We must start using pharmacogenomic information to optimise medicines for older people

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Training in pharmacogenomic testing needs to be prioritised so that the NHS can benefit from the many advantages it has to offer, especially for older people at higher risk from adverse drug reactions.

Pharmacogenetics and pharmacogenomics

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By **Victoria David**, **Ciarán McInerney**, **Beth Fylan**, **Eleanor Bryant**, **Gurdeep Sagoo**, **V-Lin Cheong**, **Marcus Rattray** & **Heather Smith**

Corresponding author **Victoria David**

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In the UK, the proportion of older adults in the population is rising steadily. It is projected that, by 2050, one in four people will be aged 65 years and over, compared with one in five in 2018[1]. And, with an ageing population comes an increase in the prevalence of multimorbidities and polypharmacy[2].

Polypharmacy, commonly defined as the regular use of five or more medicines concomitantly, is associated with an increased likelihood of significant negative health outcomes[3–5]. Older people are at increased risk of adverse drug reactions (ADRs) and one in five hospital admissions in patients aged over 65 years is attributed to ADRs[6].

Risk factors for ADRs — such as age, renal function and comorbidities — are often considered by prescribers in medication reviews, but much more can be done to reduce the risk of harm and improve the efficacy of medicines. In particular, there is a growing body of evidence to support the use of pharmacogenomic testing to predict an individual's response to many commonly used medicines and this could be an extremely useful tool when it comes to optimising treatment and reducing the risk of ADRs in older people[7].

Actionable variants

There have been rapid advances in the development of genotyping technologies that can quickly identify individual variations in key genes that affect a person's ability to metabolise, or respond to, certain medicines[8].

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It is estimated that more than 90% of individuals carry at least one genetic variant that is 'actionable' and could help inform a prescribing decision[9–11]. The results from pharmacogenomic tests could therefore enable prescribers to ensure that the right medicine is given at the right dose to the right patient.

The NHS is committed to embedding pharmacogenomic testing into routine practice by 2025, and the available evidence suggests that incorporating pharmacogenomic testing into the care of older people specifically will reduce ADRs and unplanned hospital admissions[12,13].

Our research

Comprising pharmacists and academics from the Medicines Optimisation Research Group at the University of Bradford, in collaboration with Leeds Teaching Hospitals NHS Trust (LTHT) and the University of Leeds, our research group is actively investigating how pharmacogenomic testing could be put into practice in the NHS.

The group is currently completing a retrospective cross-sectional study of 59,973 hospital admissions of people aged 65 years and over to LTHT throughout 2018 and 2019 and the 560,163 medicines that were prescribed to them.

As expected, polypharmacy was high, with 83% of patients taking five or more medicines and 43% taking ten or more medicines. Our analysis also showed that over one in five (22%) medicines being taken on admission had known pharmacogenomic associations, with 63 different medicines with known pharmacogenomic associations being used.

> Many of the medicines identified as having pharmacogenomic links are also considered 'high risk' regarding their potential to contribute to unplanned hospital admissions

On average, each patient was taking nine medicines, two of which were actionable from a pharmacogenomics perspective.

Many of the medicines identified as having pharmacogenomic links are also considered 'high risk' regarding their potential to contribute to unplanned hospital admissions[14]. These include antiplatelets, anticoagulants, analgesics and antidepressants (see Table 1 [15]). The Clinical Pharmacogenetics Implementation Consortium (CPIC) evaluates the level of existing clinical and research evidence for drug–gene pairs; those at level A or level B have potential prescribing recommendations[15].

Drug	Class	Frequency of occurrence (%)	CPIC evidence level	Gene pair(s)
Amitriptyline	Antidepressant	0.84	Level A	CYPD6, CYP2C19
Citalopram	Antidepressant	0.48	Level A	CYP2C19
Clopidogrel	Antiplatelet	1.39	Level A	CYP2C19
Codeine	Opioid analgesic	1.85	Level A	CYP2D6
Escitalopram	Antidepressant	0.03	Level A	CYP2C19
Ibuprofen	Non-steroidal anti-inflammatory drug	0.75	Level A	CYP2C9
Paroxetine	Antidepressant	0.07	Level A	CYP2D6
Sertraline	Antidepressant	0.60	Level A	CYP2C19
Tramadol	Opioid analgesic	0.41	Level A	CYP2D6
Venlafaxine	Antidepressant	0.16	Level A/B	CYP2D6
Warfarin	Anticoagulant	1.03	Level A	CYP4F2, VKORC1, CYP2C19

Table: Some high-risk medicines prescribed to older people that have a pharmacogenomic association defined by Clinical Pharmacogenetics Implementation Consortium guidelines

In the following sections, we will look at three of the most prevalent medicine groups in our sample and assess the evidence for intervention based on pharmacogenomic testing.

