St. Elizabeth HEALTHCARE

Precision Medicine and Genomics Health | Pharmacy

FROM PILOT TO EXPANSION: FEASIBILITY OF PHARMACOGENETICS BEYOND LARGE ACADEMIC HOSPITALS

Nihal El Rouby, Pharm.D., Ph.D.
University of Cincinnati , St Elizabeth Hospital

May 4th, 2023

OBJECTIVES

- Discuss the structure of the pharmacogenetic implementation program at St Elizabeth, a community-based hospital
- Demonstrate successful implementation stories in two settings: primary care setting and oncology
- Identify future needs, gaps and steps to maintain growth

MOST EARLY IMPLEMENTATION CAME FROM ACADEMIC HOSPITALS

Clinical Pharmacology & Therapeutics / Volume 92, Issue 1 / p. 87-95

Articles

Operational Implementatio

Medicine: The Design of the

J M Pulley, J C Denny, J F Peterson, G F
K Brothers, K Johnson, D C Crawford,

First published: 16 May 2012
https://doi.org/10.1038/clpt.2011.37

PHARMACOGENOMICS, VOL. 1

Institutional Profi

Personalized Me

pharmacogenetic

Julie A Johnson, Amanda

AJMG AMERICAN JOURNAL OF medical genetics PART C
Seminars in Medical Genetics

Research Article

Implementa

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AJMG AMERICAN JOURNAL OF medical genetics PART C
Seminars in Medical Genetics

Research Article

clinical implementation of pre-

lkinson, Kristine R. Crews, Donald K. Baker,
ui, Ulrike M. Reiss ... See all authors

Clinical Pharmacology & Therapeutics / Volume 100, Issue 1 / p. 63-66

Reviews

Implementa

INGENIOUS

MT Eadon, Z
AM Holmes, CA

First published:

Clinical Pharmacology & Therapeutics / Volume 105, Issue 1 / p. 49-52

Practice

Implementation of Pharmacoger

Center: Lessons Learned Over 14

Laura B. Ramsey, Cynthia A. Prows, Kejia
Richard J. Wenstrup, Alexander A. Vinks, Tra

First published: 29 July 2018
https://doi.org/10.1002/cpt.1165

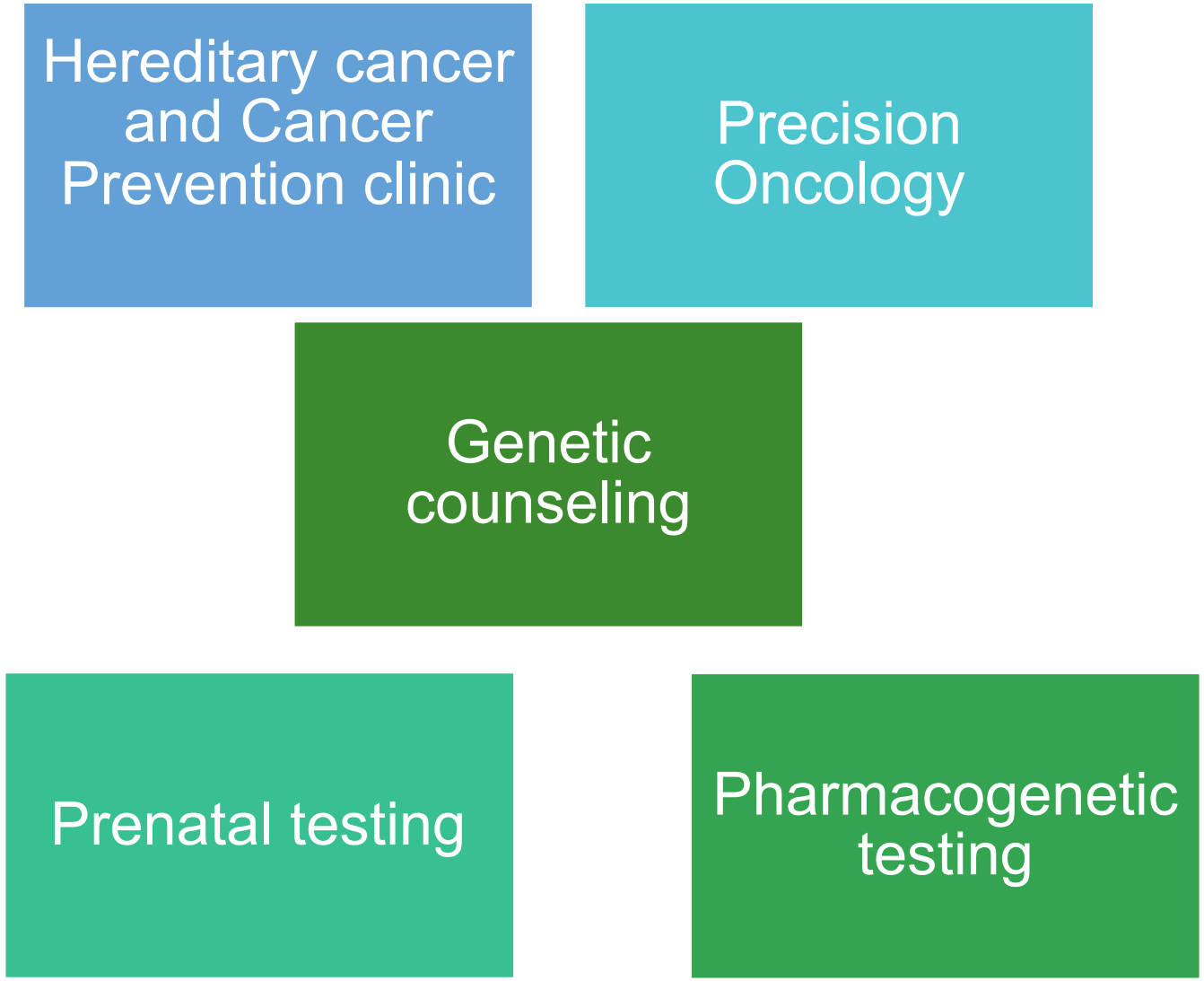
Clinical implementation of pharmacogenomics via a health system-wide research biobank: the University of Colorado experience

Christina L Aquilante‡, David P Kao‡, Katy E Trinkley, Chen-Tan Lin, Kristy R Crooks, Emily C Hearst, Steven J Hess, Elizabeth L Kudron, Yee Ming Lee, Ina Liko, Jan Lowery, Rasika A Mathias, Andrew A Monte, Nicholas Rafaels, Matthew J Rioth, Emily R Roberts, Matthew RG Taylor, Connie Williamson & Kathleen C Barnes

Published Online: 20 Feb 2020 | https://doi.org/10.2217/pgs-2020-0007

ST ELIZABETH CANCER PRECISION CENTER

Precision Center – Structure



Adapted from Jody Wallace's slide

PILLARS OF SUCCESSFUL AND SUSTAINABLE IMPLEMENTATION

Align with implementation goals of stake holders and clinicians

Select the implementation area or areas

Infra-structure: integration within Electronic Health records, Discrete data, clinical support

