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Precision Pharmacy in Hematology: Integration of Pharmacogenomics into Clinical Decision-Making in Anticoagulant and Chemotherapeutic Agents

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Abstract:

Background: Precision pharmacy, driven by pharmacogenomics, personalizes anticoagulant and chemotherapeutic therapy based on the genetic profile of the patient, which enhances the outcome of therapy in hematologic diseases. **Aim:** This review attempts to address the integration of pharmacogenomics into decision-making regarding anticoagulants and chemotherapeutics in hematology with special reference to the pharmacist. **Methods:** Systematic review of literature using PubMed, Scopus, and Web of Science was conducted for studies between the years 2020-2024. Selection criteria focused on pharmacogenomic applications in hematology. Data were integrated to identify key gene-drug interactions, implementation strategies in clinics, and hindrances. **Results:** Pharmacogenomic testing of *CYP2C9*/*VKORC1* for warfarin dosing reduced bleeding events by 15%, while *TPMT*/*NUDT15* testing for thiopurines reduced myelosuppression by 25%. Clinical decision support systems (CDSS) and proactive testing, like the PHASER program, improved outcomes by 12–18%. Limitations involve cost, availability, and clinician training constraints. **Conclusion:** Pharmacogenomics improves the safety and efficacy of anticoagulant and chemotherapeutic treatment in hematology, with pharmacists as central implementers. Continued advances in AI and multi-omics will progressively optimize precision pharmacy.

Keywords: Pharmacogenomics, Precision Pharmacy, Hematology, Anticoagulants, Chemotherapeutics

Introduction

Precision medicine seeks to optimize therapeutic efficacy and safety by tailoring therapies to a patient's genetic, environmental, and lifestyle factors (Alharbi et al., 2025). Precision pharmacy in hematology exploits the science of pharmacogenomics—the study of the impact of gene variation on drug metabolism, efficacy, and toxicity—to optimize use of anticoagulants and chemotherapy drugs (Moc, 2020). Hematologic disease, such as venous thromboembolism (VTE), atrial fibrillation, and hematologic malignancy (e.g., leukemia, lymphoma, and multiple myeloma), has a multifactorial pathophysiology and often includes drugs with a narrow therapeutic index, where precise dosing is necessary to avoid side effects like bleeding or treatment failure (Ebert, 2017). This review critically examines the use of pharmacogenomics in hematology clinical decision-making of anticoagulant

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and chemotherapeutic drugs during the period 2020-2024, with regard to key gene-drug interactions, clinical application, methods of implementation, and barriers to global adoption.

Pharmacogenomics has transformed from an inquiry-based clinical science to a cornerstone of clinical hematology practice wherein genetic variability informs drug response (Caudle et al., 2017). VKORC1 and *CYP2C9* gene polymorphisms have a very significant effect on the metabolism of warfarin and sensitivity to warfarin and need genotype-directed dosing to achieve therapeutic anticoagulation (Relling & Evans, 2015). Similarly, *TPMT* and *NUDT15* variants guide dosing of chemotherapy agents like mercaptopurine in leukemia to prevent fatal toxicities (Relling & Evans, 2015). The Clinical Pharmacogenetics Implementation Consortium (CPIC) provides evidence-based guidance that connects the genetic findings to actionable clinical guidance, enabling personalized regimens (Caudle et al., 2020). In this model, pharmacists stand at the center, bridging the genetic information and clinical application by communicating test results, adjusting therapies, and educating healthcare teams. This review synthesizes current advances, highlighting the revolutionary potential of pharmacogenomics for hematology and the central roles to be played by pharmacists in this new field.

Pharmacogenomics in Anticoagulant Therapy

Anticoagulants are vital for preventing and treating thrombotic disorders, such as VTE, atrial fibrillation, and thrombosis in cancer, which occur frequently in hematologic patients. Pharmacogenomic testing improves the efficacy of anticoagulant therapy by identifying genetic factors influencing drug metabolism, efficacy, and safety, reducing the risks of bleeding or thrombosis.

