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Diagnosis of Congenital Toxoplasmosis: Performance of Four IgG and IgM Automated Assays at Birth in a Tricentric Evaluation

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ABSTRACT For postnatal diagnosis of congenital toxoplasmosis (CT), the gold standard for the detection of anti-*Toxoplasma* IgM in newborns relies on the immunosorbent agglutination assay (ISAGA), which is manufactured from whole *Toxoplasma* parasites that become difficult to maintain. For IgG, only the Platelia assay provides a validated assay for cord blood according to the manufacturer, allowing its use in this context. We compared the analytical performance of four commercialized automated assays, Platelia, Abbott, Vidas, and Liaison, for the detection of IgG and IgM in the cord blood or peripheral blood of newborns from women infected during pregnancy. The assays were performed on samples from 509 newborns, collected from the university hospitals of Montpellier, Nîmes, and Toulouse. For IgM, the four assays appeared to be sufficiently informative to be used for congenital toxoplasmosis diagnosis (area under the curve [AUC] > 0.8, receiver operating characteristic [ROC] analysis), with Platelia showing the best performance, similar to ISAGA with regard to accuracy (83%). For the Vidas (76%), Abbott (75%), and Liaison (74%) assays, the accuracy was significantly lower. Maternal treatment significantly decreased the sensitivity of all the assays. For IgG, the four evaluated assays showed a sensitivity of over 90%, with Abbott (95%) and Liaison (94%), exhibiting a significantly higher sensitivity than Platelia (90%). Furthermore, Abbott showed its superiority in the cases of maternal infection during the third trimester. In the context of the newborns of mothers infected by *Toxoplasma gondii* during pregnancy, to ensure efficient care, Platelia and Abbott seemed to be the most suitable reference tests for the detection of IgM for the former and IgG for the latter.

KEYWORDS immunodiagnosis, postnatal diagnosis, *Toxoplasma gondii*, newborns, maternal seroconversion

Toxoplasmosis is a zoonotic infection that can cause a large spectrum of clinical diseases. Generally asymptomatic, infection with *Toxoplasma gondii* during pregnancy can cause severe symptoms or sequelae or have fatal consequences for the fetus (1). In France, a national program for prevention has been set up to prevent congenital toxoplasmosis (CT); this program provides prophylactic recommendations and monthly serological monitoring (IgG and IgM) in nonimmune pregnant women (2). Despite this, almost 0.02% of the newborns presented with congenital toxoplasmosis in 2018 in France (3). In about 10% of cases, neonates present with a proven toxoplasmosis at birth, despite a negative prenatal diagnosis, and in about 30% of cases, prenatal diagnosis is not performed because of late infection during gestation (4). Therefore, postnatal diagnosis is essential to diagnose infected neonates and start treatment, as recommended (5–7). Postnatal diagnosis relies on a combination of several methods: parasite detection on amniotic fluid or placenta collected during delivery, cord

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blood, and newborn blood by PCR and/or serological screening (detection of neosynthesized IgG, IgM, or IgA) (7, 8). To minimize the risk of mother-to-child transmission, an anti-parasitic treatment is initiated as soon as maternal contamination is established and is pursued until delivery. This strategy has an impact on the serological screening of the newborn, lowering the sensitivity of the tests (9). In this context, it appears that the serological assays used need to be highly efficient to avoid false-negative results and allow early treatment of the newborn, thus minimizing the risk of complications (10). Currently, the gold-standard test for the detection of anti-*Toxoplasma* IgM in newborns relies on a non-automated immunocapture assay manufactured from a *Toxoplasma* tachyzoite suspension, the immunosorbent agglutination assay (ISAGA) (11). This kind of process becomes difficult to maintain, and the evaluation of other commercialized automated tests is required to preserve efficient care. Concerning the detection of IgG in newborns, only one test, Platelia, provides the “validate on blood cord” mention on its manufacturer’s notice, allowing its use in this context. The main objective of this study was to evaluate the analytic performance of four commercial automated assays for detecting IgG and IgM in cord blood or newborn blood in children born from women who contracted toxoplasmosis during pregnancy.

