multidisciplinary collaboration to maximize assessment and outcomes for youths with ASD.

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Thoughtful Clinical Use of Pharmacogenetics in Child and Adolescent Psychopharmacology

To the Editor:

AACAP's recent policy statement on Clinical Use of Pharmacogenetic Tests in Prescribing Psychotropic Medications for Children and Adolescents[1] recommends that "clinicians avoid using

pharmacogenetic testing to select psychotropic medications in children and adolescents." We agree that there are limitations to the nascent evidence base for using pharmacogenetics, especially in combinatorial form (eg, test results that bin medications based on multiple genes). However, all-or-nothing recommendations fail to recognize the nuance and context of this testing and contrast with the AACAP Facts for Families on pharmacogenetic testing. Moreover, pharmacogenetic testing may inform dosing for antidepressants that are commonly used in child and adolescent psychiatry (eg, sertraline, escitalopram, citalopram, fluvoxamine) as well as the tolerability of some psychotropic medications. With this in mind, we wish to remind the AACAP community of the accumulating evidence and to highlight important principles of pharmacogenetic testing in youths. Specifically:

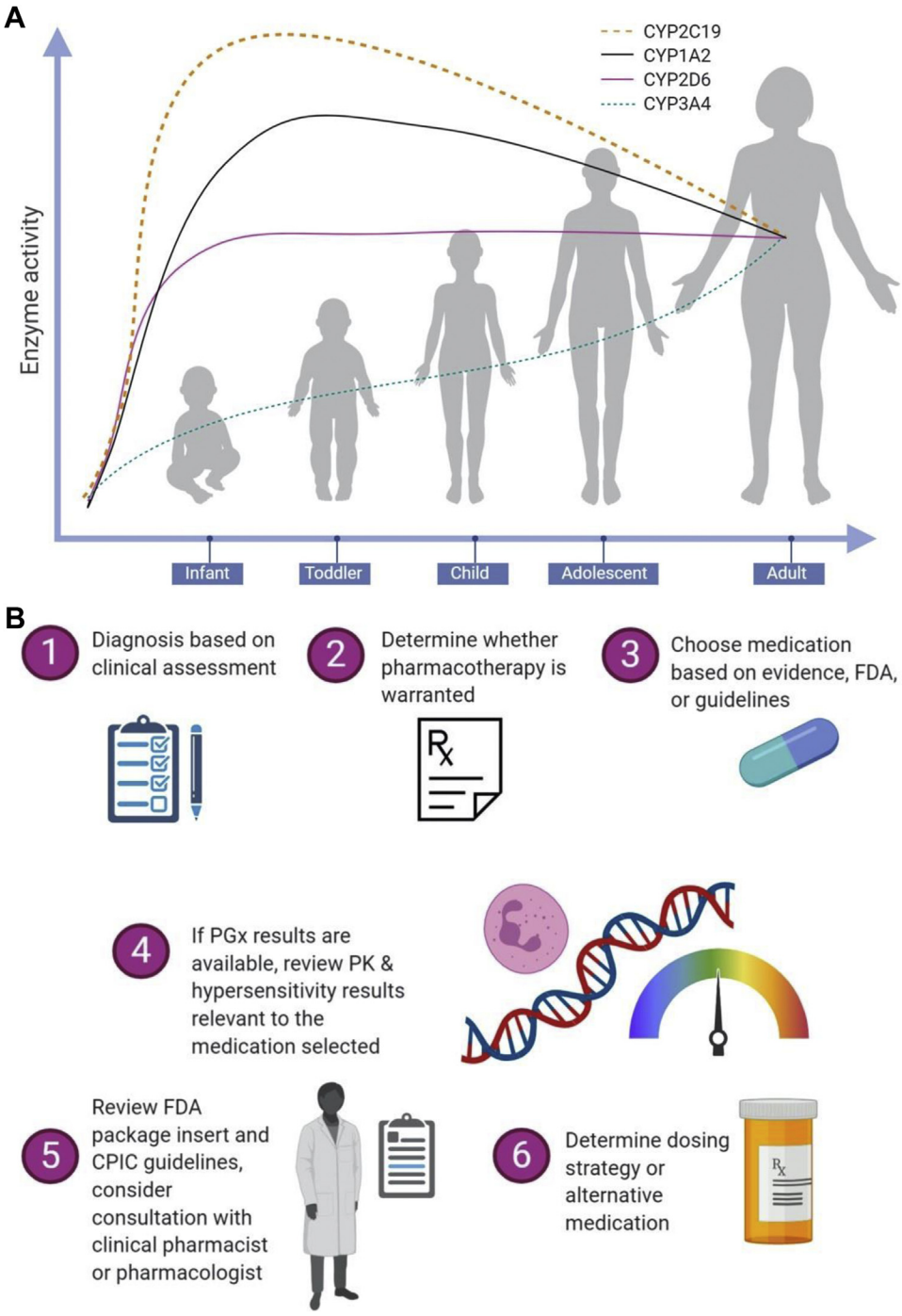
1) pharmacogenetic testing is not always performed by commercial companies and is not always combinatorial;
2) dosing recommendations or assessment of risk for severe hypersensitivity reactions are based on pharmacogenetics in the Food and Drug Administration (FDA)—approved product inserts for several medications commonly prescribed to children (eg, citalopram, aripiprazole, atomoxetine, carbamazepine, oxcarbazepine at www.fda.gov/drugs/science-and-research-drugs/table-pharmacogenomic-biomarkers-drug-labeling);
3) expert consensus guidelines for dosing or identifying hypersensitivity risk for these drugs are available from the National Institutes of Health (NIH)—supported Clinical Pharmacogenetics Implementation Consortium (CPIC, www.cpicpgx.org/), which provides transparent, regularly updated, and evidence-based evaluations of pharmacogenetic data;[2] and
4) randomized trials are not required for clinical dose adjustments; for example, dose adjustments because of decreased hepatic function or concomitant interacting medications are based on pharmacokinetic data, similar to many pharmacokinetic gene-based recommendations from CPIC.