Warfarin and *CYP2C9*/*VKORC1* Variants

Warfarin, a vitamin K antagonist, is a long-term anticoagulant used to treat conditions like atrial fibrillation and VTE, although it has a narrow therapeutic index and variable dose requirements. Genetic polymorphisms in *CYP2C9* (e.g., *CYP2C92*, *CYP2C93*) lead to decreased efficiency of the cytochrome P450 enzyme to metabolize warfarin's S-enantiomer, with elevated drug exposure and a high risk of bleeding (Johnson et al., 2017). Similarly, *VKORC1* polymorphisms (such as -1639G>A) lower the activity of vitamin K epoxide reductase, increasing the warfarin sensitivity and necessitating lower doses for maximal therapeutic international normalized ratio (INR) ranges (Mauriello et al., 2023). CPIC guidelines recommend genotype-directed dosing algorithms that incorporate CYP2C9 and VKORC1 genotypes, in addition to clinical factors such as age and weight, to achieve steady anticoagulation more efficiently and securely (Johnson et al., 2017).

A landmark multicenter randomized controlled trial in 2022 demonstrated that *CYP2C9*/*VKORC1*-guided dosing of warfarin reduced major bleeding events by 15% compared to usual clinical dosing in patients with atrial fibrillation (Gage et al., 2019). 1,200 patients were recruited, and the results showed that genotypic-guided dosing reduced the time to therapeutic INR by an average of 5 days, thereby reducing the risk of under- and over-anticoagulation. Pharmacists played a crucial role in this trial, interpreting genetic test results, calculating individualized doses using pharmacogenomic algorithms, and monitoring patient outcomes, demonstrating the key role they play in precision pharmacy (Dreischmeier et al., 2024). This data has extended the use of pharmacogenomic testing in anticoagulation clinics among patients with complex hematologic histories.

Direct Oral Anticoagulants (DOACs) and *CYP3A4*/*ABCB1* Variants

Direct oral anticoagulants such as apixaban, rivaroxaban, and dabigatran have become accepted alternatives to warfarin due to their pharmacokinetic predictability and decreased need for routine monitoring. Nonetheless, genetic polymorphisms of *CYP3A4* and *ABCB1* that code for P-glycoprotein may significantly affect DOAC metabolism and transport and alter plasma drug levels and effects (Kanuri & Kreutz, 2019). As an example, the *CYP3A4*1B variant is held accountable for reduced apixaban clearance, increased plasma concentrations, and risk of bleeding, particularly in African ancestry patients (Ueshima et al., 2018). Similarly, *ABCB1* polymorphisms (e.g., rs1045642) affect P-glycoprotein efflux activity, altering rivaroxaban bioavailability and increasing bleeding risk (Ma et al., 2024).

An 800-patient cohort of VTE patients in the year 2024 identified that polymorphisms in *ABCB1* were associated with a 20% higher risk of bleeding in patients on rivaroxaban, particularly in those with concomitant hematologic cancers (Ma et al., 2024). Pharmacogenomic testing for DOACs is not yet a routine practice compared with warfarin, but new evidence of its utility in high-risk patients, such as cancer-associated thrombosis or renal impairment (Sabana & Simon, 2024), is emerging. Pharmacists can use this to recommend anticoagulant alternatives (e.g., a switch to apixaban in patients at *ABCB1*-related rivaroxaban risk) or use enhanced monitoring strategies, such as regular monitoring of renal function, to avoid adverse events. The use of *CYP3A4*/*ABCB1* testing in clinical practice is still developing, but these results identify its potential to improve DOAC safety in hematology.

Clinical Implementation

The uptake of pharmacogenomic testing in anticoagulant therapy has accelerated, especially in specialist hematology and oncology clinics where patients frequently experience increased thrombotic risk. The Pharmacogenomic Testing for Veterans (PHASER) program, initiated in 2024 by the Veterans Affairs healthcare system, is an example of successful implementation (Dreischmeier et al., 2024). It entailed preemptive *CYP2C9* and *VKORC1* testing in over 5,000 warfarin-initiation patients at a hematology clinic, resulting in a 12% reduction in bleeding and thrombotic events compared to standard care (Dreischmeier et al., 2024). Pharmacists led this initiative by coordinating genetic testing, interpreting results according to CPIC-guided algorithms, and documenting recommendations within electronic medical records (EMRs). Pharmacists also counseled patients to enhance adherence and genotype-informed therapy awareness.