MATERIALS AND METHODS

Ethics. All routine analyses were performed during routine work-up as implemented in the three participating centers. The evaluated methods were performed retrospectively on sample excess. Data were recorded anonymously. The study design was approved by the local ethics committees of the university hospitals of Toulouse/Montpellier and Nîmes (approval number 210361/210600).

Patients and samples. All congenitally infected infants diagnosed from January 2007 to February 2021 in the three laboratories (Département de Parasitologie-Mycologie, CHU Montpellier, Laboratoire de Parasitologie-Mycologie, CHU Nîmes and Service de Parasitologie-Mycologie, CHU Toulouse) were retrospectively included if a cord blood sample or a peripheral blood sample had been analyzed. Diagnosis of congenital toxoplasmosis relied on a positive prenatal diagnosis (parasite DNA detection by qPCR on amniotic fluid (12, 13) and mouse inoculation) and/or detection of specific IgM or IgA in peripheral blood, positive *Toxoplasma* qPCR in peripheral blood, detection of neosynthesized IgG or IgM by Western blot (TOXOPLASMA WB IgG-IgM, LDBio, Lyon, France), or a persistence of IgG after 1 year of follow-up. As a control, toxoplasmosis-free children from prepartum-infected mothers were randomly selected. The absence of congenital toxoplasmosis was confirmed by IgG loss after 1 year of follow-up.

Routine diagnostic methods. Sera were prospectively assessed for the presence of IgG and IgM using at least one of the following assays: (i) in Montpellier, AxSYM Toxo IgM and IgG and then Architect Toxo IgG and IgM assays on the related automated analyzer (Abbott Laboratories, Wiesbaden, Germany) and the ISAGA Toxo IgM non-automated test (bioMérieux, Marcy l’Etoile, France); (ii) in Nîmes, Liaison Toxo IgG II and Liaison Toxo IgM on the automated analyzer Liaison XL (DiaSorin, Saluggia, Italy) and the ISAGA Toxo IgM non-automated test; (iii) in Toulouse, AxSYM Toxo IgM and IgG, then Architect Toxo IgG and IgM on the related automated analyzer (Abbott Laboratories, Wiesbaden, Germany) and Platelia Toxo IgG and IgM tests on the automated analyzer Evolis (Bio-Rad, Marnes-La-Coquette, France). Samples were frozen at -20°C until further analysis.

Evaluated diagnostic methods. For all samples, depending on the residual volume, data were completed using one of the following methods: (i) in Montpellier, with ISAGA Toxo IgM; (ii) in Toulouse, with Alinity Toxo IgG and Toxo IgM assays on the automated analyzer Alinity (Abbott Laboratories, Wiesbaden, Germany), Liaison Toxo IgG II, and Liaison Toxo IgM on the automated analyzer Liaison XL, Vidas Toxo IgG II and IgM on the automated analyzer Mini Vidas (bioMérieux, Marcy l’Etoile, France), and Platelia Toxo IgG and IgM tests on the automated analyzer Evolis.

All tests were performed according to the manufacturers’ instructions. The cutoff values for IgG or IgM detection recommended by the manufacturers were used to interpret the results (Table 1). For easier understanding, since several studies, including local ones (data not shown), have asserted an equivalence between AxSYM and Architect Toxo assays on the one hand (14, 15) and between Architect and Alinity Toxo assays on the other hand (16); these assays were renamed Abbott Toxo IgG and IgM assays. All immunoassays reported the test results in IU/mL for IgG and index for IgM.

Statistical analysis. The characteristics of the studied population were described using percentages and medians along with interquartile ranges (IQR) instead of means and standard deviations when distributions were found to be non-Gaussian. Screening tests were evaluated against the final diagnosis of congenital toxoplasmosis. Diagnostic sensitivity, specificity, and accuracy calculations were performed on all sera included in the study. The results were analyzed using receiver operating characteristic (ROC) curves. The sensitivity, specificity, and accuracy results were compared using a test of equality of proportions. The areas under the ROC curves (AUC) were compared using a χ^2 test. The threshold of significance was set at 5%. All statistical tests and procedures were performed using the Intercooled Stata 9.2 statistical package (StataCorp, College Station, TX).

