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FROM MOLECULAR TARGET TO GREEN MOLECULE: MODERN APPROACHES IN DESIGNING IMATINIB-BASED ANTICANCER AGENTS

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ABSTRACT

Leukemia is a type of hematological malignancy of clinical importance as part of cancer that is a serious health burden in the globe with its development being precipitated by molecular defects such as the BCR-ABL fusion kinase. The finding of BCR-ABL as an oncogenic driver constitutively active BCR-ABL revolutionized the entire oncology arena with the opportunity to develop rational therapy the most memorable being imatinib, a selective tyrosine kinase inhibitor that made chronic myeloid leukemia to become a manageable disease. Although it has been successful clinically, imatinib therapy has been associated with a number of limitations such as drug resistance, inadequate selectivity and pharmacokinetic obstacles. As a result, a significant amount of medicinal chemistry work has been done to create new imatinib analogs by structural modification, bioisosteric replacement, optimization of side chains, hybrid pharmacophore, and computational control of selectivity. Along with these developments, increased environmental issues that come with traditional drug production have also spurred the need to use green chemistry technologies. Solvents free reactions, microwave and ultrasound catalysis, biocatalysis, ionic liquid, multicomponent reactions, nanocatalysts and flow chemistry are all environmentally benign and have better yields, reduced toxicity, and sustainable production benefits. Rational drug design as a means of integrating the green synthetic technologies with an interdisciplinary approach towards the creation of next-generation BCR-ABL inhibitors, which are therapeutically effective and green at the same time, is a promising approach. This review will identify new developments in green-engineered imatinib derivatives design and synthesis and optimization, and comment on future trends such as personalized therapy, nanotechnology-based delivery, artificial intelligence-aided drug discovery, and sustainable pharmaceutical production.

INTRODUCTION

Cancer has been identified to be one of the greatest global health problems in terms of death rates per year which is in the millions and a big socioeconomic burden on the global population [1]. More effective and safer treatment methods are required to combat the cancer pandemic, which is rapidly rising to the ranks of the world's leading causes of death, with an expected worldwide burden of 10 million cases in 2020, according to recent projections [2-4].

The most well-known example of this is chronic myeloid leukemia (CML), which develops when the chromosomes 9 and 22 move back and forth, creating the Philadelphia chromosome. This causes the BCR-ABL fusion gene to produce a tyrosine kinase that is always active, which in turn causes hematopoietic stem cells to multiply uncontrollably and undergo malignant transformation.[5]

This discovery of BCR-ABL as a pathogenic kinase was eventually used to create imatinib mesylate, the first successful molecularly-focused anticancer agent designed to selectively inhibit one oncogenic protein, and it established a paradigm shift in the sphere of oncology into precision medicine.[6]

Although still commonly deployed, conventional chemotherapy has shortcomings due to its lack of selectivity since cytotoxics attack rapidly growing cells whether malignant or normal [7]. Unlike that, the targeted therapies target a specific molecular abnormality present in cancer cells, leading to high therapeutic responses and minimum harm to normal tissues, which is why they are a significant development in the field of oncology today and provides motivation to the development of drugs that use selective molecular inhibition [8-10.

Also in parallel with therapeutic innovation, increased environmental and sustainability awareness of the problems with pharmaceutical synthesis. These principles have become of great significance to medicinal chemistry, where atom economy, improved toxicity, and reduced environmental impact are sought through the implementation of hazardous reagents, toxic solvents, and energy-intensive methodologies prone to generate large quantities of chemical waste.[11-12] The design of BCR-ABL-targeted imatinib derivatives based on green chemistry is a promising interdisciplinary approach, which can be used to incorporate precision oncology with environmental responsible drug development.

BCR-ABL Kinase as a Therapeutic Target

The constitutively active tyrosine kinase known as the BCR-ABL fusion protein is created when the BCR gene and the Abelson murine leukemia (ABL1) gene are fused together through a reciprocal translocation of t(9;22)(q34;q11) [13].ABL -BCR is a chimeric oncoprotein, with retained oligomerization domains, and an active catalytic kinase domain, leading to the generation of an open active conformation which re-phosphorylates its downstream targets [14]. BCR-ABL does not exhibit tight autoregulation of its catalytic activity as ABL does but instead acquires a stable open active conformation which can keep re-phosphorylating downstream targets.