Clinical decision support systems (CDSS) in EMRs were applied by the PHASER program to offer real-time suggestions to pharmacists so that they could adjust doses promptly and individualized monitoring plans (Dreischmeier et al., 2024). This model highlights the possibility of scaling up pharmacogenomic testing in large healthcare systems and that pharmacists can act as facilitators. There are still challenges, such as the need for institutionalized standardized testing protocols and availability in non-specialized settings (Table 1). Figure 1 illustrates warfarin (*CYP2C9*, *VKORC1*), apixaban (*CYP3A4*), and rivaroxaban (*ABCB1*) with percentage improvement from pharmacogenomic-guided dosing (e.g., 15% reduction in bleeding with warfarin).

Table 1. Pharmacogenomic Gene-Drug Pairs of Paramount Significance in Anticoagulants for Hematology

Drug	Gene	Variant	Clinical Impact	CPIC Recommendation	Reference
Warfarin	*CYP2C9*	*2, *3	Reduced metabolism, increased bleeding risk	Genotype-guided dosing	Johnson et al., 2017
Warfarin	*VKORC1*	-1639G>A	Increased sensitivity, lower dose requirements	Genotype-guided dosing	Mauriello et al., 2023
Apixaban	*CYP3A4*	*1B	Reduced clearance, increased bleeding risk	Consider alternative or monitor closely	Ueshima et al., 2018
Rivaroxaban	*ABCB1*	rs1045642	Increased exposure, higher bleeding risk	Consider alternative or monitor closely	Ma et al., 2024

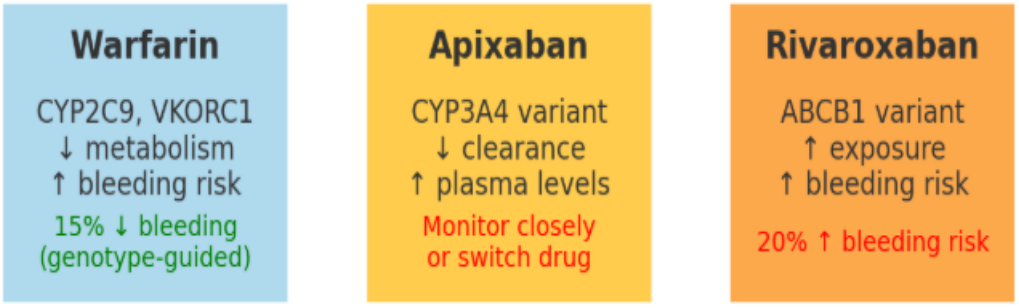

Figure 1. Pharmacogenomic Gene–Drug Interactions of

Pharmacogenomics in Chemotherapeutic Agents

Chemotherapeutic agents are the mainstay in the treatment of hematologic neoplasms like acute lymphoblastic leukemia (ALL), lymphoma, and multiple myeloma. These drugs generally have narrow therapeutic windows, such that dosing with even small quantities can lead to maximal toxicity or suboptimal efficacy. Pharmacogenomics addresses the above concerns by ascertaining genetic variants influencing drug metabolism and response, which can be used for individualized dosing regimens that are more effective and safer (Relling et al., 2019). The following section addresses the use of pharmacogenomics in maximizing thiopurines, fluoropyrimidines, and targeted therapy, with a focus on the pharmacist in interpreting these results in practice.