TABLE 1 IgG/IgM cutoff values recommended by the manufacturers

Assay	IgG			IgM		
	Negative	Gray zone	Positive	Negative	Gray zone	Positive
Abbott ^a	<1.6	1.6 ≤ x < 3	≥3	<0.5	0.5 ≤ x < 0.6	≥0.6
Platelia	<6	6 ≤ x < 9	≥9	<0.8	0.8 ≤ x < 1	≥1
Vidas	<4	4 ≤ x < 8	≥8	<0.55	0.55 ≤ x < 0.65	≥0.65
Liaison	<7.2	7.2 ≤ x < 8.8	≥8.8	<6	6 ≤ x < 8	≥8
ISAGA ^b				<3		≥3

^aAbbott comprised data from AxSYM, Architect, and Alinity.

^bISAGA cutoff for infant.

RESULTS

Description of population, samples, and dosages. From 1 January 2007 to 31 January 2021, 289 cases of congenital toxoplasmosis were diagnosed in the Toulouse, Montpellier, and Nîmes university hospitals. Among these patients, 120 presented at least positive parasite DNA detection by quantitative PCR (qPCR) on amniotic fluid. As a control group, 220 toxoplasmosis-free children from prepartum-infected mothers were randomly selected. Table 2 shows the criteria which allowed the confirmation of CT diagnosis, the trimester of pregnancy at the time of infection, the maternal treatment, and the origin of the newborn sample. The median age at peripheral blood sampling was 4 days (IQR: 3 to 24). According to residual volumes, for IgG dosage, 447 sera were assessed with Abbott and Platelia, 310 with Liaison, and 309 with Vidas. For IgM dosage, 446 sera were assessed with ISAGA, 432 with Platelia, 384 with Abbott, 357 with Vidas, and 311 with Liaison.

Analytical performance of IgM assays. In Table 3, the specificity, sensitivity, and accuracy of the IgM assays were assessed for each reagent at the suppliers' thresholds.

TABLE 2 Description of the population (*n* = 509)

Characteristics	<i>N</i> (%)
Infants included in the study (<i>n</i> = 509)	
Infected	289 (56.8)
Noninfected	220 (43.2)
Sample origin (<i>n</i> = 509)	
Cord blood	413 (81.2)
Peripheral blood	96 (18.8)
Biological test allowing CT diagnosis (<i>n</i> = 289)	
Amniotic fluid PCR	120 (41.5)
Blood PCR/mouse inoculation	60 (20.8)
IgM/IgG neosynthesis on cord blood by western blot	35 (12.1)
IgM/IgA synthesis on peripheral blood at ≥ 10 days of life	66 (22.8)
Increased IgG during follow-up	8 (2.8)
Date of maternal seroconversion (<i>n</i> = 509) ^a	
Periconceptional	51 (10.0)
1st trimester	68 (13.4)
2nd trimester	164 (32.2)
3rd trimester	173 (34.0)
Childbirth	36 (7.1)
Postpartum	2 (0.4)
Unknown	15 (2.9)
Maternal treatment (<i>n</i> = 509)	
None	63 (12.4)
Spiramycin alone	260 (51.1)
Malocide/Adiazine (preceded or not by spiramycin)	106 (20.8)
Treatment (no information on which one)	5 (1)
No information	75 (14.7)

^aCT, congenital toxoplasmosis.

TABLE 3 Overall relative sensitivity, specificity, and accuracy by testing samples for anti-*Toxoplasma* IgM, impact of maternal treatment, and sample origin^a

