Urinary Disease in 2 Dogon Populations with Different Exposure to Schistosoma haematobium Infection: Progression of Bladder and Kidney Diseases in Children and Adults

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Background. Schistosoma haematobium infection causes severe urinary disease and considerable mortality. The factors that determine disease progression from mild to severe stages are not fully understood.

Methods. Here we describe a cross-sectional epidemiological study of kidney and bladder diseases in 2 Dogon populations with different exposure to S. haematobium infection.

Results. Early and high exposure resulted in more-severe disease, especially among young subjects, without clear evidence of a more-rapid development of immunity. Nevertheless, 50%–60% of subjects of all age classes in both villages showed no evidence of disease. Kidney and bladder disease peaked biphasically among young subjects and adults >25 years old. The first peak corresponded with infections of maximum intensity, whereas the second peak occurred among adults with infections of very low intensity. Kidney disease was correlated with circulating anodic antigen concentration in serum, whereas bladder disease was correlated with egg count and eosinophil cationic protein concentration in urine. Kidney and bladder disease did not correlate. Severe kidney disease was more frequent in certain families.

Conclusions. The frequency of urinary disease is increased by infections acquired early during life, is regulated by strong clinical immunity in certain subjects, and may be dependent on hereditary factors. Kidney and bladder disease may involve different mechanisms of pathogenesis, which may differ between children and adults.

Schistosomiasis is the second leading parasitic disease after malaria [1]. It comprises a group of chronic diseases caused by helminth digenetic trematodes of the Schistosoma genus. S. haematobium inhabits the human vesical venous plexus, where female worms lay eggs. These eggs either pass into the urine or remain trapped in the tissues, particularly in the bladder and ureter walls, where they cause major pathological alterations of the bladder, ureter, and kidneys [2–5]. S. haematobium is an important cause of hematuria and generates considerable morbidity [6–8]. Mortality due to S. haematobium varies between 2.5% and 10% in areas of high endemicity [4, 9] and may reach 50% among patients with advanced hydronephrosis [10, 11].

We describe here a cross-sectional epidemiological study that was performed in 2 Dogon populations in Mali. These populations are almost identical in terms of socioeconomic status, ethnic origin, and economic activity. They differ, however, in their exposure to S. haematobium infection: one population becomes infected at a very young age and exhibits infections of high intensity, and the other becomes infected later...
during life and exhibits infections of low to medium intensity. The aims of the present study were to evaluate resistance and susceptibility to disease in both populations, to identify epidemiological factors that affect disease progression, and to determine whether these factors are the same for kidney and bladder disease.

**SUBJECTS, MATERIALS, AND METHODS**

**Study population and water contacts.** This study was conducted in the populations of 2 Dogon villages (Boul and Ségué) in the district of Bankass, which is 200 km from Mopti in Mali. The populations are stable, with no major migrations of new people. Only Dogons live in the villages. The study was conducted in all residents ≥4 years old (693 subjects in Ségué and 148 in Boul), excepting pregnant women and those who either refused to participate or were traveling (2%). Informed consent was obtained from subjects or their parents or guardians, and all of the experimentation guidelines of the authors’ institutions were followed.

Boul is located up a hillside. Ségué is located up at the top of the hill. All health and educational facilities (a secondary hospital and a primary school) are located in Ségué. A seasonal (June to January) river provides water for drinking, domestic use, and irrigation to both villages. Both villages are a similar distance from the river, which is contaminated by *S. haematobium*–infected snails, and subjects from both villages have water contacts at the same river sites. The inhabitants of Ségué, however, can pump water from a deep well. Small dams have been built in the river, to allow the cultivation of vegetables from October to June. Agriculture is the main economic activity.

Water contacts were recorded from August 1999 to January 2000, when the river was full. Two observers visited water-contact sites from 6:00 AM to 6:00 PM and recorded (by direct observation) which subjects came into contact with the river by sex, age, activity, and exposure time. For each subject, results were expressed as the number of hours of water contacts. These studies indicated a clear difference in water contacts with the river between the subjects from Boul and those from Ségué: the subjects from Boul washed clothes, fetched water (with immersion of part of the body), washed their bodies, drank the water, and performed agricultural activities (field irrigation and watching animals) 2, 67, 4, 4, and 2.5 times more frequently than did the subjects from Ségué, respectively. There was no difference in bathing (activity mostly observed for children) and crossing the river. For the subjects from Ségué, water contacts were mostly observed among those <20 years old, whereas, for the subjects from Boul, water contacts were evenly distributed among all age classes (4–7, 8–11, 12–15, 16–19, 20–24, 25–30, 31–35, 36–40, and >40 years) and were 5–10 times higher among subjects ≥10 years old than they were among the subjects from Ségué.

