TPM Vol. 32, No. S3, 2025
ISSN: 1972-6325
https://www.tpmap.org/

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THE ROLE OF PHARMACOGENOMICS IN OPTIMIZING DRUG THERAPY

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Abstract
In recent years, the clearance procedure for new medications has slowed significantly. novel approaches to drug research are required in order to expedite the development of novel medications. While pharmacogenomics examines how many gene variants work together to determine a patient's reaction to medication treatment, pharmacogenetics studies genetic determinants underlying interindividual variability in drug response. As a result, these techniques can be applied in the drug development process to pinpoint patient subgroups that have enhanced response and/or benefit/risk ratio following therapy. The authors give examples of how pharmacogenetics and pharmacogenomics are used in the development of lung, cardiovascular, cancer, and bone diseases, as well as illustrate the possible economic benefits of their development.
Keywords: Pharmacogenomics, simultaneous, effective responses

1. INTRODUCTION

With the goods medication development appears to have hit a standstill today. We need to try something different. That thing that bridges the gap between the lab and the clinic is called translational medicine. By enabling tailored drug creation, this advancement may help to expand clinical research and illness management[1]. The number of new molecular entities that have received marketing authorization has declined in recent years, but development costs have sharply grown. Pharmacogenetics investigates how variations in DNA sequence affect drug response, whereas pharmacogenomics looks at changes in the characteristics of ribonucleic acid and deoxyribonucleic acid in connection to drug response. Pharmacogenomics is a subfield of precision medicine that examines the impact of genetic variants on the metabolism and response to medications [2]. More precisely, pharmacogenetics examines how changes in one or a few genes affect medication responsiveness using genetic, epigenetic, and nutrigenetic techniques[11]. Additionally, before late-stage research, we may be choosing the wrong dose or dose regimens or evaluating candidates in inappropriate trials. The last and most significant issue is that translational medicine techniques are not being effectively applied during the medication development process.

Translational medicine is a cross-disciplinary science that connects laboratory investigation with clinical investigation. Translational medicine aims to try out, in humans, new therapeutic approaches that are developed through experimentation [3]. A similar medication development philosophy has been laid out by the US FDA. Considering that most compounds that enter clinical development will fail, translational research could help improve medication development by making it quicker, better, or less expensive. Translational medicine, in particular, can help detect failures at an earlier stage of development [4]. The outcome of the trial, whether good or negative, may be much more confidently predicted when a sample of patients who are more likely to respond favourably to a certain medication is found. Improving a molecule's data quality as it progresses through later stages of development is also crucial[16].

Pharmacogenomics investigates the relationships between particular genes and medications by applying pharmacogenetics to the complete genome [12]. The study of pharmacogenetics examines how a patient's genetic makeup influences their pharmaceutical action, dosage, and usage. Precision medicine is based on pharmacogenetics research, which can determine which patients will respond before medication is administered. Pharmacogenetics is very interested in genetic variations that affect liver enzymes and drug transporter proteins. Of particular importance are genetic variations that impact medication pharmacodynamic profiles, such as variations in receptor protein expressions. However, pharmacogenomics is associated with the entire genome

rather than just a specific gene's SNP. Pharmacogenomics is the study of all an organism's genes, whether or not they are expressed in each physiological condition.

2. REVIEW OF LITERATURE

Examples from the fields of cardiovascular disease, asthma, cancer, and osteoporosis highlight the potential benefits of using translational medicine to inform decisions throughout the drug development stage. The identification of a more homogeneous patient population enriches the target population in all of these examples, making it simpler to decide whether to proceed with later stage development with particular compounds. It has long been known that some medications' metabolism is influenced by genetics [10]. Drug metabolizing enzyme genetic variations have been identified that explain interindividual variations in drug concentrations and the pharmacodynamics (safety, effects, etc.) that go along with them[5]. Both pharmacogenetics and pharmacogenomics can provide these insights; the former focuses on the effects of a single gene mutation, while the latter examines the combined effects of several mutations that could affect the drug's toxicity and effectiveness. Pharmacogenetics is especially useful in predicting a severe idiosyncratic reaction or in explaining a patient's ability to metabolize the therapeutic intervention in question, which increases the likelihood of delivering a therapeutic plasma level of the active reagent that would interact with the target in question. By using germ-line DNA or tumor DNA in the oncology example to identify patients with the target disease entity based on a more predictable pharmacodynamic response to the therapeutic intervention, pharmacogenomics, by extension, has the potential to be important.

In the case of congestive heart failure, combining metoprolol with a pharmacogenomic and pharmacogenetic approach may be particularly appealing. A few years ago, a study looked at two particular germline mutations on the alpha 2c and beta 1 receptors that appeared to predict increased risk for congestive heart failure and hyperadrenergic activity in the myocardium. In the former, myocyte hypertrophy, elevated cardiac contractility, and ultimately congestive heart failure are caused by a polymorphism that favours higher intrinsic activity of the beta 1 receptor. In the latter, a deletion polymorphism of the presynaptic alpha receptor inhibits the presynaptic control of norepinephrine release, hence increasing adrenergic tone [13]. Maintaining constant plasma concentrations of metoprolol in a subgroup of patients who are most at risk for illness is one of the primary objectives of translational research, which aims to make drug development more consistent and predictable [6].