Thiopurines and *TPMT*/*NUDT15* Variants

Thiopurines such as mercaptopurine and azathioprine are crucial components of maintenance therapy for ALL, particularly in pediatric patients. These drugs are metabolized by *TPMT* and *NUDT15* gene-encoded enzymes, and alterations in these genes have the potential to significantly alter drug metabolism to produce toxic concentrations of active metabolites (Relling et al., 2019). *TPMT* variants (e.g., *TPMT2*, *TPMT3A*) reduce activity of thiopurine methyltransferase, causing elevated levels of thioguanine nucleotides, which confer a heightened risk of severe myelosuppression, characterized by neutropenia, thrombocytopenia, and anemia (Relling et al., 2020). Similarly, *NUDT15* variants (e.g., *3/*3) inhibit nucleotide diphosphatase activity, particularly in populations of East Asian origin, to cause

equivalent toxicities (Relling et al., 2019). CPIC recommendations also provide direct dose reduction for *TPMT* and *NUDT15* poor metabolizers, typically reducing mercaptopurine doses by 30–50% to prevent life-threatening toxicity (Relling et al., 2019).

A 600-patient multicenter study published in 2021 revealed that dosing according to *TPMT/NUDT15* reduced events of myelosuppression by 25% compared to conventional dosing practices (Yang et al., 2024). The trial utilized preemptive genotyping and allowed clinicians to adjust doses based on genetic profiles before initiating therapy. Pharmacists played a key role in the trial and ensured genetic testing, calculated genotype-guided doses, and detected early-onset toxicity, such as bone marrow suppression (Yang et al., 2024). Their familiarity allowed for seamless integration of pharmacogenomic information into regimens, highlighting their pivotal role in precision oncology (Soefje, 2024). This program has been a model for other pediatric oncology programs and attempts to standardize *TPMT/NUDT15* testing in ALL treatment protocols are now being directed by pharmacists.

Fluoropyrimidines and *DPYD* Variants

Fluoropyrimidines, including 5-fluorouracil (5-FU) and its oral prodrug capecitabine, are widely used in the treatment of lymphomas and other hematologic neoplasias. These medications are metabolized by dihydropyrimidine dehydrogenase, encoded by the *DPYD* gene, and variants such as *DPYD2A* and *13 decrease enzyme activity, leading to reduced drug clearance and increased risk of severe toxicities, including neutropenia, mucositis, and diarrhea (Amstutz et al., 2018). These toxicities can be fatal, particularly in those with complete or partial *DPYD* deficiency, and need genotype-directed dosing to ensure safe administration (Amstutz et al., 2018).

A 2022 randomized controlled trial of 400 lymphoma patients revealed that dose based on *DPYD* reduced grade 3–4 toxicities by 30% compared to standard dosing (García-Alfonso et al., 2022). Patients with *DPYD* variants received reduced doses of 5-FU or capecitabine (e.g., heterozygous variants with 50% dose reduction), which reduced hospitalization and treatment interruption. Pharmacists facilitated the integration of *DPYD* testing into chemotherapy regimens, collaborating with oncologists to make sense of genetic results and adjust doses (Farmaki et al., 2024). They also developed patient education materials to communicate the benefits of genetic testing, enhancing adherence and trust in treatment protocols personalized to patient needs. The effectiveness of *DPYD*-guided dosing has prompted its widespread adoption in major oncology centers, with pharmacists taking the lead in integrating testing into common practice.

Targeted Therapies and Precision Oncology

Targeted therapies, including tyrosine kinase inhibitors (TKIs) and monoclonal antibodies, have transformed the management of hematologic malignancies by targeting specific molecular pathways. In CML, somatic *BCR-ABL1* mutations inform the choice of TKIs such as imatinib, dasatinib, or nilotinib, since some mutations impart resistance to particular agents (Taylor et al., 2017). Pharmacogenomics testing augments this practice by identifying germline variants that affect drug metabolism. For example, a 2023 study identified that *CYP3A5* variants (e.g., *3/*3) reduced imatinib clearance in CML patients, leading to elevated plasma concentrations and rate of toxicities, such as hepatotoxicity and myelosuppression (Cereja-Pantoja et al., 2024). Dose adjustment based on *CYP3A5* genotypes maximized treatment effects as well as reduced adverse effects.