**Parasitological methods.** *S. haematobium* infections were quantified by counting eggs in urine samples [12] and by measuring concentrations of circulating anodic antigen (CAA), a parasite antigen excreted via the gut of the adult worm, in serum samples [13]. No *S. mansoni* eggs were encountered in feces samples obtained on 2 different days from 200 young subjects. Urine samples were collected on 3–7 consecutive days between 8:00 AM and 2:00 PM. Twenty-milliliter samples were filtered through 25-mm Whatman paper, which was stained with 2% ninhydrin solution; eggs were then counted by microscopic examination (magnification, ×4). Individual infection intensity levels were considered to be the arithmetic mean egg counts of the 3–7 positive and negative samples. To ensure quality control, 10% of the filters were randomly selected and recounted by another microscopist. All subjects were treated with praziquantel after ultrasonographic evaluation.

For CAA titration, 5 mm of fresh venous blood was centrifuged; the serum was immediately placed in liquid nitrogen, transported to the laboratory, and stored in a −80°C freezer. Serum samples were pretreated with trichloroacetic acid, to dissociate immune complexes and to remove interfering components. CAA concentration was determined by a monoclonal antibody–based antigen-capture ELISA. The cutoff (giving a specificity of 98%) for CAA concentration in serum was considered to be the threshold of detection of the assay (40 pg/mL of serum).

Eosinophil cationic protein (ECP) concentration was used as a marker of eosinophilic inflammation. ECP is released into urine from inflammatory granulomas localized in the urinary tract wall and from eosinophils in urine itself. ECP was quantified in urine as described elsewhere [14]. The threshold of detection for ECP concentration was 15 pg/mL of urine.

**Clinical evaluation.** Ultrasonography was performed using a Hellige-Alloka SSD 500 Echo camera and a 3.5-MHz probe. The urinary tract and kidneys were examined according to the methods recommended by the World Health Organization (WHO) [15]. Each subject was given water a half hour before examination; bladder wall thickness was measured at the trigone in subjects, with their bladders entirely filled. Mucosal irregularities, masses, and polyps were classified as recommended by the WHO [15]. Ureter dilatation was evaluated in the retrovesical region. If present, the examination was repeated after micturition, to exclude vesicoureteral reflux.

Kidney length, pyelon depth, and parenchyma thickness were measured with the patient in a prone position. Kidney disease was defined as pyelone dilatation in combination with reduced renal parenchyma thickness and was graded on a scale from 0 (no disease) to 4 (severe disease), as described elsewhere [15].
To evaluate the robustness of the kidney disease scale, grades were plotted against measurements of renal medullar atrophy and renal pelvic dilatation. Shown are the arithmetic means of the measurements made on the right and left kidneys, because the disease can affect either 1 kidney only or both kidneys; bars represent SDs of the mean. Results are presented separately for Boul and Ségué. Note that pelvic dilatation leading to compression of the renal parenchyma, which causes hydronephrosis, was clearly observed in subjects with KD2, KD3, and KD4. Subjects with KD3 and KD4 were pooled, because few subjects had KD4. For definitions of the kidney disease grades, see Subjects, Materials, and Methods.

Figure 1. Correlation between kidney disease grades and medullar atrophy (A) and renal pelvic dilatation (B) in subjects from Boul and Ségué. To evaluate the robustness of the kidney disease scale, grades were plotted against measurements of renal medullar atrophy and renal pelvic dilatation. Shown are the arithmetic means of the measurements made on the right and left kidneys, because the disease can affect either 1 kidney only or both kidneys; bars represent SDs of the mean. Results are presented separately for Boul and Ségué. Note that pelvic dilatation leading to compression of the renal parenchyma, which causes hydronephrosis, was clearly observed in subjects with KD2, KD3, and KD4. Subjects with KD3 and KD4 were pooled, because few subjects had KD4. For definitions of the kidney disease grades, see Subjects, Materials, and Methods.