3. MATERIALS AND METHODS

The effectiveness and possible side effects of prescription medications are predicted using pharmacogenomics testing. However, pharmacogenomics research in Africa is not up to par with global norms. To put pharmacogenomics into reality, researchers throughout Africa require data sharing and infrastructure support. Digital storage and quick, secure access to data for authorized users are essential for pharmacogenomics. Pharmacogenomics data is frequently linked to electronic medical record systems, which are dreadfully inadequate in Africa, particularly Ethiopia. Different medication sensitivities are caused by individual variances in genetics, environment, and illness. In addition to affecting the local and systemic exposure of a drug, genetic variations can alter its pharmacokinetics and pharmacodynamics, which can alter the drug's response by altering the function of the drug target. the majority of pharmacogenomics indicators that have been shown to improve treatment outcomes [7]. Individual variability in the genes generating proteins involved in immunological or pharmacological reactions to drugs account for a large portion of the diversity in treatment efficacy and side effect risk. Given the enormous advances in genetic analysis technology, a real tailored drug response prediction must take into account millions of uncommon mutations. The number of genetic variants crucial for medicine action is significantly greater than previously thought. As far as the reviewers are aware, no thorough investigation of the ramifications of pharmacogenetics and pharmacogenomics has been carried out in Ethiopia, either through systematic reviews or scoping studies.

To summarize the body of information and pinpoint areas that may require more investigation, a scoping review is a helpful technique [8]. Research involving patients, papers written exclusively in English, and any type of study, original work, review, or publishing in the grey literature were the criteria used to decide which studies should be included. No limitations on the ages or years of publication. Studies without pharmacognosy, medicine, or indicators are not included[14].

4. RESULT AND DISCUSSION

The potential clinical advantage of being able to identify a subset of persons with a superior benefit-risk profile must be weighed against the expense of any diagnostic tests necessary to do so. A model has been developed to

predict the potential cost impact of selecting a preferred starting medication based on a hypothetical pharmacogenomic test [9]. When the "Test All" approach is used, more patients end up in the less costly regions of the distribution.

Table1:DemographicDetails

Parameter	Test (%) (n=2025)	Control(%) (n = 2040)	Total(%) (n=4065)	P value
Males	1140 (56.2)	1146(56)	2286 (56.2)	0.889
Females	885(43.7)	894(44)	1779 (43.8)	
Age <18 18-29 30-49 50-59 60-79 ≥80	 1(0.05) 45(2.2) 779(38.4) 814(40.1) 216(10.6) 170(8.39)	 1(0.05) 66(3.23) 723(35.4) 825(40.4) 265(12.9) 160(7.8)	 2(0.05) 111(2.7) 1502 (36.9) 1639 (40.3) 481(11.8) 330(8.1)	0.022
AverageAge	51.56±15.87 (range–17-90)	49.32±16.46 (range–16-91)	53.52±15.54 (range–16-91)	0.11
Education Illiterate Upto5Grade 6-10 Grade PreUniversity Graduate and above	 427(21) 519(25.6) 554(27.3) 475(23.4) 50(2.4)	 580(28.4) 480(23.5) 686(33.6) 274(13.4) 20(0.9)	 1007(25) 999(24.5) 1240 (30.5) 749(18.4) 70(1.7)	<0.001
Averagenumberof medications prescribed	8.10± 3.67 (range–3-17)	6.60± 2.83 (range–2-17)	7.34± 3.35 (range–2-17)	0.024
CKDStages Stage1 Stage2 Stage3 Stage4 Stage5	 280(13.8) 311(15.3) 530(26.1) 590(29.1) 314(15.5)	 298(14.6) 330(16.1) 499(24.4) 583(28.5) 330(16.1)	 578(14.2) 641(15.7) 1029 (25.3) 1173 (28.8) 644(15.8)	0.647
No of Co-morbidities **0** 1 2 3 4 >4	 180(8.8) 215(10.6) 485(23.9) 561(27.7) 510(25.1) 74(3.65)	 170(8.3) 227(11.1) 478(23.4) 578(28.3) 501(24.5) 86(4.2)	 350(8.6) 442(10.9) 963(23.7) 1139(28) 1011 (24.9) 160(3.9)	0.758

KuppuswamySES Upper(>25) UpperMiddle(16-25) LowerMiddle(11-15) Upper-lower(5-10) Lower(<5)	 18(0.8) 34(1.6) 985(48.6) 945(46.6) 48(2.4)	 22(1) 14(0.7) 1014 (49.7) 913(44.7) 77(3.8)	 40(1) 48(1.1) 1994(49) 1858(45) 125(3.9)	 0.001

The cost savings per patient during a typical run of the testing strategy simulation range from 200 to 767 US dollars (5th and 95th percentile) under the base case, which includes 15% prevalence of the 200 US dollars test phenotype, 74% of overall first line treatment efficacy, and 60% second-line therapy efficacy. Two important factors affecting the financial viability of pharmacogenomics as a treatment approach are the price of genetic variant prevalence testing and the cost of choosing the wrong drug[15].