There are limitations to some commercially available pharmacogenetic tests.[3] First, these tests may promise to predict which medications will be effective and provide optimal drug concentration (ie, exposure) based on combinations of pharmacokinetic genes. Second, there is often no explicit acknowledgement of the variable strength of evidence for gene—drug pairs on many of these tests. Evidence for gene—drug pair associations in pediatric patients has recently been reviewed.[4] Third, pharmacogenetic variants may be included on commercial tests for which there are limited or no data in pediatrics (eg, *CYP3A4* and *DRD4*), and we caution against applying these to child and adolescents based solely on theoretical application. Fourth, many clinical factors must be considered when deciding which medication to use for an individual patient, including developmental effects on pharmacokinetically related enzymes (eg, cytochromes) (Figure 1A). We do not advocate selecting medications based solely on pharmacogenetics, ignoring clinical factors or the evidence base for medications. However, commercial and noncommercial pharmacogenetic testing has clinical utility, when combined with patient and clinical characteristics that are incorporated into an evidence-based practice (Figure 1B).

In both child and adolescent as well as adult psychiatry, there is no consensus on the utility of pharmacogenetic testing, but dosing recommendations based on pharmacokinetic genes are included in FDA labels and CPIC consensus guidelines. These guidelines are used at many institutions, in adult as well as child and adolescent psychiatry, although in child and adolescent psychiatry, we must remain cautious in adapting pharmacokinetic models in adults to pediatric patients, given that developmental factors also have an impact on medication metabolism (Figure 1A). Currently, several institutions have implemented in-house (noncommercial) pharmacogenetic testing for *CYP2D6* and *CYP2C19*, which encode enzymes that metabolize many of the medications used in child and adolescent psychiatry (which are also included on commercial tests). Furthermore, in prospective and retrospective pediatric studies, variation in these pharmacokinetic genes affects drug concentration, tolerability,[5] and response for SSRIs metabolized by these enzymes.[4] Dose adjustments for citalopram, escitalopram, and sertraline based on CYP2C19 metabolizer status are supported by modeling studies in adolescents[6] and recommended by CPIC,[2] in addition to dose adjustments for atomoxetine, fluvoxamine, and paroxetine based on CYP2D6 metabolizer status.

The FDA also recommends dosing based on CYP2C19 for citalopram and CYP2D6 for aripiprazole and atomoxetine in the product inserts. These dose recommendations are based on pharmacokinetic parameters that aim to equalize exposure (ie, area under the concentration—time curve). Such dose recommendations have the potential to reduce side effects associated with high exposure or treatment failure associated with low exposure to these medications, although the relationship between exposure and efficacy may be difficult to observe, as it may be confounded by tolerability and other factors. Increased metabolism in rapid and ultrarapid metabolizers may cause patients to seem "treatment resistant" when their medication blood concentrations (exposure) are sub-therapeutic, as was

FIGURE 1 Ontogenic Changes in Activity of Cytochrome P450 Enzymes and Incorporation of Pharmacogenetic Testing into Clinical Practice



Note: *In panel A, the lines representing CYP2C19, CYP1A2, and CYP2D6 are based on in vitro data described in Upreti et al.[11], whereas, due to the difficulty assessing CYP3A4 individually in vivo, the line representing CYP3A4 was based on in vitro data from the same reference. Panel B graphically describes a pharmacogentic testing approach that is based, in part, on a process described in the text of Namerow* et al[3]. *These original figures were created with biorender.com. Please note color figures are available online.*

observed for fluoxetine and sertraline in the Treatment of SSRI-resistant Depression in Adolescents Study.[7] Although more data will be helpful to refine our understanding of exposure response relationships and dosing recommendations for children and adolescents, pharmacogenetic testing could help clinicians to be comfortable with faster titration or higher-than-usual doses. In addition, there is a boxed warning in carbamazepine's product insert that patients with ancestry in

genetically at-risk populations (Asians) should be screened for the presence of *HLA-B*1502* prior to initiating treatment, and because of the high risk of severe dermatologic reactions, including Stevens–Johnson Syndrome (odds ratio >100), carriers should not receive carbamazepine unless benefits clearly outweigh risks. These data stand in contrast with the AACAP policy statement that "genetic variations are managed clinically with slow and thoughtful medication management."

