of duplicate target proteins, 222 target proteins were
identified and then converted to official gene names according to the UniProt database.

3.2. Target Screening for LUSC. We searched the DisGeNET database and selected 119 target genes with a gene-disease association score $\geq 0.01$; the GeneCards database was searched, and totally 669 target genes of LUSC were screened by the correlation score $\geq 40$; the OMIM database was searched, and totally 500 target genes of LUSC were screened; totally 35 target genes of LUSC were screened from the DrugBank; TTD database was searched and selected 1 target gene of LUSC. Taking the union of the five and removing duplicates, 1165 target genes of LUSC were obtained.

3.3. GO Function Enrichment Analysis. The intersected gene sets include the target genes of functional monomer components in S. barbata and target genes in LUSC; that is, the predicted target genes of S. barbata acting on LUSC, which revealed 104 common target genes (Figure 1). The 104 genes corresponded to 14 active S. barbata monomer components, as shown in Table 1. GO analysis comprises three domains of biology: cellular component, biological process, and molecular function. A total of 740 functions, including 55 cellular components, 102 molecular functions, and 583 biological processes, have been predicted to be enriched in the intersected genes. The threshold for screening and analyzing the enriched GO functions was set at $P < 0.05$. A smaller $P$ value indicated a stronger correlation between GO functions and the anticancer effect of S. barbata functional monomers against LUSC. Therefore, the top 10 significantly enriched functions were screened and visualized. From GO enrichment analysis, biological processes of target genes of S. barbata against LUSC mainly involved the signal transduction and positive regulation of transcription from the RNA polymerase II promoter, negative regulation of apoptosis, and etc. (Figure 2). The cellular component of target genes of S. barbata against LUSC was largely enriched to the cytosol, plasma membrane, and cytoplasm (Figure 3). The significantly enriched molecular functions were enzyme binding, protein binding, ATP binding, and DNA binding (Figure 4).

3.4. KEGG Pathway Enrichment Analysis. The signaling pathway enrichment analysis was performed on 104 target genes of S. barbata against LUSC, and a total of 229 pathways were significantly enriched via the KOBAS database. We selected the top 30 signaling pathways enriched in the target genes and visualized them according to the sum of enriched genes in each pathway, as shown in Figure 5. The threshold for screening and analyzing the enriched pathways was set at $P < 0.05$. A smaller $P$ value indicated a stronger correlation between the KEGG pathways and the anticancer effect of S. barbata functional monomers against LUSC. The intersected genes were mainly involved in several KEGG pathways, including the AGE-RAGE signaling pathway, PI3K-Akt signaling pathway, p53 signaling pathway, MAPK signaling pathway, and IL-17 signaling pathway.

3.5. Construction and Analysis of the Integrated Gene Network. The relationship network of S. barbata functional monomers-target-disease (LUSC)-pathway was analyzed in Cytoscape (Figure 6). In the network, red represented disease, yellow represented active drug monomer components, blue represented target genes, green represented signal pathways, and connectivity represented interrelationships. The network diagram showed the relationship between the functional monomers of S. barbata and their corresponding targets, as well as the complex network between the multiple targets and pathways of LUSC and S. barbata functional monomers, suggesting that the anticancer effect of the functional monomers of S. barbata involved multiple components, targets, and channels.

3.6. Construction of the PPI Network and Screening of Hub Genes. The online tool STRING was utilized to plot a PPI graph. A total of 20 hub genes (JUN, AKT1, MAPK1, TNF, RELA, etc.) were obtained by scoring the PPI network (according to degree centrality $\geq 15$) with the CytoNCA plug-in of Cytoscape (Figure 7), suggesting the critical functions of these genes in the network, and they were considered potential genes in LUSC targeted by functional monomers of S. barbata.

3.7. Expression of Hub Genes in LUSC Analysis. Simultaneously, the expression of these hub genes in LUSC and normal samples from the GEPIA database was analyzed, and the expression of 6 hub target genes, namely, CDK1, FOS, IL6, JUN, MYC, and TP53, was significantly differentially expressed in normal and LUSC samples ($P < 0.05$, Figure 8). These results indicate that the six hub genes are strongly linked to the development of LUSC.