Table2:DemographicDetailsofAdmittedStudySubjects

Parameter	**Test (n=1991)**	**Control (n=2006)**	**Total(%) (n=3997)**
Males	1115(56)	1119 (55.8)	2234 (55.9)
Females	876(44)	887(44.2)	1763 (44.1)
Age <18 18-29 30-49 50-59 60-79 ≥80	 1(0.05) 39 (1.96) 767(38.52) 509(25.57) 505(25.36) 170(8.54)	 1(0.05) 61 (3.04) 710(35.39) 517(25.77) 557(27.77) 160(7.98)	 2(0.05) 100(2.5) 1477 (36.9) 1026 (25.6) 1062 (26.7) 330(8.3)
AverageAge	53.64±16.48 (range–17-90)	50.04±16.90 (range–16-91)	52.03±16.44 (range–16-91)
Education Illiterate Upto5Grade 6-10 Grade PreUniversity Graduateand above	 425(21.35) 510(25.62) 550(27.62) 460(23.1) 46 (2.31)	 576(28.71) 470(23.43) 680(33.9) 260(12.96) 20(1.0)	 1001(25) 980(24.5) 1230 (30.8) 720(18) 66(1.7)
Average number of medicationsprescribed	8.35± 3.91 (range–3-17)	7.33± 8.92 (range–2-17)	7.83± 6.92 (range–2-17)
Averagelengthofstay in hospital (days)	6.19± 5.31 (range–3-27)	6.74± 1.74 (range–3-31)	6.37± 2.97 (range–3-31)
CKDStages Stage1 Stage2 Stage3 Stage4 Stage5	 280(14.06) 311(15.62) 530(26.62) 590(29.63) 280(14.06)	 298(14.86) 330(16.45) 499(24.88) 583(29.06) 296(14.76)	 578(14.5) 641(16) 1029 (25.7) 1173 (29.4) 576(14.4)

NoofCo-morbidities 0			
1	180(9.04)	170(8.47)	350(8.8)
2	210(10.55)	222(11.07)	432(10.8)
3	480(24.11)	470(23.43)	950(23.8)
4	550(27.62)	570(28.41)	1120(28)
>4	501(25.16)	489(24.38)	990(24.8)
	70 (3.52)	85 (4.24)	155(3.8)
KuppuswamySES			
Upper(>25)	18 (0.90)	22 (1.10)	40(1)
UpperMiddle(16-25)	32 (1.61)	12 (0.60)	44(1.1)
LowerMiddle(11-15)	971(48.77)	1005 (50.10)	1976(50)
Upper-lower(5-10)	935(46.96)	904(45.06)	1839(46)
Lower(<5)	35 (1.76)	63 (3.14)	98(2.4)

Therefore, this review's objectives were to outline important research areas, gather substantial information, and ascertain how examined variations related to Ethiopian patients' treatment outcomes. While wild type tumors were unable to stabilize the binding in a similar manner, extensive laboratory analysis of the reactions (an example of the "bedside to bench" paradigm) made it easier to identify mutations in the ATP binding site of the receptor's tyrosine kinase domain. Gefitinib response rates among Japanese patients were higher than the wild type prevalent incidence in Caucasians due to significantly higher proportions of Japanese patients with this particular mutation. One may imagine a few distinct clinical investigations if the translational research paradigm's "bench to bedside" methodology were applied. First, gefitinib could only be taken into consideration in patient types that had EGFR-mutant malignancies other than non-small cell lung cancer (NSCLC). Additional research might be conducted to facilitate future tumor type registrations (such head and neck cancer [HNC]) if it was effective in small, enriched populations of mutant tyrosine kinases.

5. CONCLUSION

CYP2B6 is the main pharmacogenetic factor influencing efavirenz's pharmacokinetics. Research on drugs and genes has frequently shown that drug-induced liver damage is harmful. Precision medicine is highly valued because of the significant genetic diversity among Ethiopians, which demands careful consideration while assessing the effectiveness and side effects of treatments. To confirm the discrepancies between the results, more pharmacogenomics research will be required. The combined effects of multiple pharmacogenomics study components were also proposed by meta-analysis and systematic review. Despite Ethiopian communities' significant genetic diversity, little genetic data is available about them. Through the identification of possible responders, the reduction of adverse drug reactions, and the optimization of dosage, pharmacogenomics research holds the potential to transform the treatment of disease and benefit Ethiopian people. Individualized therapy is crucial for reducing adverse drug reactions (ADRs) and maximizing effectiveness because a single country may have multiple ethnic communities.

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