Education: providers and patients

Sustainability: data (feasibility, utility and economic) and assessment

Dictates your implementation areas and resources

Guarantees your sustainability, success and expansion

PMID: 33829852, PMID: 36059837, PMID: 30926959

PHARMACOGENETICS IMPLEMENTATION TIMELINE

PRIMARY AREAS OF PHARMACOGENETIC IMPLEMENTATION

Primary care

- Guide antidepressant medications

After treatment failure or side effects

Oncology

- Fluoropyrimidine +/- irinotecan
- Supportive care medications

Before Initiation

Cardiovascular

- Statins
- Antiplatelet

After Initiation

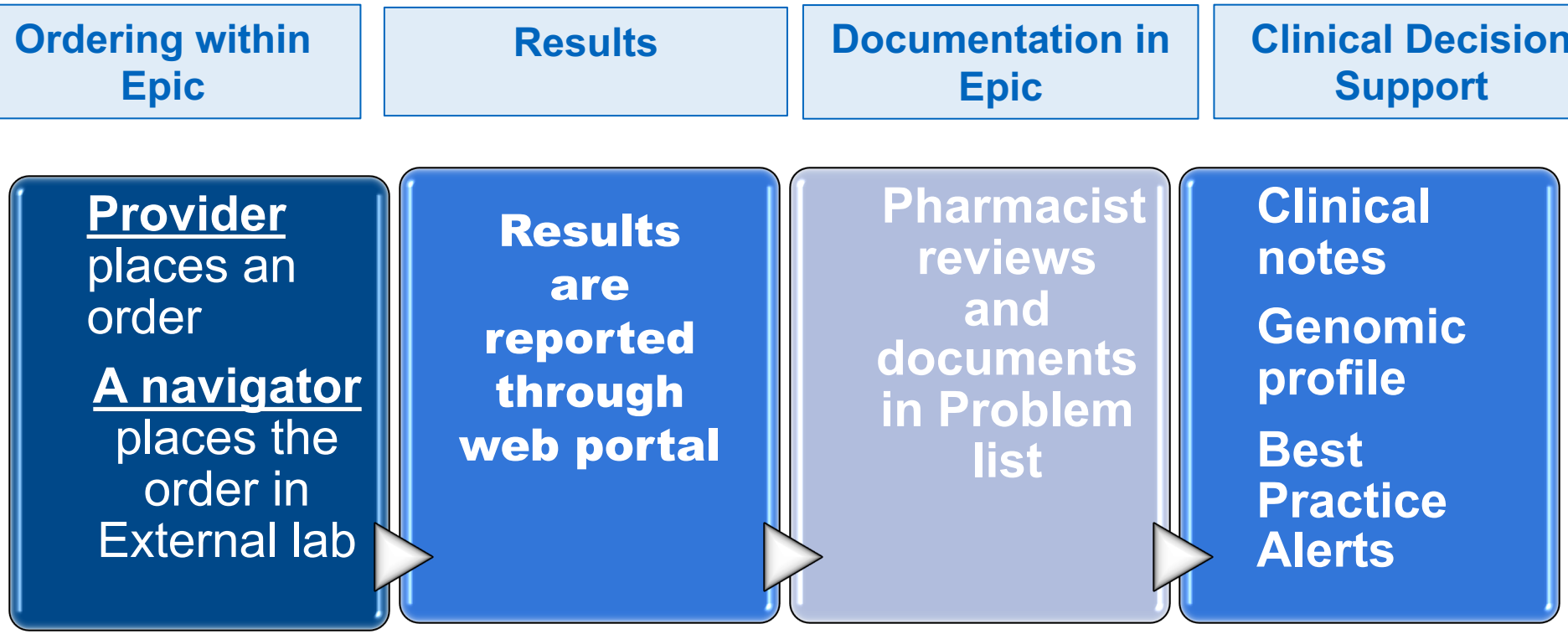
A hybrid of reactive and preemtive testing

PHARMACOGENETIC TEAM AND STRUCTURE

- Pharmacogenetic team
  - Two pharmacists
  - A genetic navigator
  - Rotating APPE students
  - Rotating residents

- Pharmacogenetic advisory committee
  - Discuss and prioritize implementation efforts
  - Representation from pharmacy, medical oncologist, PCPs

OUR PHARMACOGENETIC WORKFLOW



CLINICAL DECISION SUPPORT (NOTES AND ALERTS)

[redacted] a 61 y.o. male who has received pharmacogenomic testing to assist with oncology medication management. Through a re[redacted]of [redacted]ion therapy and genetic results, pharmacogenetic results could provide guidance on the patient's current therapy, and pote[redacted] inform future therapy.

Problem List

Patient Active Problem List

Diagnosis

- Open-angle glaucoma
- Nummular eczema
- Allergic rhinitis, seasonal
- Gastroesophageal reflux disease without esophagitis
- Rectosigmoid cancer (HCC)
- Malignant neoplasm of colon, unspecified
- CYP2D6 poor metabolizer (HCC)
- DPYD intermediate metabolizer (HCC)
- Monoallelic mutation of VKORC1 gene
- CYP2C19 intermediate metabolizer (HCC

Allergies

Allergies

No Known Allergies

Current Medications

Current Outpatient Medications

Medication

- acetaminophen (TYLENOL)
- ascorbic acid (vitamin C) (VITAMIN C)
- BIMATOPROST (LUMIGAN OPHT)
- calcium carbonate/vitamin D3 (VITAMIN D ORAL)
- dorzolamide-timolol (COSOPT) 22.3-6.8 Opht Drops
- multivitamin Oral Capsule
- pantoprazole (PROTONIX) 40 mg Oral Ta Delayed Release (E.C.)

Genetic Findings Pertinent to Current Medica

- DPYD: DPYD Intermediate Metabolizer A

Genetic Findings Pertinent to Future Medications:

- CYP2C19: CYP2C19 Intermediate Metabolizer
- CYP2D6: CYP2D6 Poor Metabolizer AS = 0

Assessment:

Medications Informed by Pharmacogenetics:

Anticipated start of FU as part of mFOLFOX
Pantoprazole 40 mg/d

[redacted] has pharmacogenomic results that affect current and future medication decisions. Based on patient's DPYD genetic results, patient is predicted to have a partial DPD deficiency and therefore, predicted to be **at an increased risk of severe or even fatal drug toxicity when treated with fluoropyrimidine drugs** (e.g. 5-Fluorouracil). We recommend reducing the starting dose by 50%, followed by dose titration based on toxicity. ***Dose can be increased in patients who do not experience toxicity in the first two cycles to maintain efficacy.*** Reduce the dose for patients who do not tolerate the starting dose to minimize toxicities; use therapeutic drug monitoring if available.

Please note that patient response to medications depend on multiple factors such as age, comorbid conditions, response history, etc. Use clinical judgement in addition to pharmacogenetic information for all medication decisions.

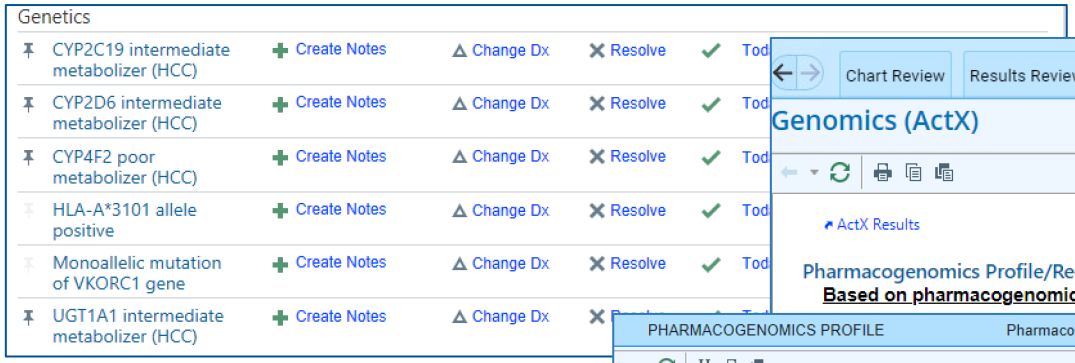
Other PGX Medications

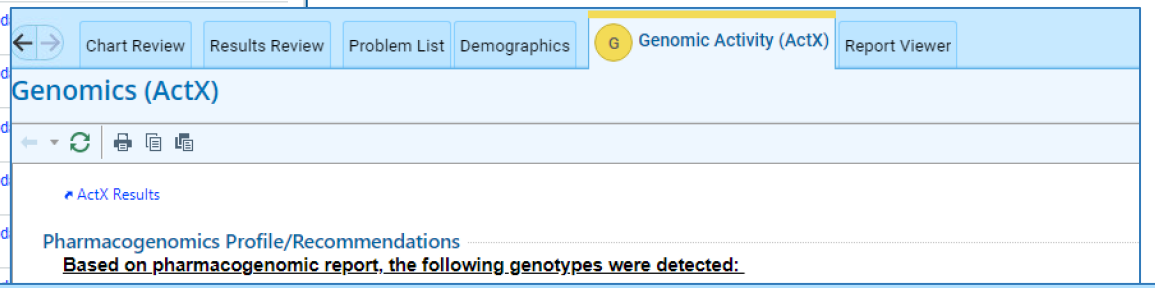
Pantoprazole: Patient is predicted to **have CYP2C19 Intermediate Metabolizer enzyme activity (reduced CYP2C19 enzyme activity) ,** which can lead to elevated drug levels of PPIs and increase the risk for PPI-related side effects (e.g. infections, electrolyte imbalance and kidney dysfunction). Patient is on 40 mg/d. For chronic therapy beyond 12 weeks, and after efficacy of acid suppression is achieved, may consider a 50% reduction in the daily dose , if appropriate, while monitoring for efficacy of pantoprazole.

Heavy Reliance on the CPIC content for clinical notes and BPAs

VISIBILITY OF PHARMACOGENETIC RESULTS

Problem list documentation and stand-alone pharmacogenetic tab





PHARMACOGENOMICS PROFILE Pharmacogenomics Profile

Alternative options:

Fluoxetine, OR escitalopram/citalopram, venlafaxine OR paroxetine are reasonable for GAD, if clinically appropriate. If any of these SSRIs is indicated, initiate therapy with the standard starting dose, while titrating to clinical response. Consider a slower titration and a lower maintenance dose with escitalopram and citalopram.

Although the patient may not be on the current medication therapy, we highlight these medications below to consider avoiding or using with caution if indicated in future therapy.

Future Medications:

Patient is predicted to be a CYP2C9 intermediate metabolizer, indicating that they will metabolize CYP2C9-metabolized medications more slowly. See below for general recommendations:

Consider dose reduction or select alternative medications for the following medications:
Fluvastatin: Prescribe < or = 40 mg per day as a starting dose and adjust doses of fluvastatin based on disease-specific guidelines. If dose > 40 mg needed for desired efficacy, consider an alternative statin or combination therapy (i.e., fluvastatin plus non-statin lipid-lowering therapy).