Antiplatelets and anticoagulants

Prescriptions for clopidogrel and warfarin accounted for between 1.0% and 1.4% (5,746–7,767) of all prescriptions for patients aged 65 years and over who were admitted to LTHT. Both of these medicines have actionable pharmacogenomic associations with strong CPIC recommendations to change prescribing[16,17].

For patients initiated on clopidogrel, a change in medication — to ticagrelor or prasugrel, for example — is recommended for poor and intermediate metabolisers of clopidogrel to improve the efficacy of antiplatelet therapy[18,19]. Point-of-care tests to identify the genetic variants that affect clopidogrel metabolism have already been developed and have a 55-minute turnaround time, meaning that results can be acted upon during inpatient admission and outpatient review, potentially in primary care and community pharmacy settings[20–26].

> Like blood glucose monitoring, point-of-care testing can also be carried out by healthcare professionals without formal laboratory training

Like blood glucose monitoring, point-of care testing can also be carried out by healthcare professionals without formal laboratory training. Implementing pharmacogenomic testing for the 4–30% of patients initiated on clopidogrel who do not derive therapeutic benefit owing to the loss-of-function gene variant they carry, could be easily incorporated into patient care pathways, although the practical application, cost and clinical utility would need to be assessed first[27].

For warfarin, dose adjustment rather than medication switching is recommended for patients with certain genetic variants that result in ADRs or poor drug efficacy[16] . However, recently there has been a rapid decline in warfarin initiation owing to a move towards direct oral anticoagulants, which may reduce the need for routine pharmacogenomic testing in this cohort[28]. It continues to be used for some indications, such as for patients with mechanical heart valves; in these cases, prospective pharmacogenomic testing may be helpful to define the dose range.

High-risk analgesics

Pain management in older people can be complicated by age-related ADRs as well as polypharmacy and multimorbidity[3]. Between 0.4% to 1.9% (2,294 to 10,369) of all prescriptions in the study sample contained codeine, tramadol or ibuprofen. These have pharmacogenomic associations and are identified as ‘high risk’ for causing medicine-related hospital admissions.

Codeine and tramadol are opioid analgesics used to treat mild-to-moderate pain and moderate-to-severe pain, respectively. Both are metabolised to their active metabolites by cytochrome P450 2D6 (CYP2D6) enzymes, encoded by the highly polymorphic CYP2D6 gene. Globally, up to 7% of individuals are poor metabolisers of codeine and tramadol, leading to reduced efficacy, while 2% are ultra-rapid metabolisers, leading to an increased risk of opiate toxicity[8,29–31]. As a result, there are strong recommendations to avoid codeine/tramadol use in ultra-rapid and poor metabolisers to help avoid these outcomes [8].

There is evidence that pharmacogenomic information could be used to alter opioid prescribing behaviours and as an additional tool to optimise medicines while considering other risk factors, such as drug–drug interactions that also contribute to drug response [32].

After aspirin and diclofenac, ibuprofen is the third most common non-steroidal anti-inflammatory drug (NSAID) to cause medicine-related hospital admission owing to its gastrointestinal effects, such as bleeding and peptic ulceration, as well as renal impairment[14]. Up to 4% of individuals globally are CYP2C9 poor metabolisers, resulting in higher plasma concentrations of ibuprofen and an increased risk of severe toxicity [29,33]. However, there is currently no drug labelling to highlight this pharmacogenomic association and only moderate recommendations for dose adjustments, primarily owing to

the lack of consensus on the clinical evidence supporting pharmacogenomic-guided prescribing recommendations[34]. Concomitant use of ibuprofen with other CYP2C9 substrates, such as warfarin, can increase the risk for ADRs, such as bleeding.

High-risk antidepressants

Just under 1% (4,698) of all prescriptions in the study sample were for antidepressants amitriptyline, venlafaxine, paroxetine, sertraline, citalopram and escitalopram. Patients are susceptible to antidepressant ADRs owing to the trial-and-error approach to prescribing for depression[35,36]. However, this could be mitigated by pharmacogenomic testing to predict a patient's response to medicines. All of these antidepressants interact with CYP2D6 or CYP2C19 gene variants. Between 1–15% and 1–20% of patients are poor metabolisers and ultra-rapid metabolisers of tricyclic antidepressants, respectively [37]. For selective serotonin reuptake inhibitors (SSRIs), about 2–15% and 5–30% of patients are poor metabolisers and ultra-rapid metabolisers, respectively[38]. Unlike SSRIs and tricyclic antidepressants, venlafaxine does not have pharmacogenomic prescribing guidelines.