BCR-ABL triggers oncogenic signaling pathways that facilitate unregulated proliferation, low apoptosis, altered adhesion, and genomic instability [13]. Fusion kinase activation involves multiple signaling pathways, including STAT, PI3K/AKT, and MAPK. In BCR-ABL+ cells, phosphorylation of STAT5 is an ongoing process that increases survival by upregulating anti-apoptotic genes [15]. Activation of the PI3K/AKT pathway aids in metabolic adaptation and resistance to apoptosis, and it also supports cell proliferation and cell-cycle progression [16–17].

In disease progression and in therapeutic resistance, BCR-ABL signaling is also at the center stage. High levels of kinase activity correlate with recurring chronic phase to accelerated and blast crisis chronic myeloid leukemia [18]. Resistance could be caused by point mutation, or amplification of other signaling pathways that could bypass BCR-ABL, hence efforts are ongoing to develop a better way of making the essential BCR-ABL an effective target of therapy [19].

Imatinib: Chemistry and Pharmacology

It is a 2-phenylaminopyrimidine derivative that was sensiblyintended to inhibit ATP-binding site of BCR-ABL tyrosine kinase [20] .The chemical structure of imatinib atoms is

composed of the key pharmacophores such as the core of pyrimidines, benzamide residue, and phenyl rings substituted with hydrogen bonds and hydrophobic interactions in the kinase active site [21]. The chemical structure of imatinib given below in fig.1.

Fig.1. Structure if Imatinib

Imatinib has a relative long oral bioavailability (98%), high plasma protein binding and it is metabolized by the liver mainly via CYP 3A4.22 It has a relatively long half-life of about 18 hours, and can be given to most patients once in a day [18].

Even in the case of its revolutionary success, imatinib therapy has its limitations. The major issues have been resistance especially when point mutation in BCR-ABL kinase domain like T315I occurs, which interrupts the binding of drugs [19]. Other limitations include adverse effects, variation in patient response, drug-drug interactions and resistance as a result of leukemic stem cells [23].

Imatinib therapy is limited in spite of its revolutionary success. The biggest complication is resistance, especially the point mutations in the BCR-ABL kinase domain (e.g. T315I) that inhibit drug binding [19]. Other limitations are adverse effects, inconsistent patient response, drug to drug interaction as well as inadequate eradication of leukemic stem cells [24]. These deficiencies have driven the process of formulating second- and third-generation tyrosine kinase blockers and structurally altered imatinib analogues of better strength, selectivity and resistance override properties [25].

Rationale for Designing Novel Imatinib Derivatives

The BCR-ABL inhibition has been established as a new paradigm in the clinical success of imatinib, however, therapeutic resistance and the absence of ideal selectivity and pharmacokinetic limitations has triggered an unprecedented medicinal chemistry effort to develop superior analogues [26]. The requirement to counteract mutations of the kinases domain, the need to enhance binding affinity, reduction of off-target toxicity and enhancement of drug-like properties have seen the adoption of rational structural modification strategies guided by structure-activity relationship (SAR) analyses and crystallographic studies of kinase-inhibitor complexes to new imatinib derivatives [27].

Structural Modification Strategies

Medicinal chemists take advantage of the systematic mutations of the imatinib scaffold to increase potency and resistance mutations. Structural modifications may be substitution of aromatic rings, alteration of heterocyclic cores or alteration of the linker regions, which influences conformational flexibility and binding orientation. The mutations can enhance hydrogen bonds, hydrophobic interaction, and van der Waals forces in the ATP-binding pocket of BCR-ABL [28]. The design using crystallographic data and guiding structure has demonstrated that such minor alterations of the substituents can alter the kinase affinity and selectivity substantially [27].