Patient is predicted to be a CYP2C19 intermediate metabolizer, indicating that they will metabolize CYP2C19-metabolized medications more slowly. See below for general recommendations:

Consider dose reduction, monitoring or select alternative medications for the following medications:

Tricyclic antidepressants (TCAs): amitriptyline, clomipramine, doxepin, imipramine, trimipramine
Selective serotonin reuptake inhibitors (SSRIs): citalopram, escitalopram, sertraline
Antifungals: voriconazole
Proton pump inhibitors (PPIs): pantoprazole, omeprazole, lansoprazole, dexlansoprazole

Select alternative medication:

Antiplatelets: Clopidogrel is a prodrug activated by CYP2C19. CYP2C19 intermediate metabolizers are less able to activate this medication, leading to increased risk of major adverse cardiovascular events. Alternative antiplatelet therapy (e.g. prasugrel, ticagrelor) is recommended.

Patient is a CYP3A5 Expressor, which can lead to lower dose-adjusted trough concentrations of tacrolimus with normal starting doses. If tacrolimus is indicated, we recommend increasing the starting dose by 1.5 to 2 times the recommended starting dose, though total starting dose should not exceed 0.3 mg/kg/day. We recommend therapeutic drug monitoring to guide dose further adjustments.

RELAYING INFORMATION TO THE PATIENT

Customized MyChart patient education explaining current and future PGX

Dear [redacted]

Your provider ordered pharmacogenetic testing to help better prescribe medications for you. These results were reviewed by the pharmacogenomic pharmacy team and our recommendations were sent to Dr. [redacted] Pharmacogenetic results could provide guidance on your current medications, and potentially impact future medication therapy.

- **We DO NOT recommend changes to your current medications at this time.**
- Your physician will decide if any adjustments are needed at this time. **DO NOT** stop or change the way you are currently taking your medications without first discussing with your doctor who prescribed the medication.

Your Pharmacogenomic Results:
Your results show that you have genetic variations in the following genes that could affect how your body breaks down certain medications:

- CYP2B6: Based on your genetic results, you are predicted to be a **CYP2B6 Intermediate Metabolizer**. This predicts that your body may break down certain medications at a slower rate than a patient who has a Normal Metabolizer status.
  - Per your records, you **ARE NOT** currently taking a medication that could be impacted by your CYP2B6 genetic results.
    - Other medications that may be impacted by this result include:
      - Antivirals: Efavirenz
- CYP2C19: Based on your genetic results, you are predicted to be a **CYP2C19 Intermediate Metabolizer**. This predicts that your body may break down certain medications at a very slow rate, causing too much of the medication to build up. The buildup of the medication is what may cause side effects.
  - Per your records, you **ARE** currently taking a medication (**omeprazole**) that could be impacted by these findings. **DO NOT** stop or change your medications without speaking to your doctor first. Your doctor will decide if a change in the medication or dose is needed.
    - Other medications that may be impacted by this result include:
      - Tricyclic antidepressants (TCAs): amitriptyline, clomipramine, doxepin, imipramine, trimipramine
      - Selective serotonin reuptake inhibitors (SSRIs): citalopram, escitalopram, sertraline
      - Antifungals: voriconazole
      - Proton pump inhibitors (PPIs): pantoprazole, omeprazole, lansoprazole, dexlansoprazole
    - Other drugs use CYP2C19 to activate the medication. Intermediate metabolizers will be less able to "turn on" these medications, making them less likely to work for you. The following medications may be impacted by this result:
      - Antiplatelets: Clopidogrel

FEASIBILITY AND UTILITY OF MULTI-PHARMACOGENETIC PANEL TESTING AMONG PATIENTS WITHIN PRIMARY CARE

St. Elizabeth HEALTHCARE

WHAT DO WE NEED? MORE ECONOMIC DATA

When do we need it? NOW!

PREEMPTIVE PHARMACOGENETIC TESTING: EXPLORING KNOWLEDGE AND PERSPECTIVES OF UNITED STATES PA

Nicholas J. Keeling, MS[1,2], Meagen M. Rosenthal, PhD[1], Donna West-Strum, RPh, Pl Amit Patel, PhD[1,3], Cyrine E. Haidar, PharmD[2], James M. Hoffman, PharmD, MS[2]

[1]Department of Pharmacy Administration, University of Mississippi School of Pharmacy, University, MS, USA

[2]Department of Pharmaceutical Sciences, St. Jude Children's Research Hospital, Memph USA

[3]Medical Marketing Economics, LLC, Oxford, MS, USA

Abstract

PURPOSE—Preemptive pharmacogenetic testing aims to optimize medication use by having genetic information at the point of prescribing. Payers' decisions influence implementation of technology. We investigated U.S. payers' knowledge, awareness, and perspectives on preempti pharmacogenetic testing.

METHODS—A qualitative study was conducted using semi-structured interviews. Participant were screened for eligibility through an online survey. A blended inductive and deductive approach was used to analyze the transcripts. Two authors conducted an iterative reading proc to code and categorize the data.

RESULTS—Medical or pharmacy directors from 14 payer organizations covering 122 millio U.S. lives were interviewed. Three concept domains and ten dimensions were developed. Key findings include: clinical utility concerns and limited exposure to preemptive germline testing, continued preference for outcomes from randomized controlled trials, interest in guideline development, importance of demonstrating an impact on clinical decision making, concerns of downstream costs and benefit predictability, and the impact of public stakeholders such as the FDA and CMS.

CONCLUSION—Both barriers and potential facilitators exist to developing cohesive reimbursement policy for pharmacogenetics, and there are unique challenges for the preemptive testing model. Prospective outcome studies, more precisely defining target populations, and predictive economic models are important considerations for future research.

Cost Effectiveness of Pharmacogenetic Testing for Drugs with Clinical Pharmacogenetics Implementation Consortium (CPIC) Guidelines: A Systematic Review

Sarah A. Morris[1], Ashraf T. Alsaidi[2], Allison Verbyla[3], Adilen Cruz[3], Casey Macfarlane[2], Joseph Bauer[3] and Jai N. Patel[1,*]

The objective of this study was to evaluate the evidence on cost-effectiveness of pharmacogenetic (PGx)–guided treatment for drugs with Clinical Pharmacogenetics Implementation Consortium (CPIC) guidelines. A systematic review was conducted using multiple biomedical literature databases from inception to June 2021. Full articles comparing PGx-guided with nonguided treatment were included for data extraction. Quality of Health Economic Studies (QHES) was used to assess robustness of each study (0–100). Data are reported using descriptive statistics. Of 108 studies evaluating 39 drugs, 77 (71%) showed PGx testing was cost-effective (CE) (N = 48) or cost-saving (CS) (N = 29); 21 (20%) were not CE; 10 (9%) were uncertain. Clopidogrel had the most articles (N = 23), of which 22 demonstrated CE or CS, followed by warfarin (N = 16), of which 7 demonstrated CE or CS. Of 26 studies evaluating human leukocyte antigen (HLA) testing for abacavir (N = 8), allopurinol (N = 10), or carbamazepine/phenytoin (N = 8), 15 demonstrated CE or CS. Nine of 11 antidepressant articles demonstrated CE or CS. The median QHES score reflected high-quality studies (91; range 48–100). Most studies evaluating cost-effectiveness favored PGx testing. Limited data exist on cost-effectiveness of preemptive and multigene testing across disease states.

PMID: 36149409, PMID: 31048813

METHODS

- A Multi-collaborative, pilot study with a payer system and pharmacogenetic lab
- Aims: Investigate the utility and economic value among St Elizabeth patients
- Outreach to patients with a recently prescribed pharmacogenetic medication
  - Outreach was made to ~ 6000 patients from PCP
- Post pharmacogenetics testing
  - Consult notes to their PCPs
  - MyChart messages to the patients

METHODS

Collected data on:

- Number and percentage:
  - Patients with intervention, MyChart messages, physicians outreached

- Prevalence of medications with pharmacogenetic guidelines:
  - Statins, opioids, clopidogrel, SSRIs, venlafaxine, NSAIDs, PPIs, metoprolol, ondansetron, warfarin, allopurinol, TCAs, antipsychotics, atomoxetine, phenytoin, thiopurine, cancer meds

- Prevalence of actionable gene-drug interactions:
  - Patients with an interacting genotype and medications (e.g. CYP2D6 PM/opioids)