Assay group	Sensitivity, % [95% CI]	Specificity, % [95% CI]	Accuracy (%)	<i>P</i> ^b
ISAGA				
All (<i>n</i> = 446)	73.26 [69.15–77.36]	90.96 [88.30–93.62]	80.71	1 ^c
Peripheral blood (<i>n</i> = 62)	82.35 [72.86–91.84]	100		1 ^d
Treated (<i>n</i> = 333)	68.91 [63.94–73.88]	92.86 [90.09–95.62]		0.006 ^e
Untreated (<i>n</i> = 42)	87.50 [77.50–97.50]	70.00 [56.14–83.86]		
Platelia				
All (<i>n</i> = 432)	75.63 [71.58–79.68]	92.78 [90.34–95.22]	83.33	0.312 ^c
Peripheral blood (<i>n</i> = 59)	80.49 [70.38–90.60]	100		0.792 ^d
Treated (<i>n</i> = 319)	71.91 [66.98–76.84]	94.33 [91.79–96.86]		0.005 ^e
Untreated (<i>n</i> = 49)	88.89 [80.09–97.69]	92.78 [90.34–95.22]		
Abbott				
All (<i>n</i> = 384)	55.77 [50.80–60.74]	97.16 [95.50–98.82]	75.00	0.024 ^c
Peripheral blood (<i>n</i> = 36)	64.29 [48.63–79.94]	100		0.022 ^d
Treated (<i>n</i> = 291)	52.17 [46.43–57.91]	96.92 [94.94–98.91]		0.042 ^e
Untreated (<i>n</i> = 32)	68.18 [52.04–84.32]	100		
Vidas				
All (<i>n</i> = 357)	58.25 [53.13–63.36]	97.55 [95.94–99.15]	76.19	0.064 ^c
Peripheral blood (<i>n</i> = 28)	59.09 [40.88–77.30]	100		0.009 ^d
Treated (<i>n</i> = 272)	54.30 [48.38–60.22]	98.35 [96.83–99.86]		0.015 ^e
Untreated (<i>n</i> = 30)	75.00 [59.51–90.49]	90.00 [79.26–100.00]		
Liaison				
All (<i>n</i> = 311)	56.55 [51.04–62.06]	97.20 [95.37–99.04]	75.24	0.036 ^c
Peripheral blood (<i>n</i> = 25)	44.44 [24.97–63.92]	100		<0.001 ^d
Treated (<i>n</i> = 248)	54.29 [48.09–60.49]	99.07 [97.88–100]		0.010 ^e
Untreated (<i>n</i> = 21)	76.92 [58.90–94.94]	87.50 [73.36–100.00]		

^aSensitivity: True Positive (TP)/(TP + False Negative [FN]); specificity: True Negative (TN)/(TN + False Positive [FP]); accuracy: (TP + TN)/(TP + TN + FP + FN). 95% CI, 95% confidence interval.

^bTests of equality of proportions.

^cFor accuracy on overall samples.

^dFor sensitivity on peripheral blood between assays (dark gray shading).

^eFor sensitivity between treated and nontreated mothers (light gray shading).

Predictive positive value (PPV) and negative predictive value (NPV) were not informative results because of the very low prevalence (0.02% [3]) of the disease. The doubtful values were considered positive ones. For Platelia, the sensitivity, specificity, and accuracy were not different from those of ISAGA. For Abbott and Liaison, the accuracy and sensitivity of the tests were significantly lower than those of ISAGA ($P < 0.001$); however, the specificity was significantly higher ($P < 0.001$). For Vidas, compared to ISAGA, the test showed a trend towards a lower accuracy, but the difference was not significant ($P = 0.064$); the sensitivity was significantly lower ($P < 0.001$) and the specificity significantly higher ($P < 0.001$). Analyzing the population by sample origin subgroup, for peripheral blood samples (Table 3), the specificity of all tests was 100%, meaning that there were no false-positive results. The sensitivity of the tests was significantly lower for Abbott, Vidas, and Liaison than for ISAGA and Platelia. On cord blood, positive results in the control group were found more often with Platelia and ISAGA than with the other tests, which explains their lower specificity. For all tests, including ISAGA, anti-*Toxoplasma* maternal treatment was associated with decreased sensitivity for the detection of IgM in newborns at birth (Table 3).