Bioisosteric Replacement

One of the most typical ones is the biosterism, when the pharmacological and physicochemical properties are improved without radical changes in the biological activity. Chemical or steric analogues to functional groups can be used to improve metabolic stability, toxicity, and solubility [29]. In the design of kinase inhibitors, bioisosteric substitution of functional groups (amide groups, aromatic rings, or heterocycles) has been demonstrated to increase binding affinity and decrease enzymatic degradation [30]. This technique has proven useful in designing derivatives that can act on

the enzyme despite the presence of resistant mutations on BCR-ABL.

Side-Chain Optimization

Side chain optimization is an important part of drug-target interaction fine-tuning and pharmacokinetics optimization. Engineering at the side-chain can also be used to either increase or decrease lipophilicity, membrane permeability, and plasma protein interactions, and also to increase selectivity between structurally related kinases in the context of imatinib analogues [31]. Side-chain engineering can also be used to enhance aqueous solubility and oral bioavailability without decreasing target affinity [32].

Hybrid Molecule Approach

Hybridization strategies entail the incorporation of pharmacophores of two or more biologically active molecules into a single framework in order to gain synergistic or dual-target activity [33]. Hybrid kinase inhibitors are receiving attention due to the ability of this approach to prevent the development of resistance. Hybrid molecules that have been engineered with other anticancer pharmacophores or epigenetic modulators in the case of BCR-ABL inhibitors have shown increased cytotoxicity and expanded antitumor activities in preclinical models [34].

Target Selectivity Improvement

To reduce adverse effects and enhance therapeutic index, selective inhibition over BCR-ABL versus other kinases is necessary. Selectivity Achieving selectivity has been made possible by having a molecular complementary between the inhibitor and specific structural motifs of the kinase binding site. Designing derivatives that are specific to inactive kinase conformations, or that are specific to mutant structural motifs has become a promising approach to overcoming resistance, as well as sparing normal kinases [35].