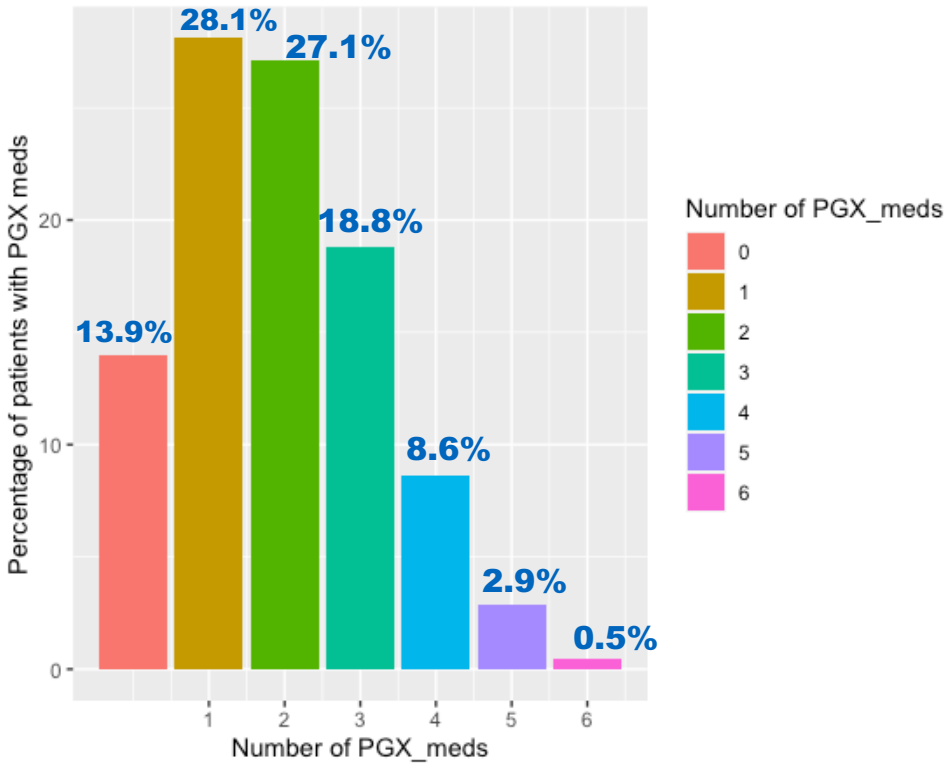
POST PHARMACOGENETIC TESTING INTERVENTIONS AND OUTREACH

Patient	Interventions	MyChart messages	Physicians out reached
1043	402 (38.5 %)	952 (91.2%)	258

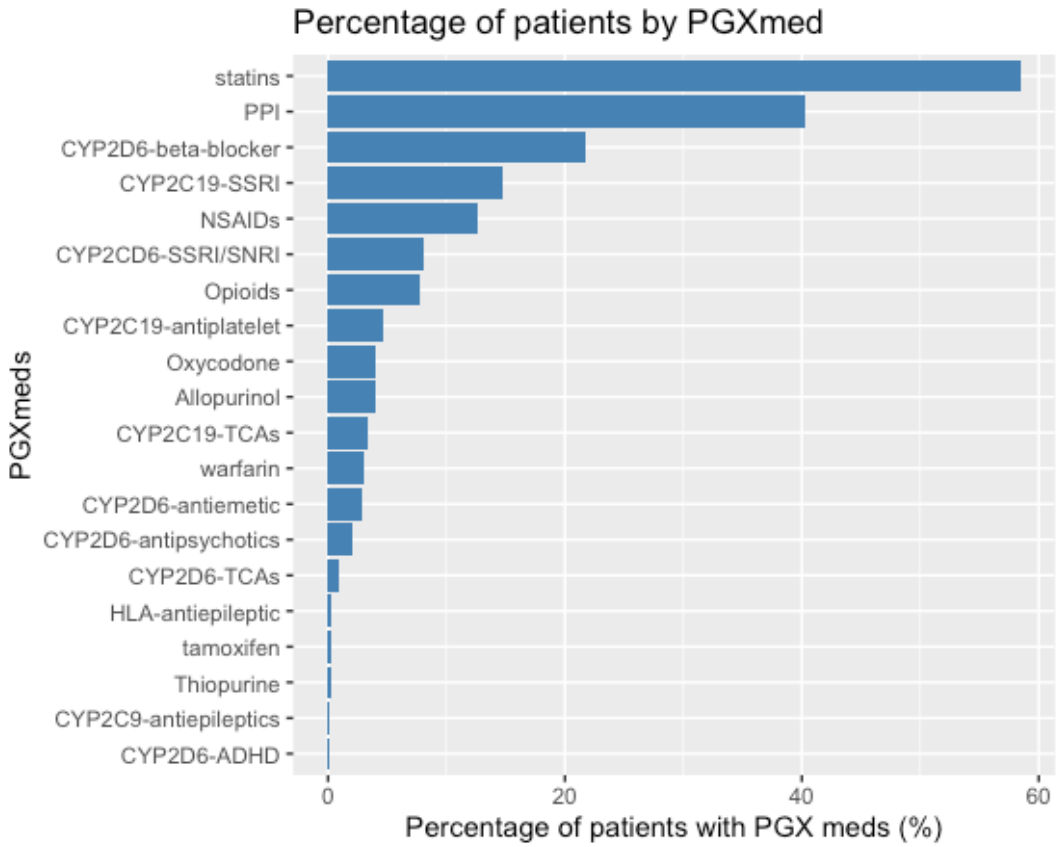
Intervention: dose increase; dose decrease; switch to alternative; discontinue