3.8. Molecular Docking between the Functional Monomer Component and Target Proteins of Hub Genes. Molecular docking was performed between the six target proteins of differentially expressed hub genes and the corresponding functional monomer component of these target proteins. AutoDock software was used to predict the binding ability. A lower binding energy was correlated with a more stable conformation. Generally, the binding energy $< 0$ kcal/mol indicates that the molecules have binding activity, while binding energy $< 5.0$ kcal/mol indicates that the molecules have strong binding activity [26]. The results of molecular docking showed that they had good binding ability, as shown in Table 2. Then, PyMOL software was employed to visually analyze the best conformation of the docking between the target protein and the component, and take baiacalcin as an example, as shown in Figures 9 and 10.

4. Discussion

Traditional Chinese medicine is the precious wealth of the Chinese medicine development concept for thousands of years. However, the complex composition of traditional Chinese medicine makes it a challenging task of studying the efficacy and mechanism of plant extracts. In view of the complex, diverse, and trace components of traditional Chinese medicine compounds/preparations, the chromatographic-mass spectrometry analysis technology and data processing strategies that have been gradually established in recent years
are greatly promoting the rapid, high-throughput qualitative and accurate quantification of complex components of traditional Chinese medicine. Previously, the study [27] has identified the 124 volatile components and 33 nonvolatile components in VTF using comprehensive two-dimensional gas chromatography hyphenated with mass spectrometry.

**Table 1: Basic information of the functional monomer component of S. barbata.**

<table>
<thead>
<tr>
<th>S. barbata functional monomer component name</th>
<th>TCMSP number</th>
<th>OB (%)</th>
<th>DL</th>
<th>Mechanism of anticancer (list some references)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta-sitosterol</td>
<td>MOL000358</td>
<td>36.91</td>
<td>0.75</td>
<td>Cytotoxic activities via inducing apoptosis and activating caspase-3 and caspase-9 [14]</td>
</tr>
<tr>
<td>Wogonin</td>
<td>MOL000173</td>
<td>30.68</td>
<td>0.23</td>
<td>Antitumor angiogenesis and negative regulation of tumor cell energy metabolism [15]</td>
</tr>
<tr>
<td>Baicalein</td>
<td>MOL002714</td>
<td>33.52</td>
<td>0.21</td>
<td>Akt/mTOR signaling pathway was downregulated, and the proliferation and migration of tumor cells were inhibited by inhibiting the expression of FAK [16]; upregulation of phosphorylation of Ste20 like kinase and activation of the Hippo signaling pathway [17]</td>
</tr>
<tr>
<td>Rhamnazin</td>
<td>MOL000351</td>
<td>47.14</td>
<td>0.34</td>
<td>Antiapoptotic effect via reduction of Bax and increase in the expression of Bcl-2; inhibit oxidative stress and inflammation [18]</td>
</tr>
<tr>
<td>Luteolin</td>
<td>MOL000006</td>
<td>36.1</td>
<td>0.25</td>
<td>Weaken the interaction on the surface of tumor cells and promote macrophage-mediated phagocytosis [19]; inhibit the phosphorylation of STAT3 and induce tumor cell apoptosis [20]</td>
</tr>
<tr>
<td>Quercetin</td>
<td>MOL000098</td>
<td>46.43</td>
<td>0.28</td>
<td>Upregulate the expression of p53, Bax, Fas, and other apoptosis-related genes and induce caspase-independent apoptosis of tumor cells [21]</td>
</tr>
<tr>
<td>Eriodictyol</td>
<td>MOL005190</td>
<td>71.79</td>
<td>0.24</td>
<td>Anticancer effects by inducing apoptosis and cell cycle arrest at the G2/M phase through restraining the mTOR/Pi3K/Akt cascade [22]</td>
</tr>
<tr>
<td>Rivularin</td>
<td>MOL012266</td>
<td>37.94</td>
<td>0.37</td>
<td>Unknown</td>
</tr>
<tr>
<td>Stigmasterol</td>
<td>MOL000449</td>
<td>43.83</td>
<td>0.76</td>
<td>Induces apoptosis and protective autophagy by inhibiting the Akt/mTOR pathway [23]</td>
</tr>
<tr>
<td>Moslossoflavone</td>
<td>MOL008206</td>
<td>44.09</td>
<td>0.25</td>
<td>Unknown</td>
</tr>
<tr>
<td>Chrysin-5-methylether</td>
<td>MOL012251</td>
<td>37.27</td>
<td>0.2</td>
<td>Anticancer activity of chrysin associated with the decreased protein expression of p-ERK/ERK and p-AKT/AKT [24]</td>
</tr>
<tr>
<td>*5-Hydroxy-7,8-dimethoxy-2-(4-methoxyphenyl)-chromone</td>
<td>MOL012248</td>
<td>65.82</td>
<td>0.33</td>
<td>*Unknown three flavonoids with the same 2-phenyl-chromone skeleton; AH5 also with the same structure, which can induce G2/M arrest and significantly apoptosis HepG-2 cell lines. AH5 induced the phosphorylation of Cdc2 and decreased the level of cyclin B1 [25]</td>
</tr>
<tr>
<td>*(2R)-5,7-Dihydroxy-2-(4-hydroxyphenyl)-4-chromanone</td>
<td>MOL001040</td>
<td>42.36</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>*(2R)-5,7-Dihydroxy-2-(4-hydroxyphenyl)-4-chromanone</td>
<td>MOL012250</td>
<td>43.73</td>
<td>0.25</td>
<td></td>
</tr>
</tbody>
</table>

