Atazanavir (Reyataz, ATV)

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Summary

- Atazanavir (ATV) concentrations are reduced during pregnancy.
    - Only ritonavir-boosted ATV (ATV/r) should be used in pregnancy, as ATV levels are too low if unboosted ATV or cobicistat (COBI)-boosted ATV (ATV/c) is used in pregnancy.
    - ATV levels are further reduced when given concomitantly with tenofovir disoproxil fumarate (TDF) or an H2-receptor antagonist.
    - Intracellular ATV levels in women taking the standard dose (ATV/r 300 mg/100 mg) without concomitant TDF appear reassuringly stable throughout pregnancy.
    - An increased dose (ATV/r 400 mg/100 mg once daily with food) during the second and third trimesters should be used if ATV is given concomitantly with TDF or an H2-receptor antagonist.
- First-trimester exposure to ATV is not associated with increased risk of congenital anomalies.
- Regimens with protease inhibitors (PIs), including ATV/r, may be associated with increased rates of preterm birth.

Human Studies in Pregnancy

Pharmacokinetics

ATV/r Without Concomitant TDF

In studies that evaluated full pharmacokinetic (PK) profiles of daily ATV/r 300 mg/100 mg without concomitant TDF during pregnancy, the ATV area under the curve (AUC) was lower during pregnancy than the ATV AUC reported in other studies of nonpregnant adults with HIV.[1-4] In one of the studies, no difference was observed in the ATV AUC during pregnancy and postpartum, but the AUC at both times was lower than the AUC observed in historic nonpregnant controls with HIV.[4] In the other studies, the ATV AUC was lower during pregnancy than it was in the same patients postpartum and in nonpregnant control populations.[1-3,5] Intracellular ATV levels in women taking ATV/r 300 mg/100 mg appear stable throughout pregnancy.[6] Genetic variants appear to partially explain the interpatient variability in third-trimester ATV exposure seen in pregnant women who receive ATV/r.[7]

In studies that evaluated the use of a once-daily increased dose (ATV/r 400 mg/100 mg) during pregnancy,[3,5] pregnant women who received this dose without TDF had an ATV AUC that was equivalent to the ATV AUC seen in historic nonpregnant controls with HIV who received the standard ATV 300-mg dose without TDF. Viral suppression (<50 copies/mL) before birth was observed in 90% to 100% of women receiving standard or increased-dose regimens; none of the infants had evidence of HIV infection.[3,5]

ATV/r With Concomitant TDF

ATV/r combined with TDF and emtricitabine (FTC) provides a complete, once-daily antiretroviral therapy regimen for use during pregnancy. However, the ATV AUC of pregnant women in the third trimester who received concomitant TDF was 30% lower than the ATV AUC of women who were not receiving concomitant TDF, an effect similar to that seen in nonpregnant adults.[1,2] The magnitude of the increase in ATV AUC postpartum relative to ATV AUC in the third trimester in women taking concomitant TDF was similar to that in women not taking concomitant TDF.[1] On the other hand, a smaller PK study demonstrated that concomitant TDF did not result in a lower ATV AUC or a higher risk of ATV trough concentrations (C_{trough}) <0.15 mg/L (the target C_{trough} for patients who are antiretroviral [ARV]-naive) in pregnant women during their third trimester.[8] In a therapeutic drug monitoring study of 103 women (most of whom were African) in Paris, the proportions of women with an ATV C_{trough} of <0.15 mg/L were similar for women who did and women who did not take concomitant TDF.[9]

Pregnant women who received the increased ATV 400-mg dose with TDF had an ATV AUC equivalent to that seen in nonpregnant patients with HIV who received the standard ATV 300-mg dose with TDF.[3,5] An increased dose of ATV during the second and third trimesters is recommended for ARV-experienced pregnant women who also are receiving either TDF or an H2-receptor antagonist.[10] For additional details about interactions between concomitant medications, see Drug–Drug Interactions in the Adult and Adolescent Antiretroviral Guidelines.