To take into account both kidneys, these grades were pooled as follows: KD0, no evidence of disease in either kidney; KD1, 1 or both kidneys with grade 1 disease; KD2, 1 kidney with grade 2 disease, and the other kidney with grade 2 disease or lower; KD3, 1 kidney with grade 3 disease, and the other kidney with grade 3 disease or lower; and KD4, 1 kidney with grade 4 disease, and the other kidney with grade 4 disease or lower. These kidney disease grades correlated well with parenchyma thickness and pyelon measurements (figure 1).

Bladder disease was assessed by measuring the increase in bladder wall thickness and by recording bladder wall irregularities, masses, and polyps. Each subject was assigned to 1 of 4 bladder wall thickness classes, as follows: BWT0, 0–3 mm; BWT1, 4 mm; BWT2, 5 mm; and BWT3, >5 mm. The classes were defined so that they would include comparable numbers of subjects.

**Statistical analysis.** Statistical analysis was done using SPSS software. Logistic regression analysis was conducted to determine which variables were associated with the risk of advanced kidney or bladder disease. Data analysis was done on the basis of the stepwise procedure. The affected-kidney phenotype was defined as KD3 + KD4, and the nonaffected-kidney phenotype was defined as KD0, but we also analyzed the KD0 + KD1 phenotype. The affected-bladder phenotype was defined as BWT3, and the nonaffected-bladder phenotype was defined as BWT0. Classes were defined as follows: sex (male or female), age (treated as a linear variable), and village of origin (Boul or Ségué). Because a number of subjects had egg counts, CAA concentrations, and ECP concentrations that were below the respective thresholds of detection, these were treated as discontinuous variables, with 4 classes for each. For egg counts and CAA concentrations, 1 class included all subjects who had counts and concentrations below the thresholds of detection, and the other 3 classes were defined such that they included equal numbers of subjects. ECP concentrations were elevated in most urine samples; therefore, data were distributed among 4 classes comprising equal numbers of subjects. The ranges used for these definitions were as follows: egg count, <0.2, 0.2–4, 5–59, and >60 eggs/20 mL of urine; CAA concentration, <30, 30–224, 225–1853, and >1853 pg/mL of serum; and ECP concentration, <614, 614–4743, 4744–90,865, and >90,865 pg/mL of urine.

**RESULTS**

**Kidney disease in Boul and Ségué.** The frequencies of kidney disease grades were similar (P > .2) among males and females (table 1) but varied with age (P < .001). Analysis of kidney disease prevalence by age produced several observations. First, kidney disease was more severe in Boul than in Ségué among subjects <20 years old. A significant fraction of 4–11-year-old children in Boul (18.8%), but not in Ségué (2.3%), had developed severe kidney disease (KD3 + KD4); among subjects <20 years old, the maximum prevalence of KD3 + KD4 was higher and occurred among younger adolescents in Boul (33% for 12–15-year-old subjects) than in Ségué (7.7% for 16–19-

**Table 1. Kidney disease grades in Ségué and Boul, by sex.**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Ségué Male</th>
<th>Female</th>
<th>Boul Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>KD0</td>
<td>162 (59.5)</td>
<td>176 (64.3)</td>
<td>34 (54.8)</td>
<td>41 (65.1)</td>
</tr>
<tr>
<td>KD1 + KD2</td>
<td>100 (36.8)</td>
<td>90 (32.8)</td>
<td>15 (24.2)</td>
<td>13 (20.6)</td>
</tr>
<tr>
<td>KD3 + KD4</td>
<td>10 (3.7)</td>
<td>8 (2.9)</td>
<td>13 (21.0)</td>
<td>9 (14.3)</td>
</tr>
<tr>
<td>Total</td>
<td>272 (100)</td>
<td>274 (100)</td>
<td>62 (100)</td>
<td>63 (100)</td>
</tr>
</tbody>
</table>