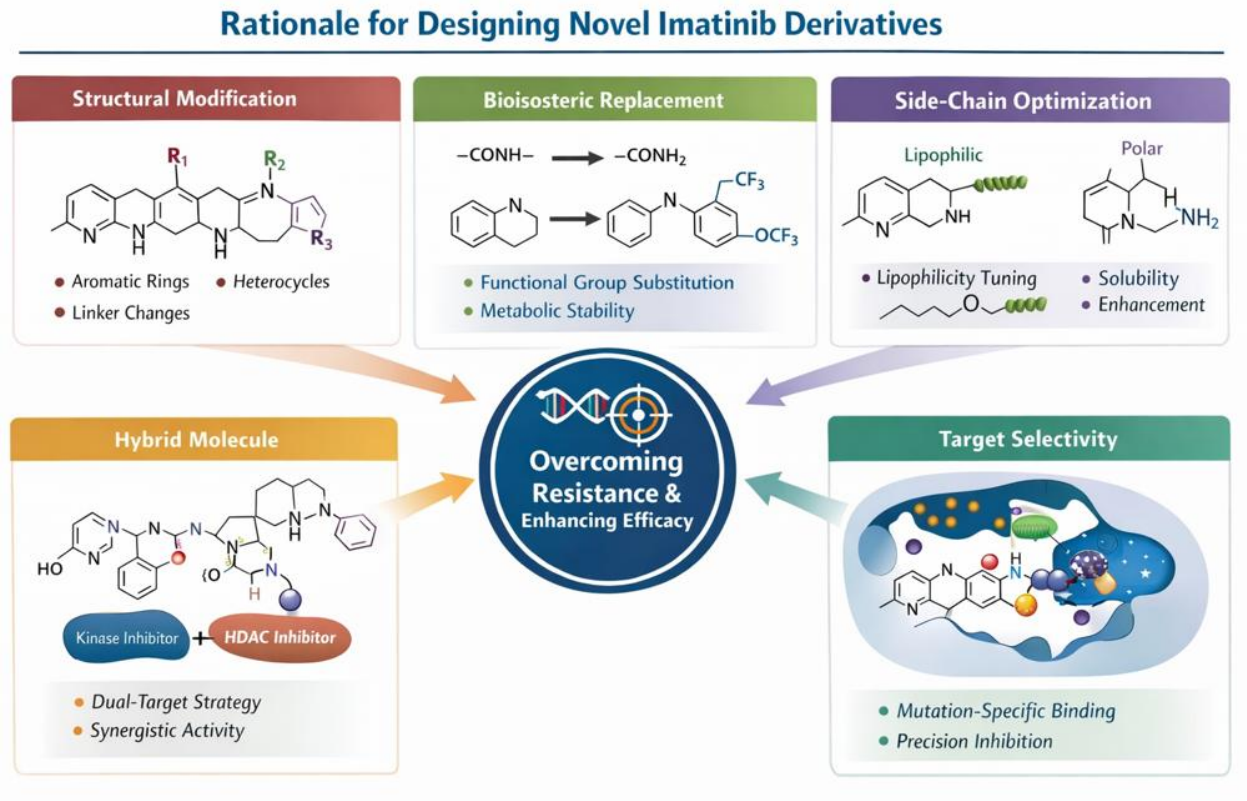

Fig.2. Rational for designing novel Imatinib derivatives

Green Chemistry Principles in Drug Synthesis

Definition and Importance of Green Chemistry

Green chemistry is a philosophy of chemical products and processes that limit or avoid the use and generation of hazardous substances [36]. It is a preventative style of controlling pollution, where safer and more efficient production of drugs is observed on a molecular design scale instead of the remediation of waste formation [37].

The Twelve Principles of Green Chemistry

These foundational principles suggested by Anastas and Warner are waste prevention, atom economy, safer solvents, renewable feedstocks, catalysis, energy efficiency, and inherently safer chemistry.37 These are the guiding principles towards processes by which medicinal chemists can design synthetic routes that are highly resource efficient and fail to expose the environment and human health to significant risks [38].

Benefits in Pharmaceutical Synthesis

Reduced toxicity: Green synthetic pathways minimize the use of reagents and by-products which are hazardous to occupational and patient safety [39].

Lower environmental impact: Less of its waste generation and environmental pollution by the use of benign solvents and catalytic methods [40].

Cost efficiency: When the reactions are conducted efficiently with high atom economy, less raw material will be used and the cost of waste disposal will be reduced [39].

Sustainable production: Recyclable catalysts, renewable reagents, and low-energy technologies are helpful in sustainability of industries in the long run [38]. All these advantages make green chemistry particularly relevant in the development of improved tyrosine kinase inhibitors such as new imatinib analogs.

Table 1. Green Synthetic Methods vs Conventional Synthesis in Preparation of Imatinib Derivatives

Method	Reaction Principle	Typical Yield	Reaction Time	Energy Requirement	Solvent Use	Advantages Over Conventional Methods	Limitations	Environmental Impact
Conventional Heating	Thermal activation via external heating	Moderate (50–75%)	Hours–days	High	Organic solvents required	Established protocols; scalable	Long reaction times, side reactions	High waste, VOC emission
Solvent-Free Synthesis	Direct reactant interaction without solvent	High (70–95%)	Minutes–hours	Low–moderate	None	Eliminates solvents, simple workup, high atom economy	Mixing limitations for viscous reactants	Very low environmental burden
Microwave-Assisted	Dielectric heating via microwave irradiation	High (80–98%)	Minutes	Low	Minimal or green solvents	Rapid reactions, improved selectivity, reproducibility	Specialized equipment	Reduced energy consumption
Ultrasound-Assisted	Cavitation-induced localized high temperature/pressure	High (75–95%)	Minutes–hours	Low	Often aqueous or green solvents	Enhanced mass transfer, mild conditions	Scale-up challenges	Environmentally favorable
Biocatalysis	Enzyme-mediated transformation	Very high (80–99%)	Minutes–hours	Very low	Water or buffer	Excellent selectivity, mild conditions, minimal by-products	Enzyme stability and cost	Biodegradable, sustainable
Ionic Liquids / DES	Ionic medium stabilizes transition states	High (75–96%)	Short	Low	Recyclable solvent system	Nonvolatile, reusable, improved solubility	Cost and purification issues	Low atmospheric emissions