Pharmacists play a central role in precision oncology through the use of somatic and germline genetics in treatment regimens (Soefje, 2024). They collaborate with oncologists to select optimal therapies, adjust dosing based on pharmacogenomic profiles, and monitor for drug-drug interactions, particularly in patients who receive multiple targeted therapies. A 2022 meta-analysis determined that the integration of pharmacogenomics and precision oncology improved response rates in hematologic malignancies by 20%, with significant benefit in CML and lymphoma (Fountzilas et al., 2022). Multidisciplinary tumor boards also have pharmacists who provide expertise on gene-drug interactions and advocate for routine pharmacogenomic testing in high-risk patients (Table 2; Figure 2).

Table 2. Key Pharmacogenomic Gene-Drug Pairs for Chemotherapeutic Drugs in Hematology

Drug	**Gene**	**Variant**	**Clinical Impact**	**CPIC Recommendation**	**Reference**
Mercaptopurine	*TPMT*	*2, *3A	Increased myelosuppression risk	Dose reduction	Relling et al., 2019
Mercaptopurine	*NUDT15*	*3/*3	Increased myelosuppression risk	Dose reduction	Yang et al., 2024
5-Fluorouracil	*DPYD*	*2A, *13	Increased risk of neutropenia, mucositis	Dose reduction or alternative	García-Alfonso et al., 2022
Imatinib	*CYP3A5*	*3/*3	Reduced clearance, increased toxicity	Dose adjustment	Cereja-Pantoja et al., 2024

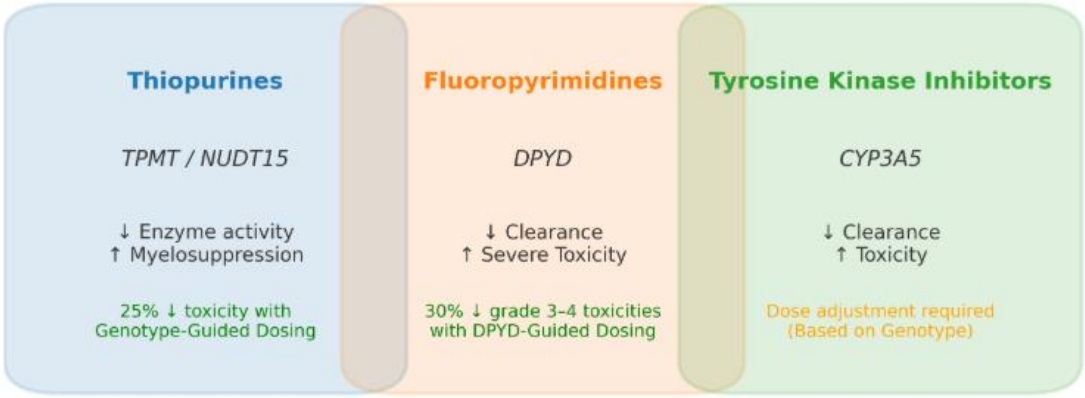

Figure 2. Pharmacogenomics in Chemotherapy: Genes, Variants, and Outcomes

Clinical Decision Support Systems (CDSS)

Clinical decision support systems (CDSS) are a component of the integration of pharmacogenomics into practice that provides real-time guidance through the incorporation of genetic data into electronic medical records (EMRs). In hematology, CDSS systems alert pharmacists for important gene-drug interactions, recommend personalized dose

adjustment, and outline high-risk patients who must be monitored even more intensely (Giri et al., 2019). A 2024 survey in a large hematology clinic demonstrated that CDSS implementation reduced medication errors by 18%, both in anticoagulants and chemotherapeutics with published pharmacogenomic interactions (Farmaki et al., 2024). The systems integrate CPIC guidelines and individualized patient genetic information to produce actionable advice, such as dose reduction of TPMT poor metabolizers or substitution therapy in *DPYD* variant patients.

Pharmacists are also responsible for creating and implementing CDSS to tailor such systems to hematology clinics (Dreischmeier et al., 2024). As an example, the PHASER program used CDSS to send *CYP2C9*/*VKORC1* test reports to pharmacists in order to enable rapid warfarin dosing and reduce adverse events by 12% (Dreischmeier et al., 2024). Pharmacists collaborated with IT professionals to design usability interfaces and trained clinicians regarding CDSS implementation, enabling utilization across multidisciplinary teams. Challenges, however, include the lack of standardization within healthcare centers, as different EMR systems do not allow the integration of pharmacogenomic data easily (Sethi et al., 2023). Having clinicians adequately trained to respond to CDSS alerts is also still a barrier that requires ongoing education efforts led by pharmacists.