As shown in Fig. 1, only one of the five tested reagents was particularly informative ($AUC \geq 0.9$). Platelia had the largest AUC, which was significantly higher than that of ISAGA ($P < 0.001$). It could have an optimal threshold of 0.42 with a sensitivity of 84.45%, a specificity of 87.11%, and an accuracy of 85.65%. At this new threshold,

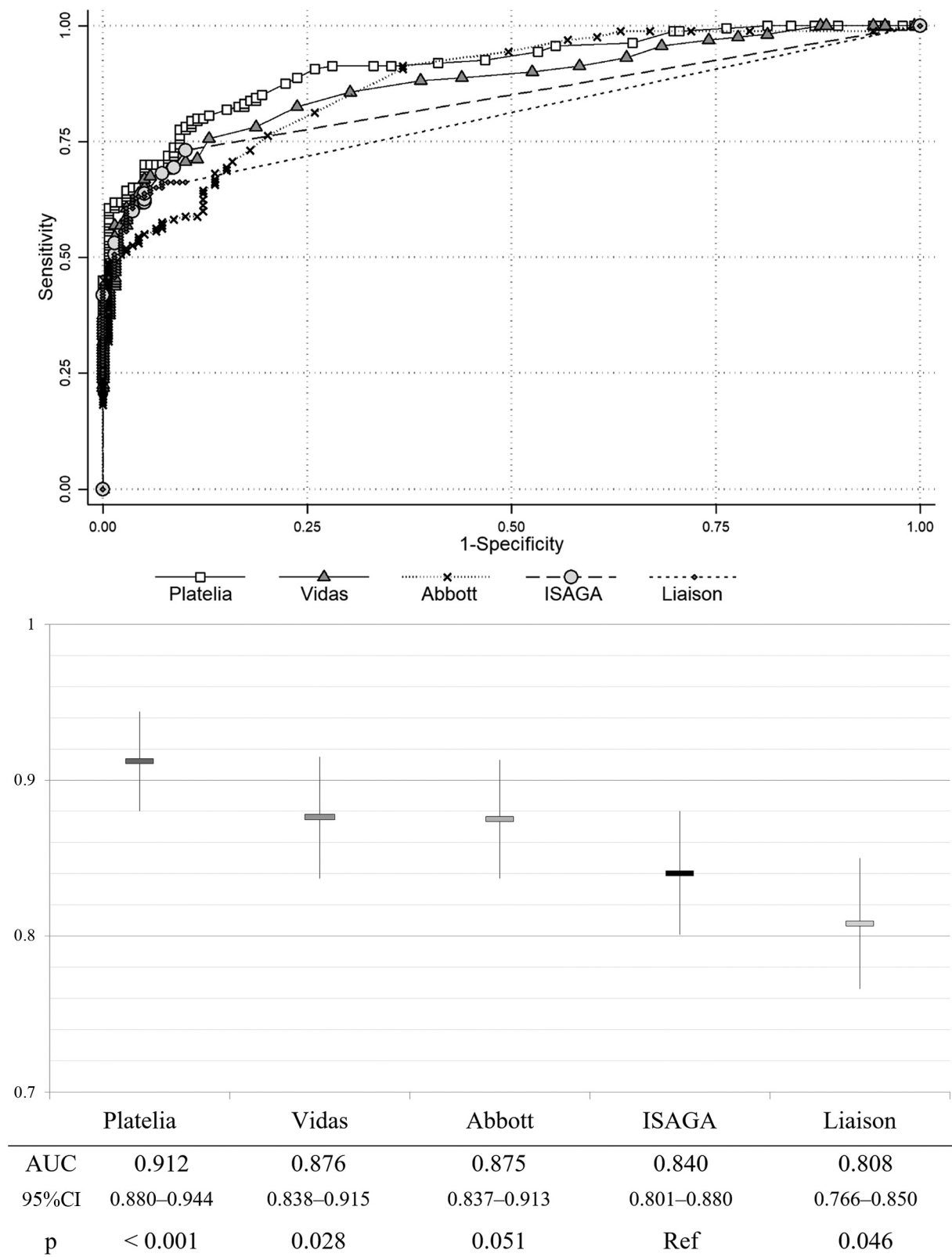
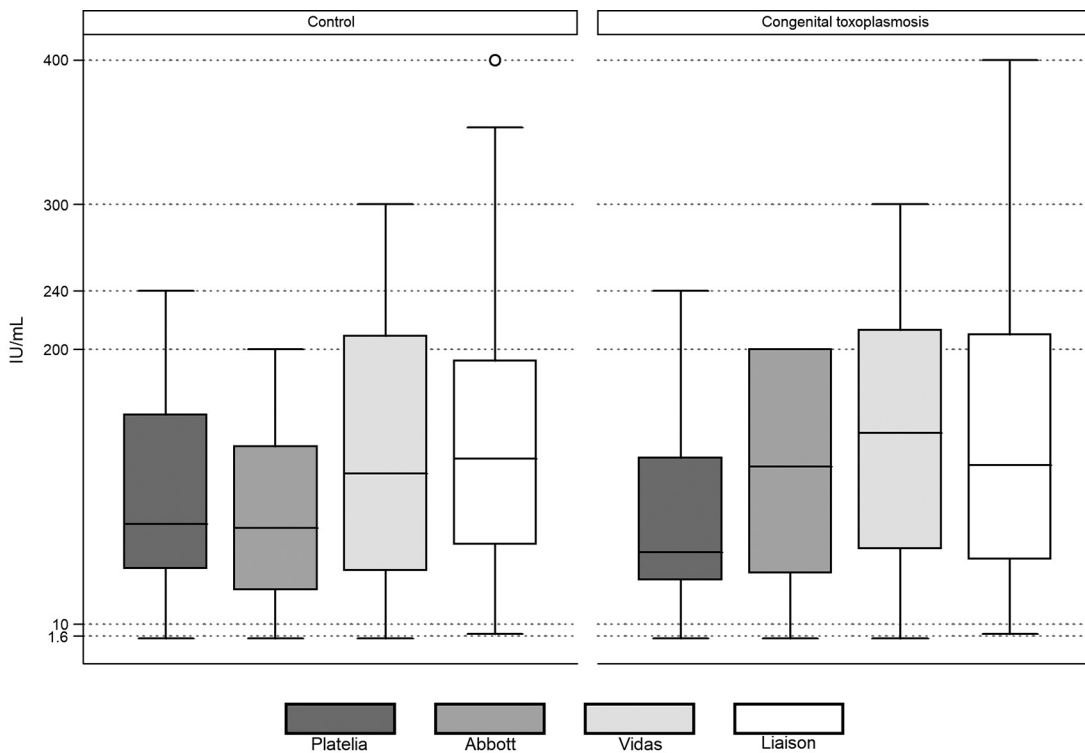