Notwithstanding the current limitations of pharmacogenetic testing, education will be critical for clinicians to feel comfortable interpreting pharmacogenetic test results.[8] This is important, in that we have seen non–evidence-based medications selected or medications for which there is no evidence of efficacy in children and adolescents (eg, desvenlafaxine) chosen based on misinterpretation of pharmacogenetic test reports. Ideally, such education will occur in medical school, residency, and at the fellowship level in addition to continuing medical education for practicing clinicians. This training and education must involve not just psychiatrists but pharmacists, primary care providers, and advanced practice nurses.

Child and adolescent psychiatry might glean experience from other medical specialties (eg, oncology, gastroenterology, and infectious disease) for the thoughtful implementation of pharmacogenetics.[9] These fields progressed with the use of pharmacogenetics without waiting for prospective, randomized controlled trials. For example, in youths with acute lymphoblastic leukemia or inflammatory bowel disease, pharmacogenetic testing is routine prior to prescribing thiopurines. In addition, in youths with hematopoietic stem cell transplants, voriconazole fungal prophylaxis is based on *CYP2C19* at some institutions, given that standard dosing produces inadequate blood levels in many patients.

The term "genetic exceptionalism" has been used to describe higher standards being applied to genetic testing compared to other clinical tests, such as the call for randomized controlled trials to test dose adjustments based on pharmacogenetics but not other clinical factors.[10] For example, there are no randomized controlled trials examining the use of standard assessments for child and adolescent psychiatry, including screening with thyroid function tests, monitoring electrocardiograms in patients treated with tricyclic antidepressants (TCAs) or pimozide, or transaminase monitoring in divalproex-treated youth. As Mrazek and Lerman wrote nearly a decade ago regarding pharmacogenetics, "waiting for data from prospective randomized clinical trials may be depriving patients of safer and more effective medications."[11] If pharmacogenetic testing is obtained or results already exist, using the *CYP2D6* and/or *CYP2C19* results based on recommendations in the product insert and/or included in CPIC's guidelines could benefit the patient.

At this juncture, accumulating data in child and adolescent psychiatry and recommendations from the FDA support integrating pharmacogenetic testing with evidence-based dosing. Thus, we recommend a policy that supports considering pharmacogenetic test results for genes with high levels of evidence if they are available, or testing if the clinician feels that results of evidence-based genes (eg, *CYP2D6, CYP2C19, HLA-A, HLA-B*) would clarify drug dosing or selection decisions in addition to standard evaluation. Our recommendation is consistent with the International Society of Psychiatric Genetics, which supports *CYP2C19* and *CYP2D6* testing for patients who have experienced an inadequate response to or adverse effects of antidepressants or antipsychotics. Insurance companies are beginning to cover pharmacogenetic testing when the clinician selects a medication that has a gene association listed in the FDA label or has a CPIC guideline. In addition, we agree with *HLA-A* and *HLA-B* testing prior to prescribing carbamazepine/oxcarbazepine (ispg.net/genetic-testing-statement). However, the decision to use pharmacotherapy should be based on a thorough clinical evaluation. Medication choice should be based on available evidence, and pharmacogenetic testing—when obtained—should influence dosing and alter the level of monitoring or choice of medication within the evidence-based class of medications for a given condition.[4]

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In Reply

Thank you for the opportunity to respond to the letter, "Thoughtful Clinical Use of Pharmacogenetics in Child and Adolescent Psychopharmacology." We appreciate the thoughtful consideration by Ramsey *et al*.[1] of the American Academy of Child and Adolescent Psychiatry (AACAP) policy statement on the Clinical Use of Pharmacogenetic Tests in Prescribing Psychotropic Medications for Children and Adolescents.[2] In this reply, we will highlight many of the important points that the authors have included and will also express our concerns regarding some of the authors' conclusions in light of the current level of evidence.

The authors make several valid and important points. These include the following:

- pharmacogenetic testing is not always performed by commercial companies and is not always combinatorial;
- patients with ancestry in genetically at-risk populations (individuals of Asian ancestry) should be screened for the presence of *HLA-B*1502* prior to initiating treatment with carbamazepine;
- the misinterpretation of pharmacogenetic test reports may lead to the selection of non−evidence-based medications;
- the decision to use pharmacotherapy should be based on a thorough clinical evaluation.

However, we believe that several of the letter's conclusions are either overly broad or inaccurate. We highlight the following 6 conclusions that are of particular concern to us.

- ***The US Food and Drug Administration (FDA) package inserts do not recommend that dosing be based on performing pharmacogenomic testing.*** The letter incorrectly states that "dosing recommendations or assessment of risk for severe hypersensitivity reactions are based on pharmacogenetics in the FDA-approved product inserts for several medications commonly prescribed to children." To the contrary, dosing recommendations are *not* based on pharmacogenomic testing for citalopram, aripiprazole, or atomoxetine. The FDA's Table of Pharmacogenomic Biomarkers in Drug Labeling[3] does not make this recommendation. It actually "lists [medications] with pharmacogenomic