One-Pot Multicomponent Reactions	Multiple bond-forming steps in single vessel	Very high (85–98%)	Short	Low	Minimal	Step economy, less purification, high efficiency	Reaction compatibility constraints	Reduced waste generation
Nanocatalysts / Heterogeneous Catalysis	Surface-mediated catalytic activation	High (80–97%)	Short	Low	Often minimal	Recyclable catalysts, easy separation, high activity	Catalyst preparation complexity	Reduced metal waste
Flow Chemistry (Emerging Green Method)	Continuous reaction under controlled flow	High (80–99%)	Very short	Low	Minimal	Precise control, scalable, safer reactions	Equipment cost	Highly sustainable

Future Perspectives

Future versions of BCR-ABL-targeted imatinib derivatives would involve more accurate and sustainable forms of therapy. The individualized medicine products that adopt the application of genomic profiling will enhance the identification of patient-specific inhibitors to defeat the resistance that arises due to mutations. The solution might be either a combination therapy with immunomodulators, epigenetic drugs, or pathway-targeted drugs in order to enhance their efficacy and prevent relapses. There are also Nanoformulations which can be employed to enhance bioavailability, tumor selectivity, toxicity through the application of nanoparticle carriers and targeted delivery systems. In addition to it, AI-aided drug design has been emerging as an effective predictor of structure-activity relationships, molecular properties optimization, and next-generation derivatives discovery. Finally, the principles of sustainable pharmaceutical production based on the principles of green chemistry, will also need to be implemented in order to offer the environmentally responsible low-cost mass production of the future anticancer agents.

CONCLUSION

It was discovered that the BCR-ABL fusion kinase formed an ultimate molecular oncosomolecular target in leukemia therapy and changed the paradigm in particular oncology. A more obvious example of a drug design success of mechanism-based drug design is imatinib, which demonstrates that selective inhibition of a disease-driving protein can achieve some rather impressive clinical outcomes. Nonetheless, recent resistance resistance mutations, toxicity and pharmacological disadvantages indicate the need to have improved derivatives that are superior in their potency, selectivity and pharmacokinetic properties. Scaffold modification, bioisosterism, side-chain engineering, design of hybrid molecules and computational modeling are rational medicinal chemistry strategies that have been effective in optimization of BCR-ABL inhibitors and resistance mechanisms.

Simultaneously, the embedded introduction of the principles of green chemistry into the drug-making process has become the key to the sustainable pharmaceutical innovation. The modern environmentally-friendly guidelines provide efficient, powerful synthetic synthesis methods and minimise the toxicity of the reagents, solvent wastes, and the consumption of energy. They are less harmful to the environment, cost effective and scalable which is aligned with the world sustainability goals in drug development.The trends into the future are expected due to the overlap of precision medicine, combination therapy strategy, nano-enabled delivery, and artificial intelligence-mediated molecular design. These innovations or a combination of both will accelerate the development of safer, more efficient and environmentally friendly agents that shall act against BCR-ABL. It can be concluded then that the more comprehensive future evolution of the green synthetic methods in combination with the rational drug design will be a core in the next generation of the anticancer molecules.

REFERENCES

1. Sung H, Ferlay J, Siegel RL, Laversanne M, Soerjomataram I, Jemal A, et al. Global cancer statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin. 2021;71(3):209-49. doi:10.3322/caac.21660.
2. World Health Organization. Cancer fact sheet. Geneva: WHO; 2024.