PREVALENCE OF PHARMACOGENETIC MEDICATIONS

~ 86% have one or more pharmacogenetic medications

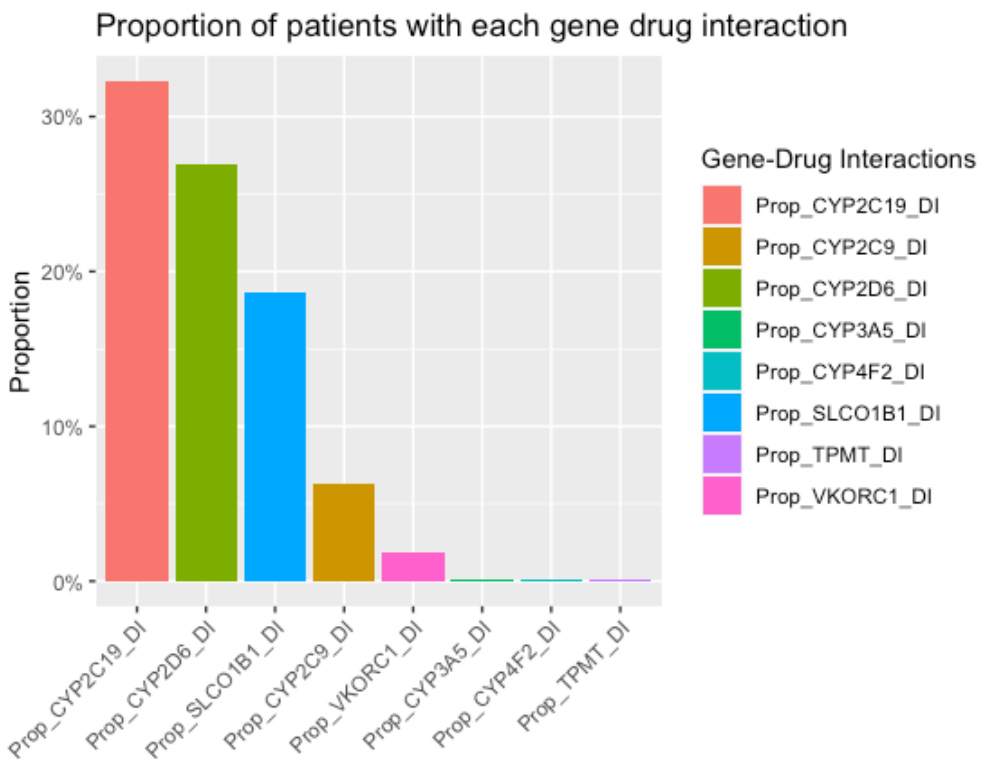


Statins, PPIs, metoprolol, SSRIs and NSAIDs are the most common PGX meds

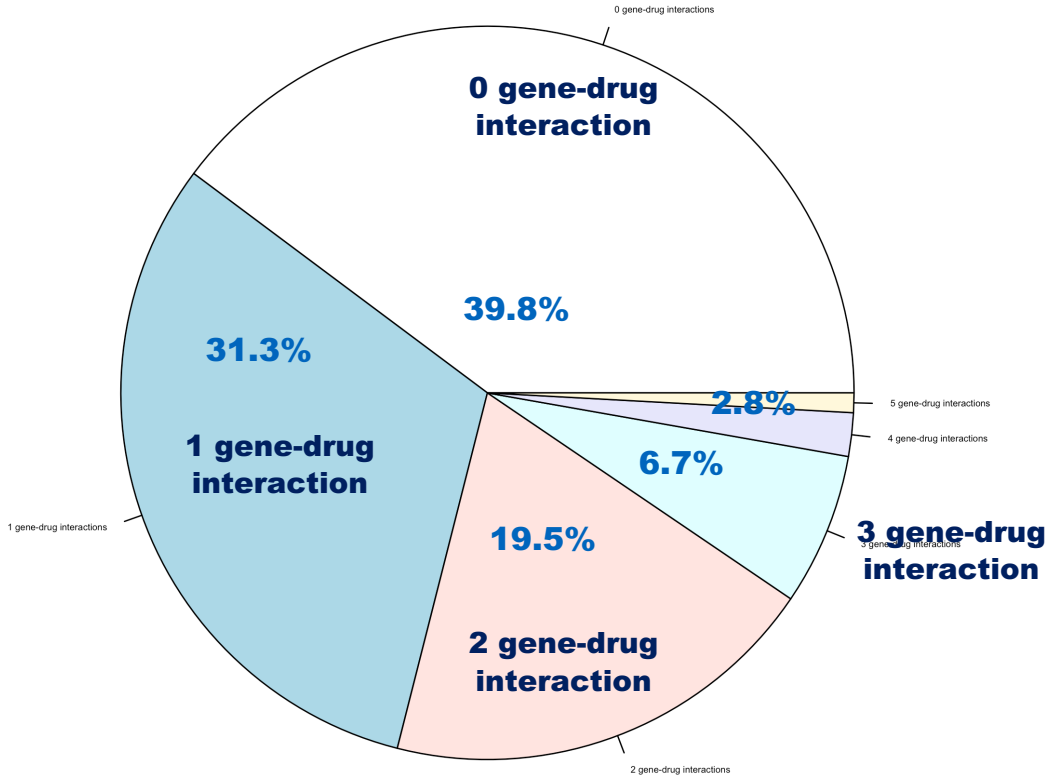


PREVALENCE OF GENE-DRUG INTERACTIONS

Interactions related to CYP2C19; CYP2D6, and SLCO1B1 were the most common



~ 60% have one or more gene-drug interaction



SUMMARY AND NEXT STEPS

Summary

- Multi-gene panel, PGx testing of patients within Primary care is feasible
- PGx meds are prevalent; 86% patients had at least one PGx med
- Sixty percent of patients had one or more gene-drug interactions with the most common interactions related to *CYP2C19; CYP2D6; and SLCO1B1* genes

Next steps

- Complete data collection on the uptake of interventions
- Providers' survey to understand the needs and improve the service
- Economic analysis
  - Medical and prescription claims

PGX TESTING TO GUIDE FLUOROPYRIMIDINE AND IRINOTECAN

St. Elizabeth HEALTHCARE

DPYD screening could save Human lives!

comments and controversies

DPYD Testing: Time to Put Patient Safety First

Sharyn D. Baker, PharmD, PhD[1]; Susan E. Bates, MD[2]; Gabriel A. Brooks, MD[3]; William L. Dahut, MD[4]; Robert B. Diasio, MD[5]; Wafik S. El-Deiry, MD, PhD[6]; William E. Evans, PharmD[7]; William D. Figg, PharmD, MBA[8]; Dan L. Hertz, PharmD, PhD[9]; J. Kevin Hicks, PharmD, PhD[10]; Suneel Kamath, MD[11]; Pashtoon Murtaza Kasi, MD[12]; Todd C. Knepper, PharmD[10]; Howard L. McLeod, PharmD[13]; Peter H. O'Donnell, MD[14]; Mary V. Relling, PharmD[7]; Michelle A. Rudek, PharmD, PhD[15]; Tristan M. Sissung, PhD[8]; D. Max Smith, PharmD[16]; Alex Sparreboom, PhD[1]; Sandra M. Swain, MD[16]; and Christine M. Walko, PharmD[10]

In 2018, a patient received capecitabine without prior testing for dihydropyrimidine dehydrogenase (*DPYD*) and later presented with vomiting, rash, and diarrhea. The hospital failed to provide uridine triacetate in a timely fashion, and the patient died. The patient's widow filed a wrongful death lawsuit against Oregon Health Sciences University (OHSU) and assisted in the formation of a nonprofit organization to advocate for *DPYD* testing for fluoropyrimidines. A settlement for $1 million US dollars was reached requiring OHSU oncologists to undergo education about *DPYD* testing and inform their patients about its availability.[1] Clinical variants and fluoropyrimidine pharmacokinetics.[10] This commentary will establish that pretreatment *DPYD* testing is well justified and recommend dose reduction in those patients with a decreased function variant. We recommend an immediate modification to the oncology treatment guidelines that include a fluoropyrimidine.

Severe Fluoropyrimidine-Associated Toxicity

A recent meta-analysis of 13,929 patients in 35 studies found that patients carrying *DPYD*2A* were much more likely to experience severe life-threatening toxicity from fluoropyrimidine therapy than those carrying only wild-

ve attitude for their three kids, the youngest of whom was 2. Kerrie's and vitality would carry her through chemo's side effects, they hoped.

Kerrie Prettitore, center, with her husband, Glenn, and children Liam, Maeve and Fiona. The Ridgewood woman had a rare

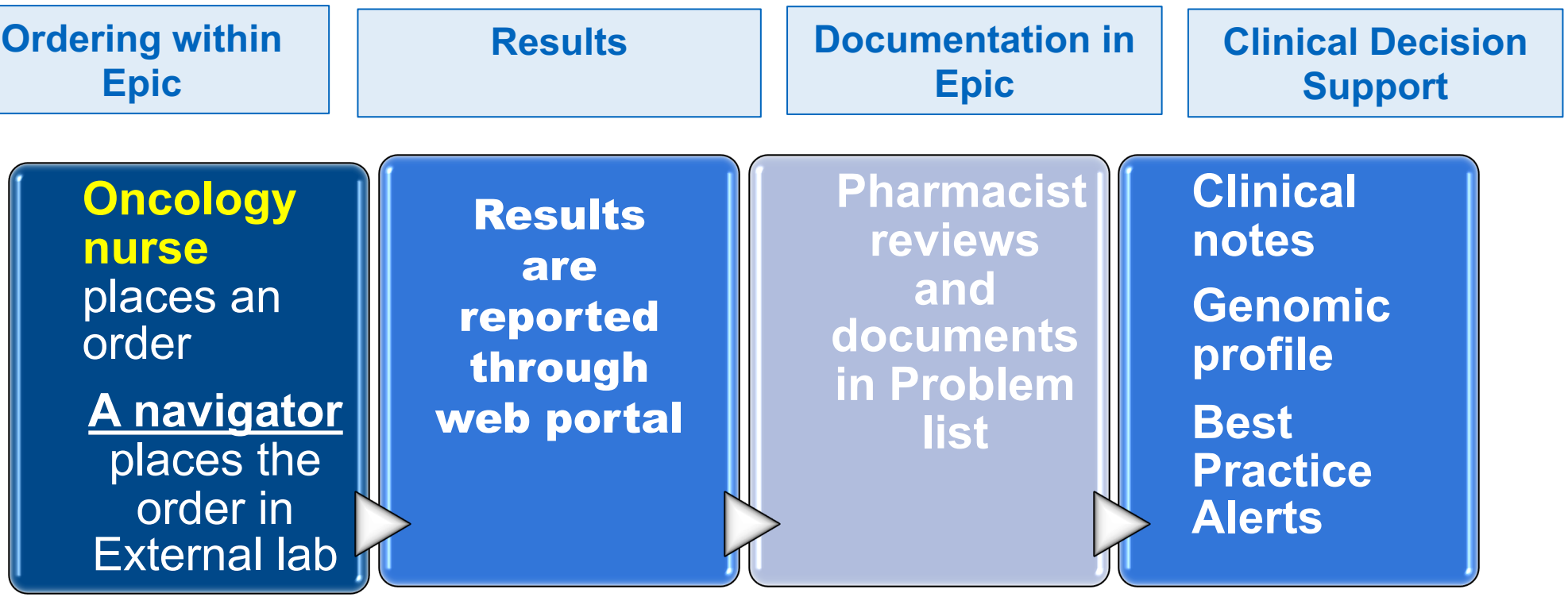
OHSU to pay $1 million, promises change to settle lawsuit from widow of cancer patient

ıted: May. 04, 2022, 11:05 a.m. | Published: May. 04, 2022, 7:00 a.m.

yre had a fatal reaction to OHSU's chemotherapy for his cancer, his ıims in a lawsuit filed against the university.

PMID: 36821823

SIMILAR PHARMACOGENETIC WORKFLOW



OBJECTIVE

Demonstrate the feasibility of testing and utility within oncology

An Exploratory analysis of disease progression for patients with *DPYD*-guided dose reduction