Coadministration of tenofovir alafenamide (TAF) and ATV/r in nonpregnant adults has no effect on ATV levels but results in 89% and 162% higher mean TAF and TFV AUCs, respectively.[11] The U.S. Food and Drug Administration advises that no clinically significant drug interactions are expected between TAF and ATV/r and therefore, does not recommend dose adjustment or drug substitution.[12]

ATV Boosted With Cobicistat

The pharmaco-enhancing effect of COBI on ATV is impacted during pregnancy. Pregnant women who received ATV boosted with COBI had ATV C_{trough} that were 66% and 72% lower during the second and third trimesters, respectively, than paired postpartum C_{trough} ($P = 0.0625$ and $P = 0.0313$, respectively).[13] Concomitant use of ATV and COBI **is not recommended** during pregnancy because of these substantial reductions in drug exposures (see Cobicistat).[14]

Based on physiologically based pharmacokinetic PK modeling, twice-daily (instead of the standard adult once-daily) ATV/r 300/100 mg may be able to maintain antiviral efficacy when given with once-daily rifampicin 600 mg in pregnancy, but direct clinical confirmation of this approach is lacking.[15] See the ATV FDA label for a full list of drugs with potential interactions and drugs for which administration with ATV/r **is contraindicated.**

Placental and Breast Milk Passage

In studies of women receiving ATV/r combination therapy during pregnancy, cord blood ATV concentration averaged 13% to 21% of maternal serum levels at birth.[1,4,10]

In a study of three women, the median ratio of breast milk ATV concentration to plasma ATV concentration[16] was 0.13.

Teratogenicity/Adverse Pregnancy Outcomes

In a multicenter study that evaluated a U.S. cohort of children who were exposed to HIV but did not contract HIV, first-trimester ATV exposure was associated with increased odds of congenital anomalies of the skin (adjusted odds ratio [aOR] 5.24; *P* = 0.02) and musculoskeletal system (aOR 2.55; *P* = 0.007).[17] On the other hand, there was no association between first-trimester ATV exposure and birth defects in a French cohort, although this study[18] had <50% power to detect an aOR of 1.5.

The Antiretroviral Pregnancy Registry (APR) provides updated birth defect data for ATV and other ARV drugs twice a year through an interim report released in June and December. The APR has monitored sufficient numbers of first-trimester exposures to ATV in humans to be able to detect at least a 1.5-fold increase in the risk of overall birth defects and at least a twofold increase in the risk of cardiovascular and genitourinary defects (the most common classes of birth defects in the general population). No such increase in the risk of birth defects has been observed with ATV. Figure 1. Summary of Birth Defects Among First Trimester Exposures in the APR interim report provides a summary of the number and prevalence of birth defects per live births among cases of first-trimester exposure to ATV and other ARV drugs reported to the APR where there are sufficient data to determine 95% confidence intervals (CIs). The data in Figure 1 can be compared with the prevalence of birth defects in the U.S. population (2.72 birth defects per 100 live births) based on the Centers for Disease Control and Prevention surveillance system (The Metropolitan Atlanta Congenital Defects Program [MACDP]) and with the Texas Birth Defects Registry ([TBDR] 5.07 per 100 live births).

See Antiretroviral Drug Regimens and Pregnancy Outcomes for a discussion of the potential association between the use of boosted PIs and preterm birth.

Other Safety Data

Elevation in indirect (unconjugated) bilirubin that can be attributed to ATV-related inhibition of the hepatic uridine diphosphate glucuronosyltransferase (UGT) enzyme occurs frequently during treatment with ATV, including during pregnancy.[19] In late pregnancy, an increased dose (ATV/r 400 mg/100 mg) without concomitant TDF, compared with the standard dose of ATV/r without concomitant TDF, is associated with twice the risk of elevated maternal bilirubin levels.[3] It is unknown whether elevated maternal indirect bilirubin throughout pregnancy has any effect on the fetus. Dangerous or pathologic postnatal elevations in bilirubin have not been reported in infants born to mothers who received ATV during pregnancy.[1,3,4,20-23] Maternal bilirubin levels do not correlate well with neonatal bilirubin levels.[24] In some studies, neonatal bilirubin elevations that require treatment with phototherapy occur more frequently after prenatal ATV exposure. However, decisions to use phototherapy frequently are subjective, and guidelines for phototherapy vary across countries, making it difficult to compare the severity of hyperbilirubinemia among patients within a study and across different studies.[21,22] Elevated neonatal bilirubin in neonates exposed to ATV is not associated with UGT-1 genotypes that have been linked to decreased UGT function.[23]