**NOTE.** Data are no. (%) of subjects. For definitions of the kidney disease grades, see Subjects, Materials, and Methods.
year-old subjects). When all age classes were considered, KD3 + KD4 was 5.3 times more frequent in Boul (17.6%) than in Ségué (3.3%) (P < .001). Second, the percentages of children <10 years old who had no evidence of kidney disease (KD0) were similar in Boul (50.0%) and in Ségué (51.5%). When all age classes were considered, prevalence curves for KD0 were similar in Boul and Ségué, as were the global percentages of KD0 (61.9% for Boul and 60.0% for Ségué). Third, figure 2 illustrates 2 interesting findings. One is that the prevalence curves for KD3 + KD4 are biphasic, with 2 peaks—one occurring during childhood/adolescence and the other occurring during adulthood (figure 2B). These peaks are defined by the low prevalence of KD3 among 20–30-year-old subjects; they are clearer when the Boul and Ségué data are analyzed separately, because kidney disease peaks at different ages in Boul (12–15 years) and Ségué (16–19 years). The other interesting finding is that, among young subjects, advanced disease peaked during the age period corresponding to the highest infection intensity levels (figure 2A), and the sharp reduction in the frequency of KD3 + KD4 occurred during the age period in which a major reduction in infection intensity occurred. The second peak of KD3 + KD4 (occurring among 36–40-year-old subjects in Boul and among 31–35-year-old subjects in Ségué) did not correspond to any significant increase in infection intensity. Moreover, the 31–40-year-old subjects with KD3 or KD4 had infections of low intensity (mean ± SD egg count, 0.1 ± 0.1 eggs/mL of urine; mean ± SD CAA concentration, 336 ± 281 pg/mL of serum).

Strong correlation between kidney disease grades and CAA concentration. The effects of age, sex, and infection intensity (as determined by egg counts and ECP concentrations in urine and CAA concentration in serum) on progression from KD0 to KD3 + KD4 were tested by logistic regression. The risk of KD3 + KD4 was associated with age (P = .02) and with the highest CAA concentration class (>1853 pg/mL) (P = .004; odds ratio [OR], 4.8 [95% confidence interval [CI], 1.6–14.0]). ECP concentrations were not associated with aggravation of kidney disease, regardless of whether CAA was included in the model. Interestingly, the model suggested that KD3 + KD4 was associated with decreased egg count (P = .03). These results are further illustrated in figure 3.

The statistical analysis was repeated, this time comparing KD0 + KD1 and KD3 + KD4. It was found that only the highest CAA concentration class correlated with the risk of KD3 + KD4 (P = .002; OR, 5.6 [95% CI, 1.9–16.6]).

Correlation of egg count and ECP concentration with severe bladder disease, which also shows a biphasic pattern in relation to age. Figure 4 shows that the bladder wall thickness classes correlated with the presence of bladder wall irregularities, masses, and polyps. Of the subjects with BWT3 in Boul, 80%, 60%, and 8% had irregularities, masses, and polyps, respectively; thus, BWT3 corresponds with severe bladder disease. Therefore, we focused our analysis on subjects with BWT3. The prevalence of BWT3 (figure 5A) followed a biphasic pattern in relation to age, similar to that of KD3 + KD4 (figure 2). The first BWT3 peak occurred among 12–15-year-old subjects in Ségué and among 16–19-year-old subjects in Boul, and the second peak occurred among 31–35-year-old subjects in Ségué.
Lack of correlation between bladder and kidney disease severity. There was no correlation between kidney disease and either BWT3 or markers of bladder disease (irregularities, masses, and polyps). Nevertheless, a possible correlation between bladder and kidney disease severity was tested using logistic regression that included other covariates (age, sex, and village of origin), and no significant correlation was observed.

**Severe kidney disease is clustered in certain pedigrees.** The distribution of severe cases of kidney disease was analyzed in all 15 pedigrees comprising the Ségé and Boul populations. Because disease occurred mostly in subjects ≥7 years old, only subjects of this age were considered. KD3 and KD4 were found to cluster in certain pedigrees: 37.0% and 29.6% of KD3 and KD4 cases occurred in 2 pedigrees that represented only 17.0% and 16.5% of the overall population, respectively. Furthermore, KD2 was highly prevalent in certain pedigrees in which very few KD3 or KD4 cases occurred; KD3 + KD4:KD2 ratios were

![Figure 3](image)