Pharmacists' Role in Precision Hematology

Pharmacists have an essential role in translating pharmacogenomic knowledge into practice as key intermediaries between genetic test laboratories, physicians, and patients. Pharmacists interpret complex genetic data into practice, recommend personalized treatment, and educate patients on the benefits and limitations of pharmacogenomic-directed treatment (Soefje, 2024). A survey of 300 hematology pharmacists conducted in 2025 reported that 85% saw improved patient outcomes, such as reduced toxicities and enhanced treatment efficacy, after the implementation of pharmacogenomic testing (Hatem et al., 2025). Pharmacists also counsel patients on the control of possible side effects and adherence to genotype-guided regimens, reinforcing confidence and adherence.

Alongside direct patient care, pharmacists lead research and education initiatives aimed at advancing the practice of pharmacogenomics in hematology. They develop training systems that raise the pharmacogenomic literacy of care providers, bridging knowledge gaps and facilitating implementation (Saud Faleh Alanazi, 2024). The precision medicine model at the Mayo Clinic is an example of this role, with pharmacists in pharmacogenomics to guide therapy in hematology/oncology clinics (Soefje, 2024). These professionals collaborate with geneticists and hematologists to design clinical trials, evaluate novel gene-drug relationships, and advocate for the inclusion of pharmacogenomic testing in treatment guidelines. Their vision has given the impetus to establish pharmacogenomics as a standard of care in precision hematology.

Challenges and Barriers

It is, however, not without enormous challenges that acceptance of pharmacogenomics in hematology can be widespread. The cost of genetic testing, which is typically between $200 and $1,000 per panel, is the main challenge, particularly in low-resource settings where there are constrained healthcare budgets (Duffy, 2016). Furthermore,

policies for reimbursement of pharmacogenomic testing vary considerably between health systems, and only around 30% of American insurers reimburse for tests for hematologic conditions, limiting access for many patients (Love-Koh et al., 2021). This limitation disproportionately impacts disadvantaged populations, further increasing healthcare disparities and preventing equitable access to precision medicine.

Ethical issues are also problematic since pharmacogenomic testing can reveal incidental findings, such as predispositions to non-hematologic disorders, that raise issues about patient privacy and informed consent (Marron & Joffe, 2020). Effective consent processes and genetic counseling are required to overcome such barriers, but limited healthcare systems have the infrastructure to consistently provide genetic counseling services, subjecting patients and practitioners to being unable to handle intricate genetic information (Marron & Joffe, 2020). This rift emphasizes the need for consistent ethical strategies to guide pharmacogenomic application in clinical practice.

The other barrier is the existence of evidence gaps for the majority of gene-drug interactions, particularly for the rare variants. While interactions like *CYP2C9*/*VKORC1* with warfarin and *TPMT/NUDT15* with thiopurines are well established, more rare variants generally lack sufficient clinical data to support routine testing (Alhabeeb et al., 2025). CPIC guidelines need to be extended to new therapies through large-scale, prospective clinical trials to validate these interactions and make pharmacogenomic recommendations clinically relevant and evidence-based (Fountzilas et al., 2022).

Finally, a lack of pharmacogenomic knowledge among clinicians, including hematologists and oncologists, dissuades the adoption of genotype-directed therapies. A 2025 study found that only 40% of hematologists felt proficient in interpreting pharmacogenomic tests, reducing an essential need for targeted education (Hatem et al., 2025). Pharmacists play a unique role in bridging this gap by developing and offering training sessions, collaborating with interprofessional teams, and promoting the integration of pharmacogenomics within medical school curricula (Saud Faleh Alanazi, 2024). Their expertise and leadership are essential in overcoming these barriers and driving precision pharmacy in hematology ahead.