In an evaluation of neurodevelopmental outcomes in 374 infants aged 9 to 15 months who were exposed to HIV but who did not contract HIV, the adjusted mean scores on the language and social-emotional domains of the Bayley-III test were significantly lower for infants with perinatal exposure to ATV than for infants who were exposed to other drugs.[25,26] In a study of language assessments among 792 children aged 1 to 2 years who were exposed to HIV but who did not contract HIV, children with ATV exposure had an increased risk of late language emergence at age 12 months (aOR 1.83; 95% CI, 1.10–3.04) compared with children without ATV exposure, but this association was not significant at 24 months.[27]

Hypoglycemia (glucose <40 mg/dL) that could not be attributed to maternal glucose intolerance, difficult birth, or sepsis was reported in 3 of 38 infants exposed to ATV who had glucose samples collected during the first day of life. All three hypoglycemic infants' glucose samples were adequately collected and processed in a timely fashion.[10] This report of infant hypoglycemia is similar to a prior report in which 2 of 14 infants who were exposed to PIs (i.e., nelfinavir, saquinavir, or indinavir) developed hypoglycemia during the first day of life; both infants with hypoglycemia had been exposed to nelfinavir.[28]

Animal Studies

Carcinogenicity

In *in vitro* and *in vivo* assays, ATV shows evidence of clastogenicity but not mutagenicity. Two-year carcinogenicity studies in mice and rats were conducted with ATV. In female mice, the incidence of benign hepatocellular adenomas increased at systemic exposures that were 2.8-fold to 2.9-fold higher than those seen in humans who received the recommended therapeutic dose (ATV/r 300 mg/100 mg once daily). There was no increase in the incidence of tumors in male mice at any dose and no significant increase in the incidence of neoplasms in rats at systemic exposures up to 1.1-fold (in males) or 3.9-fold (in females) higher than those seen in humans who received the recommended therapeutic dose.[10]

Reproduction/Fertility

No effect of ATV on reproduction or fertility in male and female rodents was observed at drug exposure levels (as measured by AUC) that were 0.9-fold (in males) and 2.3-fold (in females) higher than the exposures achieved in humans who received the recommended therapeutic dose.[10]

Teratogenicity/Adverse Pregnancy Outcomes

In animal reproduction studies, no evidence of teratogenicity was observed in offspring born to animals that had systemic ATV exposure levels (as measured by AUC) that were 0.7 times (in rabbits) and 1.2 times (in rats) those observed in humans who received the recommended therapeutic dose. In developmental toxicity studies in rats, maternal dosing (through pregnancy and lactation) that produced systemic ATV exposure that was 1.3 times the human exposure resulted in reversible neonatal growth retardation. However, offspring were unaffected at lower maternal doses that produced systemic drug exposures equivalent to those observed in humans who received the recommended therapeutic dose.[10] A separate study demonstrated an association between maternal PI use (including the use of ATV) and lower progesterone levels, which correlated with lower birthweight in mice.[29,30] Maternal administration of ATV (with TDF/FTC or abacavir/lamivudine) was associated with delayed postnatal (infant) growth and with effects on brain development and cognitive and motor outcomes in mice.[31,32]

Placental and Breast Milk Passage

ATV maternal-to-fetal (transplacental) transfer is reduced, possibly because ATV is a substrate of the p-glycoprotein, which is an adenosine triphosphate–binding cassette transporter responsible for drug efflux across the placenta.[33]

ATV is excreted in the milk of lactating rats. Maternal ATV use in rats that produced systemic ATV exposure that was 1.3 times the human exposure was associated with neonatal growth restriction that reversed after weaning.[10]

Excerpt From Table 14

Note: When using fixed-dose combination (FDC) tablets, refer to other sections in Appendix B and Table 14 in the Perinatal Guidelines for information about the dosing and safety of individual drug components of the FDC tablet during pregnancy.