FIG 1 Areas under receiver operator curves for each IgM assay and comparison (χ^2 test) of analytic performances ($n = 299$).



Assay	Platelia		Abbott		Vidas		Liaison	
	CT-	CT+	CT-	CT+	CT-	CT+	CT-	CT+
Group	n = 192	n = 255	n = 201	n = 246	n = 144	n = 165	n = 143	n = 167
Med	78.9	59.6	76.1	118.6	114	136	124	120
[IQR]	[47.9–154.6]	[40.4–124.9]	[33.5–132.6]	[45.2–200]	[46.5–209]	[61–211]	[64.6–192]	[54.7–210]
p	0.008		< 0.001		0.572		0.665	

FIG 2 Distribution of IgG values according to different methods and comparison (Kruskal-Wallis equality-of-populations rank test).

Platelia’s accuracy was significantly higher than ISAGA’s ($P = 0.025$). Vidas was moderately informative ($AUC < 0.9$) but better than ISAGA ($P = 0.028$). It could have an optimal threshold of 0.25 with a sensitivity of 71.65%, a specificity of 90.80%, and an accuracy of 80.39%. At this new threshold, the accuracy of Vidas became similar to that of ISAGA. Abbott’s performance only showed a trend of being better than ISAGA ($P = 0.051$). Liaison, while acceptable, showed a significantly lower performance than ISAGA ($P = 0.046$). For Abbott and Liaison, modulation of the thresholds did not significantly improve accuracy.

Analytical performances of IgG assays. Figure 2 shows the distribution of IgG values according to the manufacturers’ thresholds. A significant difference in the range of IgG values was observed for Platelia and Abbott reagents between the CT group (CT+) and the control group (CT-). Platelia showed higher values in the control group than in the CT group ($P = 0.008$), whereas the opposite was the case for Abbott ($P < 0.001$). No differences were observed between groups for the Vidas and Liaison reagents ($P > 0.05$).