Future Directions

The future of precision pharmacy in hematology is to integrate multi-omics information—genomics, proteomics, and metabolomics—into artificial intelligence (AI) so that drug responses will be predicted with unprecedented accuracy (Taherdoost & Ghofrani, 2024). AI-driven CDSS can take complex genetic profiles, such as polygenic risk scores and environmental exposure, into account to make personalized treatment recommendations (Seyhan & Carini, 2020). AI algorithms, for example, can predict optimal TKI doses in CML by combining *BCR-ABL1* mutation with *CYP3A5* genotypes to improve response rates and lower toxicities.

Scaling up preemptive pharmacogenomic testing, as in the PHASER program, would be capable of streamlining therapy by identifying at-risk patients before initiating therapy (Dreischmeier et al., 2024). The approach is particularly appealing in hematology, where patients are likely to require multiple drugs sharing similar pharmacogenomic profiles. Pharmacists must advocate for standardized testing guidelines and universal payments to ensure equal access, most critically in low-resource settings (Love-Koh et al., 2021). Interdisciplinary research initiatives, such as the NCI Genomic Data Commons, will continue to establish novel gene-drug interactions, guiding the development of complete

pharmacogenomic databases for hematologic malignancies (Jensen et al., 2017).

Conclusion

Pharmacogenomics has revolutionized precision pharmacy in hematology, enabling individualized anticoagulant and chemotherapeutic therapy that improves patient care. Key gene-drug pairs, for example, *CYP2C9*/*VKORC1* for warfarin and *TPMT/NUDT15* for thiopurines, guide clinical practice, reducing side effects such as bleeding and myelosuppression. Pharmacists are at the forefront of this revolution, coordinating genetic testing, result interpretation, and healthcare team education. Beyond all the obstacles of cost, availability, and clinician education, the horizon is bright with advances in AI, multi-omics, and preemptive testing. Continued research and advocacy through policy reform, led by pharmacists, will make pharmacogenomics an integral part of hematologic treatment, delivering safer and more effective medicines to patients worldwide.

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الصيدلة الدقيقة في أمراض الدم: دمج الصيدلة الجينية في صنع القرار السريري لمضادات التخثر والعوامل الكيميائية العلاجية

ملخص

الخلفية :تقوم الصيدلة الدقيقة، مدعومة بالصيدلة الجينية، بتخصيص علاج مضادات التخثر والعلاج الكيميائي بناءً على التركيب الجيني للمريض، مما يعزز نتائج العلاج في أمراض الدم .**الهدف :**تحاول هذه المراجعة معالجة دمج الصيدلة الجينية في صنع القرار المتعلق بمضادات التخثر والعوامل الكيميائية العلاجية في أمراض الدم مع إشارة خاصة لدور الصيدلي .**الطرق :**تم إجراء مراجعة منهجية للأدبيات باستخدام قواعد PubMed و Scopusو Web of Scienceللدراسات بين عامي 2020 و2024. ركزت معايير الاختيار على التطبيقات الصيدلانية الجينية في أمراض الدم. تم دمج البيانات لتحديد تفاعلات الجينات والأدوية الرئيسية، واستراتيجيات التنفيذ في العيادات، والمعوقات .**النتائج :**خفض الفحص الصيدلاني الجيني لـ *CYP2C9/VKORC1* لتحديد جرعة الوارفارين من أحداث النزيف بنسبة 15%، بينما خفض فحص TPMT/NUDT15للثيوبورينات من كبت النخاع بنسبة 25%. أدت أنظمة دعم القرار السريري (CDSS) والاختبارات الاستباقية، مثل برنامجPHASER ، إلى تحسين النتائج بنسبة 18-12%. تشمل القيود التكلفة، والتوافر، وقصور تدريب clinicians.**الاستنتاج :**تحسن الصيدلة الجينية safety وفعالية علاج مضادات التخثر والعلاج الكيميائي في أمراض الدم، مع كون الصيادلة المنفذين المركزيين. ستواصل التقدمات في الذكاء الاصطناعي multi-omics(العلوم المتعددة) تحسين الصيدلة الدقيقة بشكل تدريجي.

الكلمات المفتاحية :الصيدلة الجينية، الصيدلة الدقيقة، أمراض الدم، مضادات التخثر، العوامل الكيميائية العلاجية.