> Between 1–15% and 1–20% of patients are poor metabolisers and ultra-rapid metabolisers of tricyclic antidepressants, respectively

Amitriptyline is a tricyclic antidepressant and the most common antidepressant in the study sample. It is used as an alternative medicine for severe chronic depressive symptoms when patients have been unresponsive to SSRIs[39]. Amitriptyline has numerous ADRs, such as anticholinergic syndrome and drowsiness that can lead to falls, with older people at an increased risk owing to increased prevalence of polypharmacy, frailty and cognitive decline[40]. In this drug–gene interaction, variants of the CYP2D6 gene affect the clearance of amitriptyline. As a result, there are strong recommendations to avoid its use in CYP2D6 poor metabolisers owing to the increased risk of ADRs and to consider alternative medication in CYP2D6 ultra-rapid metabolisers owing to reduced drug efficacy and the potential for therapeutic failure[37]. Amitriptyline is mostly used for neuropathic pain, and pharmacogenomic-based dose changes are not currently recommended for this indication since the drug is used at a lower dosage, meaning patients are less likely to experience ADRs owing to poor drug clearance. There is also insufficient data describing the use of amitriptyline for neuropathic pain in patients that are CYP2D6 ultra-rapid metabolisers[37].

SSRIs present in the dataset included paroxetine, sertraline, citalopram and escitalopram. Apart from escitalopram, these SSRIs can be used in first episode depression[39]. Paroxetine is extensively metabolised by CYP2D6 enzymes and ultra-rapid metabolisers will have reduced plasma concentrations of the active compound compared with more efficient metabolisers, increasing the risk of treatment failure[38]. However, since there are insufficient data to recommend an initial paroxetine dose for CYP2D6 ultra-rapid metabolisers, there is a strong recommendation to avoid it altogether in these patients[38] . The same bypass approach is recommended in CYP2C19 ultra-rapid metabolisers of citalopram and escitalopram where an alternative SSRI, not extensively metabolised by

CYP2C19, is recommended[38]. Sertraline is also metabolised by CYP2C19 but differences in opinion exist in prescribing recommendations for poor and ultra-rapid metabolisers[38].

The future

In conclusion, patients are commonly taking high-risk medicines with pharmacogenomic associations when they are admitted to hospital. Results from our retrospective cross-sectional study, alongside the published evidence, suggest that carrying out point-of-care pharmacogenomic testing prior to initiating these medicines in older people could effectively optimise medicines and reduce ADRs. However, there are numerous challenges for effective implementation of this type of testing.

> Carrying out point-of-care pharmacogenomic testing prior to initiating these medicines in older people could effectively optimise medicines and reduce ADRs

A joint report from the Royal College of Physicians and British Pharmacological Society, published in March 2022, highlighted key areas that require consideration, including standardising the consent process; storing and returning pharmacogenomic results in a user-friendly manner; building patient trust; and increasing the confidence of healthcare prescribers in pharmacogenomics[7].

Furthermore, current approaches to point-of-care testing could be restrictive in the genetic variants they test for as the frequency of genetic variants can differ significantly between ethnicities. A panel test approach, whereby multiple variants for multiple medicines are tested together, could provide the coverage required for testing variants that interact with the medicines discussed in this article.

Pharmacy teams

The expertise of pharmacists will be invaluable in delivering pharmacogenomic-guided medicines optimisation, for instance, when conducting structured medication reviews in England and under independent prescribing initiatives in Scotland and Wales. However, major initiatives in training and education are required to enable healthcare professionals to support improvements in personalised patient care led by genetic information[41]. This must happen as a matter of urgency, so that the health service can make effective use of pharmacogenomic testing to improve the care of older people.

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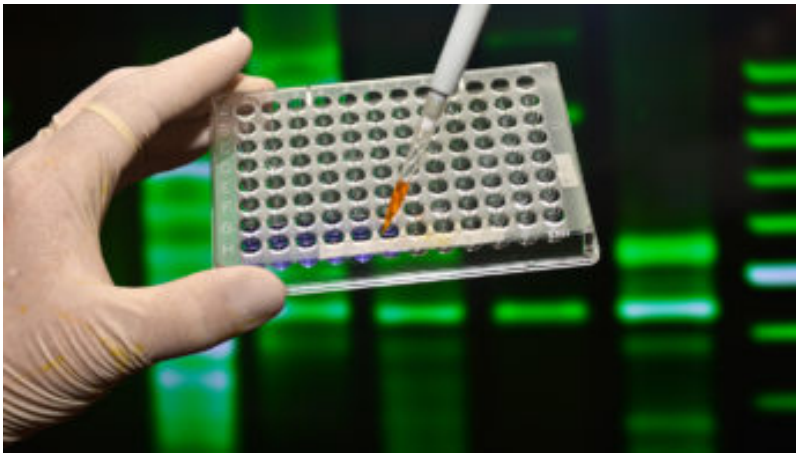

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