METHODS

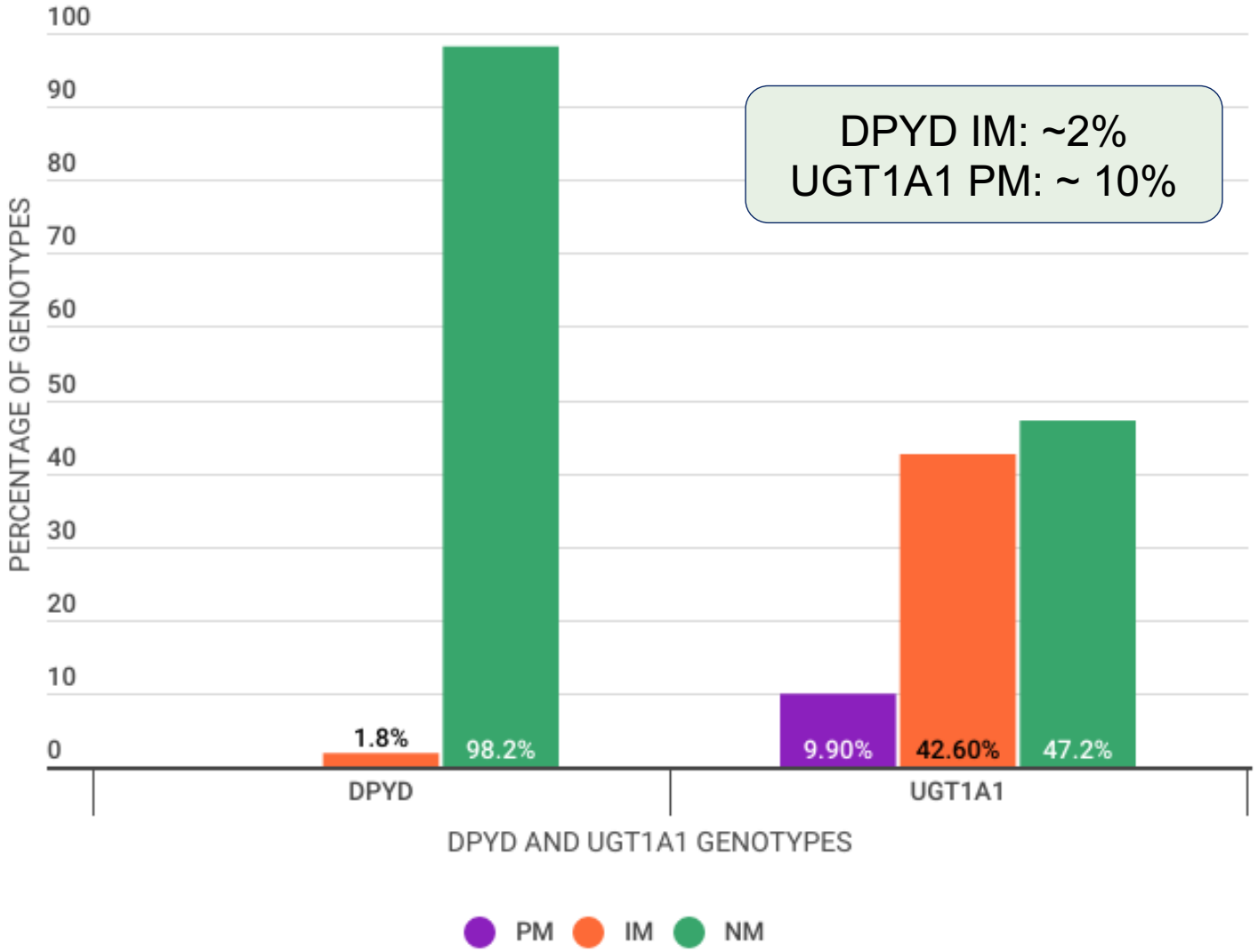
Collected data on the number and percentage:

- Prevalence of actionable UGT1A1 (PM) *and* DPYD (IM, PM)

- Pharmacogenetic results returned, and interventions provided before the start of chemotherapy

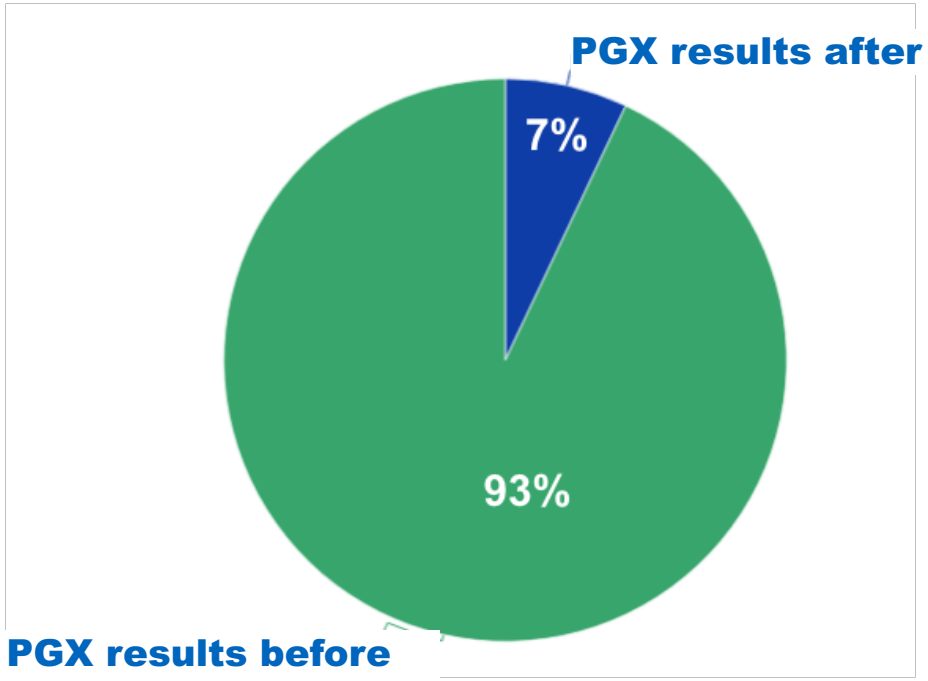
Megan Muldoon

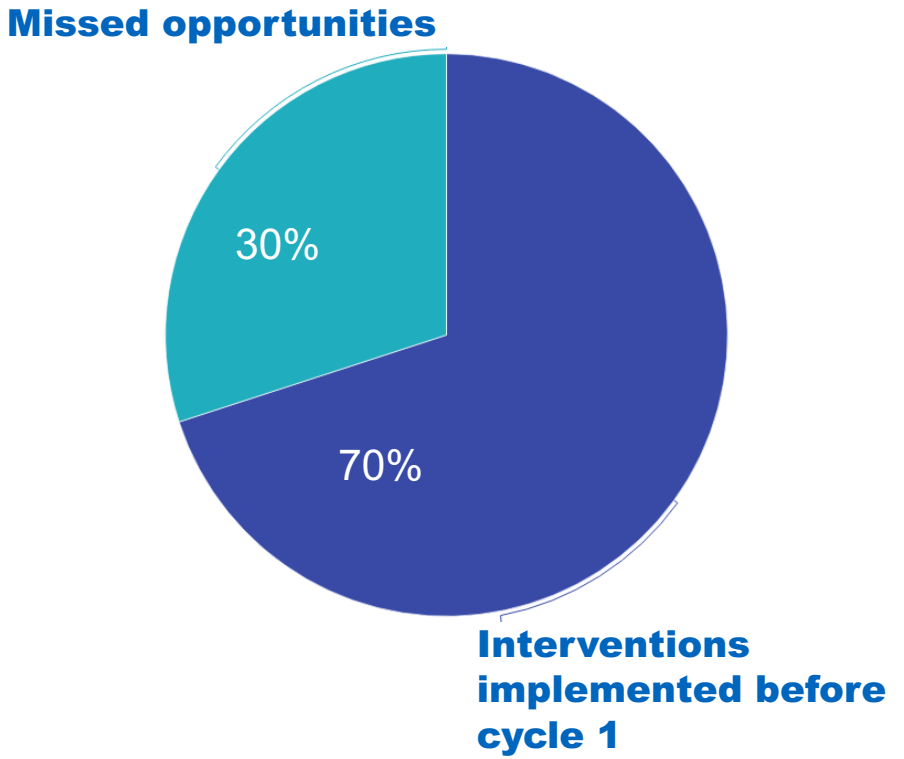
PREVALENCE OF ACTIONABLE DPYD AND UGT1A1 (N=282)



DPYD IM: ~2%
UGT1A1 PM: ~ 10%

CHEMOTHERAPY PHARMACOGENETIC TESTING IS FEASIBLE





- The Average and standard deviation of results turn-around time were 5.8 and 15.8 days, respectively

- Approximately four percent of patients (N=10/282) had DPYD Intermediate Metabolizer or UGT1A1 Poor Metabolizer and received a chemotherapy that required a dose reduction

CANCER PROGRESSION IN CURATIVE SETTING

Pt	Age	Cancer staging	Fluoropyrimidine base	Recommendation implemented	Treatment completed	Follow up (days)	Progression
1	74	IIIB colon	Capecitabine (CapOx)	Yes	Yes	104	No
2	46	stage III, sigmoid	Capecitabine (CapOx)	Yes	Yes	326	No
3	62	Stage IIIB recto-sigmoid	5 FU (mFOLFOX)	Yes	Ongoing	Ongoing	No
4	65	Stage III, pancreas	5-FU (FOLFIRINOX) then capecitabine/radiation	Yes	Ongoing	Ongoing	No