3. Deininger MW, Goldman JM, Melo JV. The molecular biology of chronic myeloid leukemia. Blood. 2000;96(10):3343-56. doi:10.1182/blood.V96.10.3343.
4. Rowley JD. A new consistent chromosomal abnormality in chronic myelogenous leukemia identified by quinacrine fluorescence and Giemsa staining. Nature. 1973;243:290-3. doi:10.1038/243290a0.
5. Lugo TG, Pendergast AM, Muller AJ, Witte ON. Tyrosine kinase activity and transformation potency of bcr-abl oncogene products. Science. 1990;247(4946):1079-82. doi:10.1126/science.2408149.
6. Druker BJ, Talpaz M, Resta DJ, Peng B, Buchdunger E, Ford JM, et al. Efficacy and safety of a specific inhibitor of the BCR-ABL tyrosine kinase in chronic myeloid leukemia. N Engl J Med. 2001;344(14):1031-7. doi:10.1056/NEJM200104053441401.
7. Cohen MH, Williams G, Johnson JR, Duan J, Gobburu J, Rahman A, et al. Approval summary for imatinib mesylate capsules in the treatment of chronic myelogenous leukemia. Clin Cancer Res. 2002;8(5):935-42.
8. Sawyers CL. Targeted cancer therapy. Nature. 2004;432(7015):294-7. doi:10.1038/nature03095.
9. Holohan C, Van Schaeybroeck S, Longley DB, Johnston PG. Cancer drug resistance: an evolving paradigm. Nat Rev Cancer. 2013;13(10):714-26. doi:10.1038/nrc3599.
10. Anastas PT, Warner JC. Green chemistry: theory and practice. New York: Oxford University Press; 1998.
11. Li CJ, Trost BM. Green chemistry for chemical synthesis. ProcNatlAcad Sci U S A. 2008;105(36):13197-202. doi:10.1073/pnas.0804348105.
12. Kümmerer K. Pharmaceuticals in the environment: sources, fate, effects and risks. 3rd ed. Berlin: Springer; 2008.
13. Nagar B, Bornmann WG, Pellicena P, Schindler T, Veach DR, Miller WT, et al. Crystal structures of the kinase domain of c-Abl in complex with the inhibitor STI-571. Cancer Res. 2002;62(15):4236-43. doi:10.2210/pdb1iep/pdb.
14. Ilaria RL, Van Etten RA. P210 and P190 BCR-ABL induce the tyrosine phosphorylation and DNA binding activity of multiple specific STAT family members. J Biol Chem. 1996;271(49):31704-10. doi:10.1074/jbc.271.49.31704.
15. Skorski T, Bellacosa A, Nieborowska-Skorska M, Majewski M, Martinez R, Choi JK, et al. Transformation of hematopoietic cells by BCR-ABL requires activation of a PI-3K/Akt-dependent pathway. EMBO J. 1997;16(20):6151-61. doi:10.1093/emboj/16.20.6151.
16. Cortez D, Kadlec L, Pendergast AM. Structural and signaling requirements for BCR-ABL-mediated transformation and inhibition of apoptosis. Mol Cell Biol. 1995;15(10):5531-41. doi:10.1128/MCB.15.10.5531.
17. Kantarjian HM, Talpaz M, O'Brien S, Giles F, Garcia-Manero G, Faderl S, et al. Chronic myelogenous leukemia: a concise update. Blood. 2002;100(2):477-88. doi:10.1182/blood.V100.5.1590.
18. Gorre ME, Mohammed M, Ellwood K, Hsu N, Paquette R, Rao PN, et al. Clinical resistance to STI-571 cancer therapy caused by BCR-ABL gene mutation. Science. 2001;293(5531):876-80. doi:10.1126/science.1062538.
19. Druker BJ, Lydon NB. Lessons learned from the development of an abl tyrosine kinase inhibitor for chronic myelogenous leukemia. J Clin Invest. 2000;105(1):3-7. doi:10.1172/JCI9083.
20. Schindler T, Bornmann W, Pellicena P, Miller WT, Clarkson B, Kuriyan J. Structural mechanism for STI-571 inhibition of Abelson tyrosine kinase. Science. 2000;289(5486):1938-42. doi:10.1126/science.289.5486.1938.
21. Peng B, Dutreix C, Mehring G, Hayes M, Ben-Am M, Seiberling M, et al. Absolute bioavailability of imatinib orally versus intravenous infusion. J Clin Pharmacol. 2004;44(2):158-62. doi:10.1177/0091270003262101.
22. Deininger M, Buchdunger E, Druker BJ. The development of imatinib as a therapeutic agent for chronic myeloid leukemia. Blood. 2005;105(7):2640-53. doi:10.1182/blood-2004-08-3097.
23. O'Hare T, Zabriskie MS, Eiring AM, Deininger MW. Pushing the limits of targeted therapy in chronic myeloid leukemia. Nat Rev Cancer. 2012;12(8):513-26. doi:10.1038/nrc3317.
24. Apperley JF. Mechanisms of resistance to imatinib in chronic myeloid leukemia. Lancet Oncol. 2007;8(11):1018-29. doi:10.1016/S1470-2045(07)70342-X.
25. Zhang J, Yang PL, Gray NS. Targeting cancer with small molecule kinase inhibitors. Nat Rev Cancer. 2009;9(1):28-39. doi:10.1038/nrc2559.
26. Liu Y, Gray NS. Rational design of inhibitors that bind to inactive kinase conformations. Nat Chem Biol. 2006;2(7):358-64. doi:10.1038/nchembio799.
27. Meanwell NA. Synopsis of some recent tactical applications of bioisosteres in drug design. J Med Chem. 2011;54(8):2529-91. doi:10.1021/jm1013693.