Generic Name (Abbreviation) *Trade Name*	**Formulation**	**Dosing Recommendations[a]**	**Use in Pregnancy**
Atazanavir (ATV) *Reyataz* **Note:** Generic products are available for some formulations. **Note:** ATV must be combined with low-dose RTV boosting in pregnancy. (ATV/c) *Evotaz*	**ATV (Reyataz)** *Capsules* • 100 mg[c] (generic product only) • 150 mg[c] (generic product only) • 200 mg[c] • 300 mg[c] *Oral Powder* • 50-mg packet **ATV/c (Evotaz)** • ATV 300-mg/COBI 150-mg tablet	**Pregnancy** *PK in Pregnancy* • ATV (Reyataz) o ATV concentrations are reduced during pregnancy, and they are further reduced when ATV is given concomitantly with TDF or an H2-receptor antagonist. o Intracellular ATV levels in women taking the standard dose (ATV/r 300 mg/100 mg) without concomitant TDF appear reassuringly stable throughout pregnancy. • ATV/c (Evotaz) o Use of ATV/c **is not recommended** during pregnancy because ATV C_{trough} are 80% to 85% lower than the ATV concentrations seen in nonpregnant adults. *Dosing in Pregnancy* • ATV (Reyataz) o Use of unboosted ATV **is not recommended** during pregnancy. o Use of unboosted ATV **is not recommended** during pregnancy for ARV-experienced patients who are taking TDF and an H2-receptor antagonist.	Low placental transfer to fetus[b] No evidence of human teratogenicity (can rule out 1.5-fold increase in overall birth defects) Must be given with RTV boosting in pregnancy Effect of *in utero* ATV exposure on infant indirect bilirubin levels is unclear. Nonpathologic elevations of neonatal bilirubin have been observed in some, but not all, clinical trials to date. Oral powder (but **not** capsules) contains phenylalanine, which can be harmful to patients with phenylketonuria. Use of ATV/c **is not recommended** during pregnancy. See Recommendations for Use of Antiretroviral Drugs During Pregnancy, Table 6 and Table 7 for discussions about avoiding the use of ATV/c during pregnancy. **Note:** Please see FDA label for full list of drugs with potential interactions, including several anticonvulsants and other drugs for which administration with ATV/r is contraindicated.

Excerpt From Table 14

Generic Name (Abbreviation) *Trade Name*	**Formulation**	**Dosing Recommendations**[a]	**Use in Pregnancy**
		○ Use of an increased dose (ATV/r 400 mg/100 mg once daily with food) during the second and third trimesters results in plasma ATV concentrations equivalent to those seen in nonpregnant adults receiving standard dosing. Increased ATV dosing is recommended for pregnant people in the second and third trimesters who are also receiving either TDF or an H2-receptor antagonist. • ATV/c (Evotaz) ○ ATV/c should not be used in pregnancy because atazanavir C_{min} is substantially reduced (see COBI). For guidance about the use of combination products in pregnancy, see the specific sections on other components (i.e., COBI). **Standard Adult Doses** *In ARV-Naive Patients Without RTV Boosting* • ATV 400 mg once daily with food; ATV without RTV boosting **is not recommended** when used with TDF, H2-receptor antagonists, PPIs, or during pregnancy. *In ARV-Naive Patients With RTV Boosting* • ATV/r 300 mg/100 mg once daily with food • When combined with EFV in ARV-naive patients: ATV/r 400 mg/100 mg once daily with food	

Excerpt From Table 14

Generic Name (Abbreviation) *Trade Name*	**Formulation**	**Dosing Recommendations**[a]	**Use in Pregnancy**
		In ARV-Experienced Patients • ATV 300 mg plus RTV 100 mg once daily with food • **Do not use** with PPIs or EFV. *In ARV-Experienced Patients Who Are Receiving an H2-Receptor Antagonist* • ATV/r 300/100 mg once daily with food *In ARV-Experienced Patients Who Are Receiving an H2-Receptor Antagonist and TDF* • ATV/r 400 mg/100 mg once daily with food *Powder Formulation* • Oral powder is taken with RTV once daily with food at the same recommended adult dose as the capsules. *ATV/c (Evotaz)* • One tablet once daily with food	

[a] Individual ARV drug doses may need to be adjusted in patients with renal or hepatic insufficiency (for details, see the Adult and Adolescent Antiretroviral Guidelines, Appendix B).

[b] Placental transfer categories are determined by mean or median cord blood–to–maternal delivery plasma drug ratio:

High: >0.6
Moderate: 0.3–0.6
Low: <0.3

[c] Generic product is available.

Key: ARV = antiretroviral; ATV = atazanavir; ATV/c = atazanavir/cobicistat; ATV/r = atazanavir/ritonavir; C_{min} = minimum plasma concentration; C_{trough} = trough concentration; COBI = cobicistat; EFV = efavirenz; FDA = U.S. Food and Drug Administration; PK = pharmacokinetic; PPI = proton pump inhibitor; RTV = ritonavir; TDF = tenofovir disoproxil fumarate

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