**Figure 1: Venn diagram of target genes of S. barbata and LUSC.**
(GC×GC-MS) and ultraperformance liquid chromatography coupled with Orbitrap mass spectrometry (UPLC-Orbitrap-MS), respectively. In our study, 14 functional monomers of *S. barbata* that target LUSC-related genes were screened out, which mainly included beta-sitosterol, wogonin, baicalein, rhamnazin, luteolin, and quercetin; all of the above-

![Figure 2: Biological process enrichment analysis for anti-LUSC target genes of *S. barbata*.](image)

![Figure 3: Cellular component enrichment analysis for anti-LUSC target genes of *S. barbata*.](image)
The mentioned functional monomers are summarized in Table 1. However, the effects (in vivo and in vitro) of these monomers on the proliferation, invasion, and metastasis of LUSC should be further explored.

In this study, the anticancer effect of functional monomers of S. barbata against LUSC and the underlying mechanism were investigated using a network pharmacology approach. There are 20 hub genes of S. barbata involved in the expression of functional compounds with anti-LUSC activity. However, a large study using the GEPIA database (from TCGA and GTEX databases) found that only 6 of the genes were significantly differentially expressed in LUSC. Molecular docking technology was used to do molecular docking between the six target proteins of differentially expressed hub genes and the corresponding functional monomer component. The results showed that the binding energy of all key active components combined with all key target proteins was less than 0 kcal/mol, suggesting that their...
binding activity is better and confirming the targeting effect of *S. barbata* for the treatment of LUSC. Due to differences in regions, races, and statistical methods, whether the expression of other hub genes among Chinese people may vary still needs further verification. The *CDK1* gene encodes cyclin-dependent kinase 1, which has a critical function in eukaryotic cell cycle control through the modulation of centrosome cycle and mitotic onset. Moreover, it also stimulates G2-M transition and regulates G1 progress and G1-S transition via interacting with multiple interphase cyclins [28]. In lung cancer cell lines, STAT3 and CDK1 play an important role in iron-mediated colony formation. Kuang et al. [29] showed that iron-dependent CDK1 activity could increase the IL-6 receptor subunit GP130 during posttranscription through 4E-BP1 phosphorylation, which has a critical function in JAK/STAT3 signaling activation. The upregulation of c-Fos and c-Jun, two strong transcription factors, leads to overactivation of other oncogenes, which seems to be related to the aggressive biological behavior of non-small-cell lung cancer [30]. Interaction between programmed cell death 1

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**Figure 6:** The functional monomer components of *S. barbata*-target-disease-signaling pathway network. Note: red represents disease, yellow represents active monomer components, blue represents target genes, and green represents signaling pathways.