In Table 4, the sensitivity of the IgG assays was assessed for each reagent at the suppliers’ thresholds. As seen for IgM detection, PPV and NPV were not informative results because of the very low prevalence of the disease. The doubtful values were considered positive. In comparison to Platelia, the only assay to have been previously

TABLE 4 Overall relative sensitivity by testing samples for anti-*Toxoplasma* IgG and impact of maternal infection date^a

Assay group	Sensitivity, % [95% CI]	<i>P</i> ^b	<i>P</i>
Platelia			
All (<i>n</i> = 447)	90.20 [87.44–92.95]		1 ^c
MI < T3 (<i>n</i> = 244)	100	<0.001 ^d	
MI ≥ T3 (<i>n</i> = 188)	84.85 [79.72–89.97]		1 ^e
Abbott			
All (<i>n</i> = 447)	95.53 [93.61–97.44]		0.007 ^c
MI < T3 (<i>n</i> = 250)	100	<0.001 ^d	
MI ≥ T3 (<i>n</i> = 182)	93.04 [89.34–96.74]		0.003 ^e
Vidas			
All (<i>n</i> = 309)	91.52 [88.41–94.62]		0.269 ^a
MI < T3 (<i>n</i> = 197)	100	<0.001 ^d	
MI ≥ T3 (<i>n</i> = 108)	85.26 [78.58–91.95]		0.462 ^e
Liaison			
All (<i>n</i> = 310)	94.01 [91.37–96.65]		0.0304 ^c
MI < T3 (<i>n</i> = 195)	98.55 [96.87–100]	<0.001 ^d	
MI ≥ T3 (<i>n</i> = 110)	90.82 [85.44–96.19]		0.069 ^e

^aMI, maternal infection. Sensitivity: TP/(TP + FN); specificity: TN/(TN + FP); accuracy: (TP + TN)/(TP + TN + FP + FN).

^bTests of equality of proportions.

^cFor sensitivity on overall samples.

^dFor sensitivity according to maternal infection date (light gray shading).

^eFor sensitivity between assays when maternal infection occurred in the third trimester (T3; dark gray shading).

validated on blood cord samples, Abbott and Liaison yielded better results in term of sensitivity ($P = 0.002$ and $P = 0.033$, respectively). The sensitivity result for Vidas was comparable to that of Platelia. The accuracy and specificity results were not informative since maternal IgG are passively transmitted to the child. Regarding the time of maternal infection (Table 4), the tests had significantly better sensitivity at birth for infections that occurred during both the first and second trimesters than for those that occurred in the third trimester. For the group of mothers infected during the third trimester of pregnancy, only Abbott exhibited a better sensitivity than Platelia for the detection of IgG in newborns ($P = 0.003$).

DISCUSSION

This study presents a comparison of the analytical performances of four reagents for the detection of anti-*Toxoplasma gondii* IgG and IgM in a large cohort of newborns from women infected with *T. gondii* during pregnancy. Among the large number of publications on toxoplasmosis immunodiagnosics, very few data are available on newborns specifically.

All of the evaluated assays are known to be effective at detecting *T. gondii* IgG and IgM, with very good sensitivity and specificity, called technical sensitivity and specificity. These technical performances must be differentiated from the diagnostic ones. In the context of congenital toxoplasmosis, several pitfalls have an impact on these technical performances. First, the presence of IgG in toxoplasmosis-free newborns due to passive maternal transmission is not a technical false positive, but it decreases the diagnostic specificity of the tests. In the same way, the detection of maternal IgM in the cord blood of a healthy newborn due to contamination that may have occurred during delivery is not a false-positive result, but it has an impact that decreases the diagnostic specificity of the tests. For these reasons, to avoid confusion, we will be talking about diagnostic performances until the end of the discussion.