**Figure 3.** Correlations between kidney disease (left) and circulating anodic antigen (CAA) concentration, between bladder disease (right) and egg count, and between bladder disease and eosinophil cationic protein (ECP) concentration. Data are the arithmetic means of CAA concentrations, ECP concentrations, and egg counts for each kidney disease class and bladder disease class. KD1 and KD2 data and KD3 and KD4 data were pooled, as were BWT0 and BWT1 data and BWT2 and BWT3 data. For definitions of the kidney disease and bladder wall thickness grades, see Subjects, Materials, and Methods.

and among 36–40-year-old subjects in Boul. Analysis of the prevalence of irregularities, masses, and polyps by age showed a different result (figure 5B and 5C). Almost none of the subjects >25 years old had irregularities, masses, or polyps, whereas these pathological manifestations were common among subjects ≤25 years old. Irregularities, masses, and polyps were more frequent among males than among females and in Boul than in Ségé (P < .01).

In the regression analysis (BWT0 vs. BWT3), severe bladder disease was associated with age (P < .001), with the highest egg count class (>60 eggs/20 mL) (P = .004; OR, 3.6 [95% CI, 1.5–8.5]), and with the highest ECP concentration class (>90,865 pg/mL) (P = .004; OR, 3.5 [95% CI, 1.5–8.2]). CAA concentration was not associated with BWT3. These results are further illustrated in figure 3.

![Figure 4](image)

**Figure 4.** Frequencies of bladder wall irregularities, masses, and polyps in infected subjects. Most pathological alterations are observed in subjects with the thickest bladder walls. The proportion of subjects with irregularities, masses, and polyps is shown for each bladder wall thickness class.
Figure 5. A, Biphasic pattern of severe bladder disease revealed by age-specific frequencies. The figure shows the frequencies of subjects in the highest bladder wall thickness class (BWT3 >5 mm), by age. B and C, Age-specific frequencies of bladder wall irregularities and masses. Irregularities and masses are seen principally in subjects <25 years old and are not associated with the second bladder disease peak. The nos. of subjects were as follows: BWT3, n = 60; bladder irregularities, n = 81; and masses, n = 42.

low in these pedigrees (0/6 = 0, 1/6 = 0.16, and 1/6 = 0.16). Conversely, these ratios were >10 times higher in other pedigrees (5/2 = 2.5, 10/6 = 1.6, and 8/2 = 4). This suggests that disease may progress more rapidly from KD2 to KD3 or KD4 in certain pedigrees than in others.

DISCUSSION

The present study was conducted in 2 villages where the populations are almost identical in terms of socioeconomic status, ethnic origin, and economic activity; they differ, however, in exposure to water contaminated by S. haematobium–infected snails. The inhabitants of Ségou have fewer water contacts than do the inhabitants of Bou, as was indicated in Subjects, Materials, and Methods; children <10 years old in Bou have, on average, 5 times more water contacts than do children of the same age in Ségou. Prevalence estimates of S. haematobium infection were 39.5% (on the basis of egg count) and 42.3% (on the basis of CAA concentration) in Ségou and 65.3% and 93.4%, respectively, in Bou. Average infection intensity levels in infected subjects were 3 (for egg count) and 6.4 (for CAA concentration) times higher in Bou than in Ségou. Kidney and bladder diseases were detected at an earlier age and reached higher prevalences in Bou than in Ségou. The possibility that a more-pathogenic strain of schistosome could account for the more-severe disease in Bou can be excluded, because the contaminating sites for both villages are close and connected and because subjects from both villages visit all sites depending on where they work.

We evaluated the possibility that exposure to infection early during life (in Bou) could allow an earlier development of immunity against clinical disease. However, we found no evidence for a clear shift in the peak of disease between Bou and Ségou: only advanced bladder disease was delayed by a few years in Bou relative to that in Ségou, suggesting that higher exposure early during life might have slightly delayed severe bladder disease. Severe kidney disease, however, peaked in children 2–4 years earlier and reached much higher levels in Bou than in Ségou. These observations do not support the hypothesis that exposure early during life allows an earlier development of immunity against clinical disease. Moreover, that KD3 + KD4 frequencies and infection intensity levels sharply regress during the same age period (figure 2) suggest that disease improvement in young subjects might be the direct consequence of worm and egg elimination rather than a specific immunopathologic modulation. On the other hand, the observation that one-half of the inhabitants of Bou do not develop kidney disease is suggestive of a certain capability to resist clinical disease, because the prevalence of infection and the infection intensity levels are high in that village among children and young adults. Nevertheless, it should be remembered that we also found a correlation between disease and infection intensity levels. Note that none of the villagers had ever received treatment for schistosomiasis before the present study was conducted.