**Figure 7:** The protein interaction network of anti-LUSC target genes of *S. barbata* (a) and interaction network of hub genes (b).
and programmed cell death ligand 2 (PD-L2) participates in tumor immune escape. The previous research indicated that, in NSCLC cells, oncogene-driven expression of PD-L2 was inhibited via knocking down the activator of transcription 3 (STAT3) and transcription factors signal transducer or c-Fos, providing important knowledge on tumor immune escape in the NSCLC [31]. IL-6 (interleukin-6) is also critical for the development of NSCLC. Depending on the NF-κB signal pathway, TIM-4 expression is upregulated in NSCLC cells. IL-6 and TIM-4 promoted epithelial-to-mesenchymal transition, invasion, and migration of NSCLC cells [32].

Studies have demonstrated that c-Myc, which is a transcription factor binding DNA in a nonspecific manner, is also a frequently amplified human oncogene that acts as a critical aerobic glycolysis regulator of cancer cells through activating

**Table 2**: Molecular docking: functional monomer components of *S. barbata* corresponding to the LUSC hub gene.

<table>
<thead>
<tr>
<th>Gene</th>
<th>Functional monomer component</th>
<th>Binding energy (kcal/mol)</th>
<th>Intermolecular energy</th>
</tr>
</thead>
<tbody>
<tr>
<td>CDK1</td>
<td>Baicalein</td>
<td>-4.04</td>
<td>-5.24</td>
</tr>
<tr>
<td>FOS</td>
<td>Baicalein</td>
<td>-4.16</td>
<td>-5.35</td>
</tr>
<tr>
<td>IL-6</td>
<td>Wogonin</td>
<td>-6.67</td>
<td>-7.86</td>
</tr>
<tr>
<td>JUN</td>
<td>Beta-sitosterol</td>
<td>-4.77</td>
<td>-6.86</td>
</tr>
<tr>
<td>MYC</td>
<td>Quercetin</td>
<td>-4.05</td>
<td>-5.84</td>
</tr>
<tr>
<td>TP53</td>
<td>Wogonin</td>
<td>-5.41</td>
<td>-6.60</td>
</tr>
</tbody>
</table>

![Figure 8](image_url) - Expression of 6 hub target genes in normal and LUSC samples.
certain glycolytic genes directly [33]. P53 shows tumor-suppressive effect on many tumor types. According to the physiological environment and cell type, P53 could induce apoptosis or growth arrest. As a trans-activator, P53 is implicated in cell cycle regulation and negatively regulates cell division through controlling a series of genes necessary for the process [34]. In particular in tumor suppressor genes such as TP53 and LKB1 (also known as STK11), some cooccurring genomic changes have been considered the critical determinants of the clinical and molecular heterogeneity of the subgroup of oncogene-driven lung cancer [35]. Huang et al. found that p53 may contribute to the cytotoxic and cytostatic effects associated with the establishment of feedback loop of erastin-induced ROS. Erastin-induced p53 can lead to ferroptotic and apoptotic cell death and suppress cell proliferation through blocking the cell cycle in the G1 phase [36].