SUMMARY OF PGX IMPLEMENTATION IN ONCOLOGY

Summary

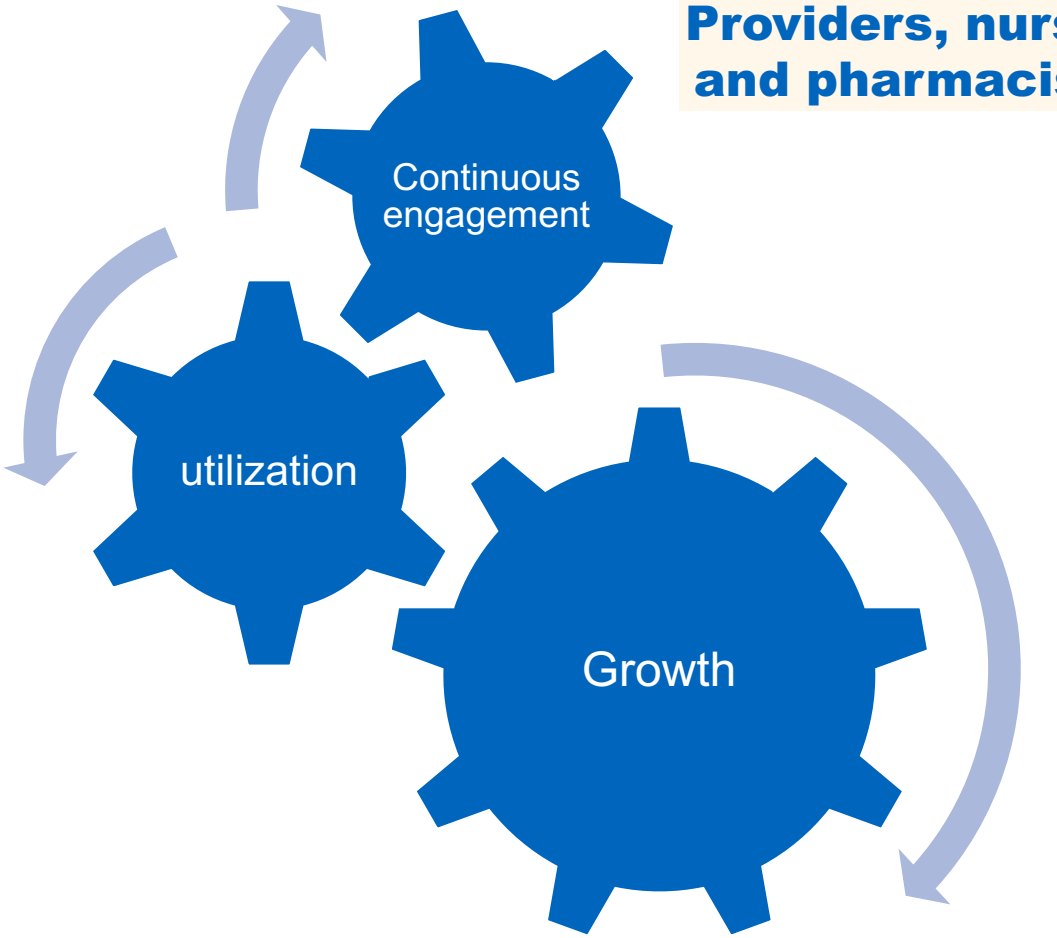
- Pharmacogenetic testing is feasible with majority of results returning before the start of treatment
- Interventions were accepted by providers with few missed opportunities for UGT1A1-guided interventions

Next steps

- Demonstrate that pharmacogenetic testing does not compromise effectiveness
- Education opportunities for UGT1A1-guided irinotecan and supportive care medications
- Demonstrate utility for other supportive care medications