For IgM, since their detection in newborn peripheral blood asserts a congenital toxoplasmosis diagnosis, high sensitivity and specificity of assays are expected to prevent misdiagnosis and late or unwarranted treatments. In our study, at the suppliers'

thresholds, all evaluated assays except for Platelia had lower accuracy (Abbott and Liaison), or their accuracy trended to be lower (Vidas), than that of ISAGA, whereas doubtful values were included in the positive values. From their AUC profiles, ISAGA, Platelia, Abbott, Liaison and Vidas Toxo IgM appeared to exhibit sufficient global performances to be routinely used for congenital toxoplasmosis diagnosis. However, Platelia and Vidas, with AUCs around 0.9, showed global diagnostic performances which were significantly superior to that of ISAGA. When the thresholds of the tests were modified, the accuracy of Platelia (threshold at 0.42) was significantly higher than that of ISAGA, and the accuracy of Vidas (threshold at 0.25) joined that of ISAGA. Abbott and Liaison were similar to ISAGA or less informative, respectively, without improvement by threshold variation. As shown by Murat et al. (17), regarding peripheral blood only, for any assays, including ISAGA, there was no false-positive IgM. The high sensitivity of ISAGA and Platelia probably led to the detection of a low level of maternal IgM in cord blood which was contaminated during delivery, enhancing the value of the comparative Western blot so as to differentiate neosynthesized antibodies in the newborn from maternal ones. In a study by Murat et al. (17) conducted on pregnant women and newborns, Liaison, Abbott, and Vidas exhibited excellent performance for IgM detection, but they were not compared to ISAGA, and the low number of subjects did not allow for analysis of the newborn subgroups. As shown by Guegan et al. (9), maternal anti-*Toxoplasma* treatment during pregnancy was associated with reduced sensitivity of IgM assays for the diagnosis of congenital infection in newborns. In our study, all tests were impacted by treatment, whereas Platelia was not included in the study by Guegan et al. Because the sensitivity figures are quite different between the two studies, it is difficult to make assumptions on that point.

For IgG, in most cases, IgG detected in the newborn are mother-to-child transmitted. To exclude the contamination of a child born from a prepartum-infected women, IgG have to be null at 1 year of life without anti-*Toxoplasma* treatment. At birth, the real goal for IgG is to differentiate maternally transmitted IgG from newborn neo-synthesized IgG. At the moment, this cannot be done using the automated immunoassays available, and only the commercialized immunoblot from LDBio allows this comparison. For automated assays, high sensitivity is expected to avoid incorrect exclusion of congenital toxoplasmosis at 1 year of follow-up. In our study, the four evaluated assays showed sensitivities, at birth, above 90%. Abbott and Liaison Toxo IgG exhibited a significantly higher sensitivity than Platelia, the reference test. There was no difference between Vidas and Platelia. Several studies (14, 17, 18) have shown that times before IgG detection during seroconversion in pregnant women were significantly different between the automated assays, with a significant advantage for Abbott. In our study, the better sensitivity of Abbott when infection occurred in the third trimester of pregnancy, leading to an absence or very low level of IgG during blood sampling in the newborn, could be explained by the antigenic composition of the assay. In these cases, Abbott could allow earlier detection of neo-synthesized IgG. The performances of the assays were not followed over a sufficiently long period to allow a conclusion about their sensitivity at 1 year of life. In neonates of women infected during pregnancy, mother-to-child transmitted IgG are from recent infection, even if detection is performed at 1 year of age; this could explain why Murat et al. (17) found in their study that Abbott detected IgG in infants for longer than Vidas or Liaison. The performances of the assays at this age should be the same as those at birth. A further study on IgG kinetics needs to be performed to clarify this point.

In conclusion, even if all the tests present satisfactory global performances for the detection of IgM and IgG, Platelia and Abbott seem the most suitable to become reference tests for the detection of IgM for the first one and IgG for the second in the context of newborns of mothers infected by *T. gondii* during pregnancy. It is not possible to propose a single platform or manufacturer which would provide optimal detection of both IgG and IgM. The Platelia solution may be the best compromise at birth, because IgM detection is the major issue at that time, but the Platelia IgG test exhibits lower sensitivity and could present a lack of reproducibility that would alter the long-

term follow-up needed for ultimately uninfected children. For IgG, test performance must be confirmed by a 1-year follow-up study to validate the accuracy of the test in noninfected children. As many investigators have already stated, on many occasions (19, 20), regardless of the assay chosen, IgG kinetics must be followed with the same test in the same laboratory to avoid misinterpretation.

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