28. Patani GA, LaVoie EJ. Bioisosterism: a rational approach in drug design. Chem Rev. 1996;96(8):3147-76. doi:10.1021/cr950066q.
29. Waring MJ. Lipophilicity in drug discovery. Expert Opin Drug Discov. 2010;5(3):235-48. doi:10.1517/17460441003605098.
30. Knight ZA, Shokat KM. Features of selective kinase inhibitors. Chem Biol. 2005;12(6):621-37. doi:10.1016/j.chembiol.2005.04.011.
31. Morphy R, Rankovic Z. Designed multiple ligands: an emerging drug discovery paradigm. J Med Chem. 2005;48(21):6523-43. doi:10.1021/jm058225d.
32. Nepali K, Sharma S, Sharma M, Bedi PM, Dhar KL. Rational approaches, design strategies, structure-activity relationship and mechanistic insights for anticancer hybrids. Eur J Med Chem. 2014;77:422-87. doi:10.1016/j.ejmech.2014.03.018.
33. Cohen P. Protein kinases: the major drug targets of the twenty-first century? Nat Rev Drug Discov. 2002;1(4):309-15. doi:10.1038/nrd773.
34. Lionta E, Spyrou G, Vassilatis DK, Cournia Z. Structure-based virtual screening for drug discovery: principles, applications and recent advances. Curr Top Med Chem. 2014;14(16):1923-38. doi:10.2174/1568026614666140929124445.
35. Anastas PT, Eghbali N. Green chemistry: principles and practice. Chem Soc Rev. 2010;39(1):301-12. doi:10.1039/B918763B.
36. Constable DJC, Jimenez-Gonzalez C, Henderson RK. Perspective on solvent use in the pharmaceutical industry. Org Process Res Dev. 2007;11(1):133-7. doi:10.1021/op060170h.
37. Kümmerer K. Sustainable from the very beginning: rational design of molecules by life cycle engineering as an important approach for green pharmacy. Green Chem. 2007;9:899-907. doi:10.1039/b618298b.
38. Tanaka K, editor. Solvent-free organic synthesis. Weinheim: Wiley-VCH; 2003. doi:10.1002/3527601821.
39. Kappe CO. Controlled microwave heating in modern organic synthesis. Angew Chem Int Ed. 2004;43(46):6250-84. doi:10.1002/anie.200400655.
40. Mason TJ. Sonochemistry and the environment: providing a "green" link between chemistry, physics and engineering. UltrasonSonochem. 2007;14(4):476-83. doi:10.1016/j.ultsonch.2006.10.008.
41. Sheldon RA, Woodley JM. Role of biocatalysis in sustainable chemistry. Chem Rev. 2018;118(2):801-38. doi:10.1021/acs.chemrev.7b00203.
42. Plechkova NV, Seddon KR. Applications of ionic liquids in the chemical industry. Chem Soc Rev. 2008;37(1):123-50. doi:10.1039/B006677J.
43. Smith EL, Abbott AP, Ryder KS. Deep eutectic solvents (DESs) and their applications. Chem Rev. 2014;114(21):11060-82. doi:10.1021/cr300162p.
44. Dömling A, Wang W, Wang K. Chemistry and biology of multicomponent reactions. Chem Rev. 2012;112(6):3083-135. doi:10.1021/cr100233r.
45. Astruc D. Introduction: nanoparticles in catalysis. Chem Rev. 2020;120(2):461-3. doi:10.1021/acs.chemrev.8b00696.

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