Monitoring Progress and Addressing gaps

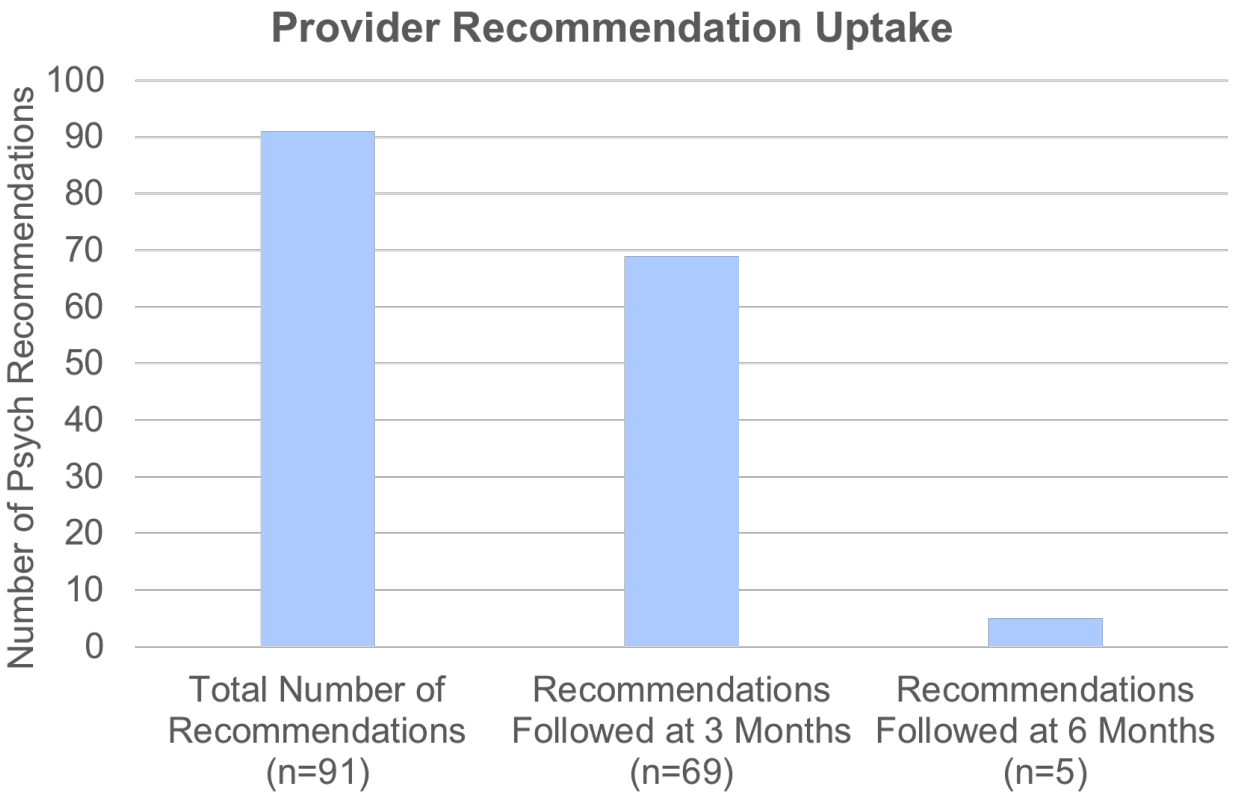
Providers, nurses and pharmacists

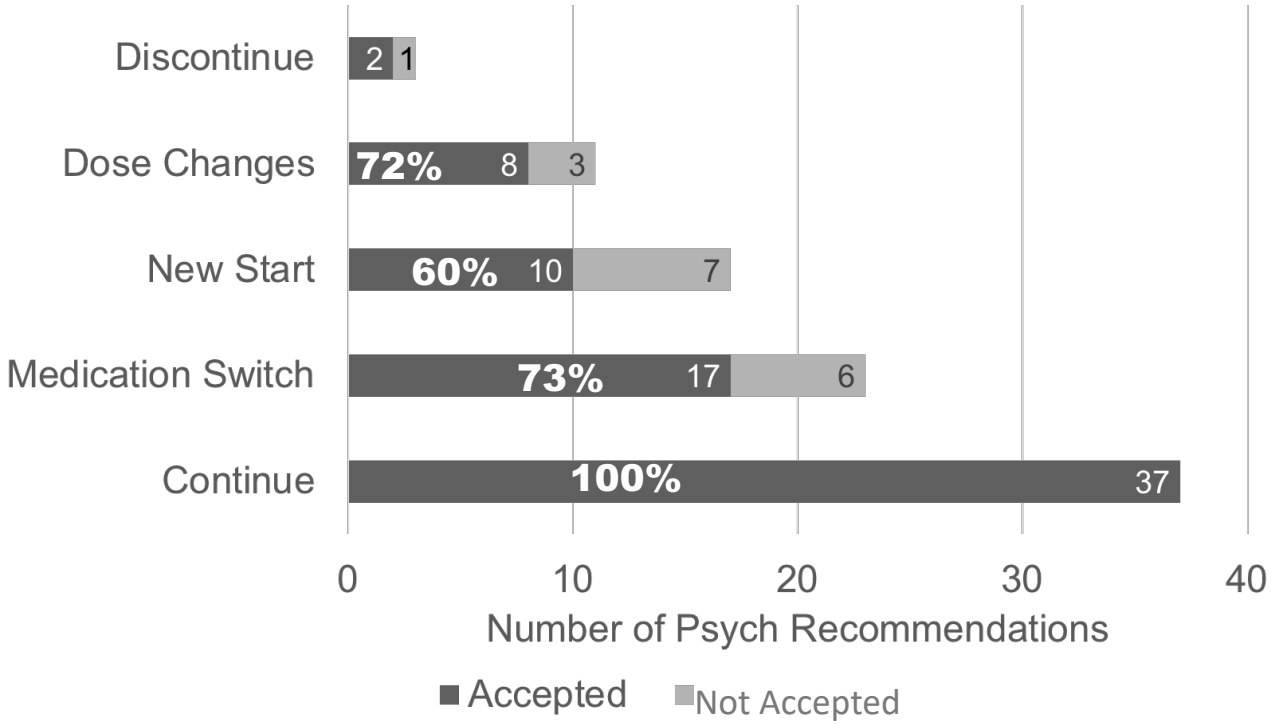


Assessing uptake of notes and alerts

FOLLOWING UP ON INTERVENTIONS

Preliminary data on uptake of recommendations within notes

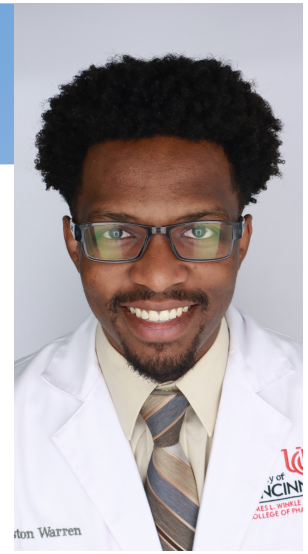




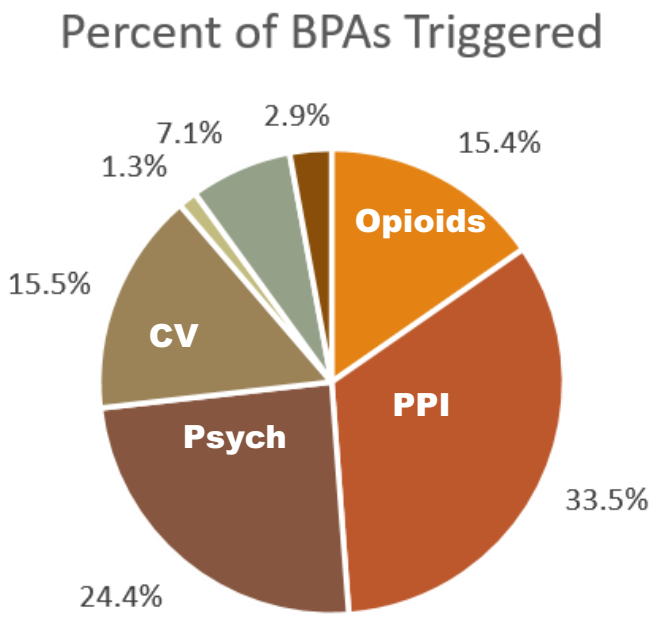
75.8% of recommendations accepted at 3 months
5.5% of recommendations accepted at 6 months

Abigle Adjei, PharmD capstone 2023

ASSESSING BPA ALERTS



Treston warren



- Many alerts are related to "monitoring"
- Alert fatigue
- Compromising the actionable ones

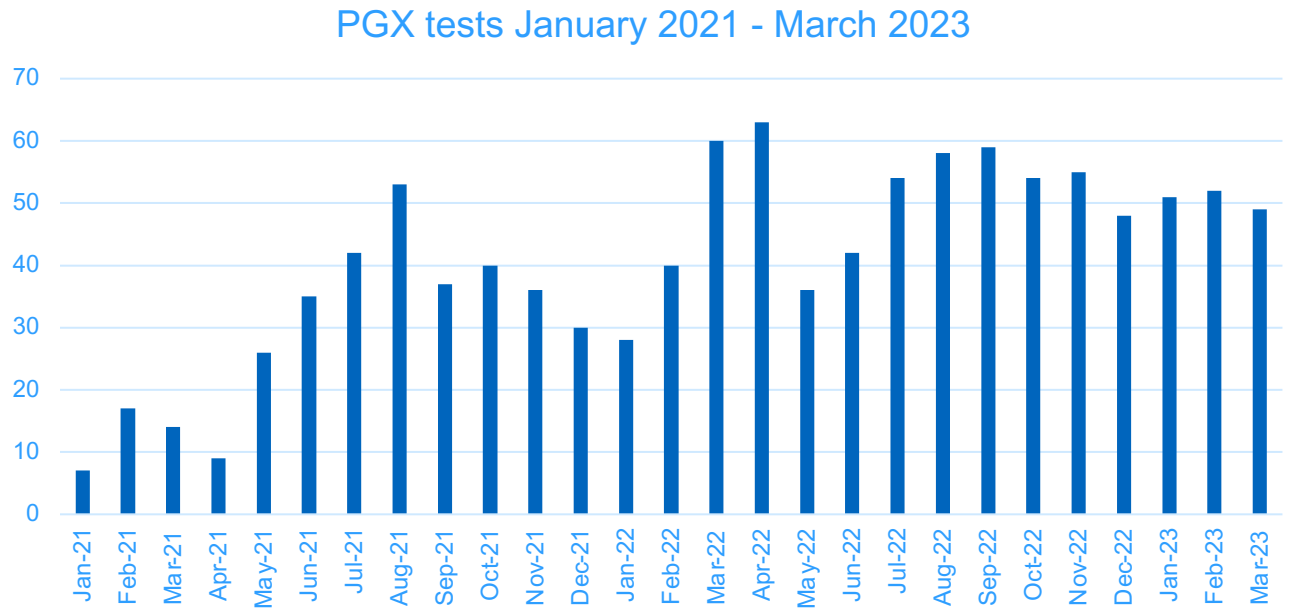
Opioids PPIs Psychological Cardiovascular Oncology NSAID Other

Class	PPIs	Opioids	Psychiatry	Cardiovascular	Oncology	NSAIDs	Other
No BPA (%)	881/2630 (33.5%)	404/2630 (15.4 %)	641/2630 (24.3%)	408/2630 (15.5%)	35/2630 (1.3%)	186/2630 (7.1%)	75/2630 (2.9%)
No patients	332	140	253	162	10	90	27

MONITORING GROWTH AND PROVIDER ENGAGEMENT

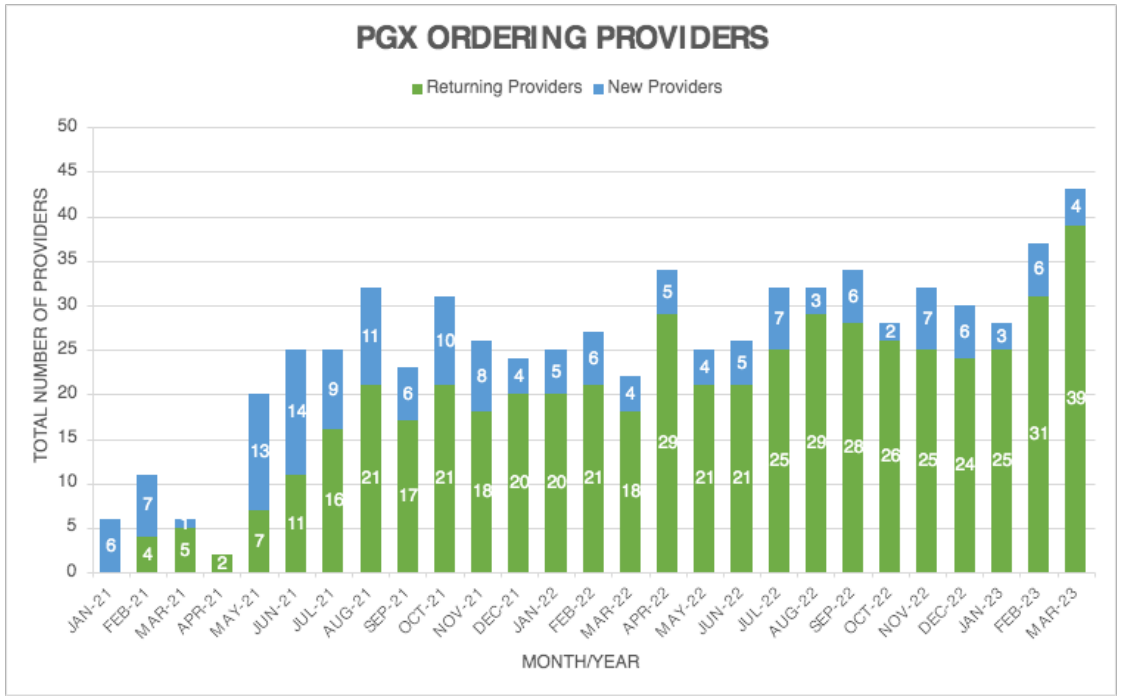
Grace Miller

Completed clinical PGX tests



Completed tests
1,095

Average per month
41



IDENTIFIED AREAS OF IMPROVEMENT

Provider education

- Triggered by:
  - Increase in the volume
  - Non acting on BPAs

Response

- System-wide educational seminars

Pharmacist education

- Triggered by:
  - Increase in the volume
  - Needs to sustain the program

Response

- Grand rounds seminars
- Continuing education

Informatics

Triggered by:

- Increase in the volume
- Non acting on BPAs
- Streamline the workflow

Response:

- Modifying alerts
- Investigating Epic genomics module

SUMMARY

- Explained the structure of the pharmacogenetic implementation program at St Elizabeth
- Demonstrated a high prevalence of pharmacogenetic medications and gene-drug interactions in Primary Care, justifying a multi-gene, panel-based approach
- Demonstrated that pharmacogenetic testing is feasible, with efforts spanning PCP and oncology
- Identified areas for sustained growth of our implementation including informatics support and education

ACKNOWLEDGEMENT

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Nurse Champion

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St. Elizabeth HEALTHCARE