From KEGG pathway enrichment analysis, we found that the anti-LUSC target genes of S. Barbata are enriched in signaling pathways including the PI3K-Akt signaling pathway, AGE-RAGE signaling pathway, IL-17 signaling pathway, C-type lectin receptor signaling pathway, p53 signaling pathway, TNF signaling pathway, and MAPK signaling pathway. The PI3K/Akt/mTOR pathway and signal cascade play a crucial role in regulating cell growth and metabolism. In NSCLC, the PI3K/Akt/mTOR pathway is closely related to tumorigenesis and disease progression. Somatic mutations and amplifications of PIK3CA are often
found in patients with NSCLC. A large study of 1,144 consecutive patients with NSCLC used next-generation sequencing (NGS) to study the PIK3CA mutation of tumor tissue, which shows that the mutation was identified in 3.7% of patients, with squamous cell carcinoma (8.9%) and adenocarcinoma (2.9%) predominant [37]. In the Chinese NSCLC population, PIK3CA amplification and highly frequent promoter methylation (CALCA, CDH1, DAPK1, and EVX2) were identified, predominantly in patients of squamous cell carcinoma histology, indicating that those epigenetic events resulted from PI3K/Akt pathway overactivation [38]. Besides, unregulated expression of certain lncRNA or miR can also activate or suppress the PI3K/AKT signaling pathway and promote the invasion and migration of lung cancer cells [39, 40]. The P53 signaling pathway is also involved in lung cancer. Mao et al. [41] confirm that the cytosolic P53RRA-G3BP1 interaction replaces p53 in the G3BP1 complex, causing more p53 to stay in the nucleus, leading to cell cycle arrest, apoptosis, and ferroptosis. Therefore, it is concluded that cytoplasmic lncRNA exerts tumor suppressor effect by activating the p53 pathway. As a novel oncogene in lung cancer, TC2N can attenuate the p53 signaling pathway via suppressing Cdk5-induced phosphorylation of p53 through disrupting the interaction between Cdk5 and p53 or the induction of Cdk5 degradation [42]. In human lung squamous carcinoma cells, under Bcl-2 downregulation, Bax upregulation, and caspase-3 activation, isoalantolactone can induce intrinsic apoptosis via the p53 signaling pathway in vitro [43]. Similarly, with the significant increase in caspase-3 and significant decrease in cyclin D1 and E2 proteins, pterostilbene has chemopreventive effects on the mouse model of lung squamous cell carcinoma through the p53 signaling pathway [44]. The development of drug resistance is still the main obstacle to the clinical efficacy of EGFR tyrosine kinase inhibitors (TKI) for NSCLC. Relevant research certificates that upregulated expression of FGFR1 (fibroblast growth factor receptor 1) by hypoxia was mediated through the MAPK pathway, which is a driving force for acquired resistance to EGFR TKIs [45]. Cancer-associated fibroblasts (CAFs) have tumor-stimulating properties, and overexpression of fibroblast-derived exosomal miR-369 activates MAPK signaling pathways through interaction with neurofibromin-1, consequently promoting the growth of LUSC cells [46]. Epithelial-mesenchymal transition (EMT) is related to tumor invasion and metastasis. Wang et al.’s [47] research shows that sotetsuflavone not only upregulated angiostatin and downregulated VEGF but also downregulated MMP-13 and MMP-9 expression at the same time. Those findings indicated that EMT could be reversed by sotetsuflavone, which was potentially related to both TNF-α/NF-κB and PI3K/AKT pathways. IL-17A and its receptor IL-17RA can promote the migration and invasion of NSCLC cells in vitro, and the phosphorylation of p38 in NSCLC cells with high IL-17RA expression is enhanced. In addition, the p38mapk-specific inhibitor SB203580 can suppress the invasion and migration of NSCLC cells, so IL-17A/IL-17RA signal transduction may be a novel promising tumor treatment target for the treatment of NSCLC [48]. In conclusion, the MAPK signaling pathway, PI3K-Akt signaling pathway, and p53 signaling pathway played an imperative role in the pathological process of lung cancer, suggesting that S. barbata may exert anti-LUSC effects by interfering with the p53 signaling pathway, MAPK signaling pathway, PI3K-Akt signaling pathway, etc.

In summary, network pharmacology demonstrated that the major active components of S. barbata, particularly baikalein, wogonin, beta-sitosterol, and quercetin, could act significantly on targets. S. barbata had effect to treat LUSC largely via the MAPK signaling pathway, p53 signaling pathway, and PI3K-Akt signaling pathway. Molecular docking showed that their binding activity is better, which indicated that they might play an important role in the treatment of LUSC. The findings of this study provide a beneficial inference for the further study of the mechanism of action of these functional compounds of S. barbata and lay a scientific foundation for the synthesis of functional monomers of S. barbata with more potent anticaner properties.

Data Availability

The data used to support the findings of this study are included within the article.

Conflicts of Interest

All authors declare no competing interests.

Authors’ Contributions

We are accountable for all aspects of the work (including full data access, integrity of the data, and the accuracy of the data analysis).

Acknowledgments

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References