The observation of a clustering of severe disease in certain pedigrees and of marked differences in the rate of disease progression from KD2 to KD3 or KD4 among pedigrees suggest that disease development might be influenced by hereditary
factors. Although further studies are required to confirm the existence of a genetic component, this observation is similar to one made in one of our previous studies of hepatic fibrosis in *S. mansoni*-infected populations in Sudan: similar familial correlations and different rates of disease progression between pedigrees were observed and later shown to be attributable to the effects of a major locus at 6q22 [16]. Studies of the effects of chemotherapy have also shown that relapse is more frequent in subjects who had the most-severe disease and suggested that some individuals are more susceptible to disease than others [17, 18].

The shapes of the prevalence curves for severe kidney and bladder disease showed a biphasic pattern in relation to age, with 2 age periods during which advanced disease was frequent separated by a period of milder disease at age 20–25 years. As discussed above, the reduction of disease prevalence between the 2 peaks probably relates to the sharp decrease in infection intensity (figure 2). In this cross-sectional study, it is not easy to interpret the second peak of disease in adults. We cannot exclude the possibility that transmission was more intensive when these adults were young; furthermore, the mechanisms of pathogenesis in adults are unclear. It is difficult to relate severe kidney and bladder diseases in adults to damage caused by eggs, because these subjects do not excrete eggs. Also, the absence of masses and polyps in the adult bladder wall argues against the possibility that eggs are produced but not excreted, although the possibility that eggs, trapped in tissues in locations that will not affect the bladder mucosa, may cause some damage cannot be totally excluded. However, the low ECP concentrations in urine also support the view that egg granulomas, which attract eosinophils, are scarce. In one of our previous studies of hepatic fibrosis in Sudanese patients infected with *S. mansoni*, we also noticed differences between severe hepatic periportal fibrosis among young adolescents and adults >25 years old [19]. We suggested that the changes that occur around puberty may be a key factor in disease regression among adolescents, possibly because they modify a patient’s immunological reactivity to parasite antigens. A second key factor was identified as the decrease in infection intensity. Interestingly, in the present study (as in the Sudanese study [19]), severe disease in adults occurred in subjects who excreted low numbers of eggs and had a low worm load. Thus, unlike in children, disease in adults is probably not the consequence of massive aggression by a large number of parasite antigens. Instead, it may result from chronic, uncontrolled inflammation that can be triggered by just a few eggs and worms. It is also possible that a cofactor, such as a urinary bacterial infection, causes more-severe disease in the urinary tract, which is already the site of intense inflammation caused by schistosome antigens.

The present study showed differences between bladder and kidney disease. Kidney disease is related to ureter disease [4, 5, 20], and a clear correlation was observed between ureter and kidney disease (data not shown) when ureters could be visualized; this was, however, not always possible. Bladder disease did not correlate well with kidney disease [4, 10, 21], suggesting different mechanisms of pathogenesis. This conclusion is also supported by our analysis of the association of organ disease with infection intensity and markers of inflammation: bladder disease correlates strongly with egg production and ECP concentration, as has been previously reported [14]. Thus, eggs, and the eosinophilic inflammation they cause, are likely to play an important role in bladder disease [14, 22–24]. Kidney disease, however, correlates with CAA production by adult worms and not with egg count or ECP concentration. This finding further supports the view that kidney disease might be more dependent on wound antigens or biologically active molecules secreted by worms than on excreted eggs. The importance of CAA and ECP in kidney lesions has already been described by Van Mark et al. [25] and Sobh et al. [26]. Other studies have shown that kidney disease responds less well to treatment than does bladder disease, suggesting that kidney disease is not just the consequence of bladder and/or ureter disease [17, 18, 27–30]. Bladder disease, which is caused by eggs, responds better to treatment than does kidney disease.

As discussed above, the observation that advanced disease is more frequent in certain families suggests that some genetic factors play roles in susceptibility to disease. Analysis of the genetic control of bladder and kidney diseases may indicate to what extent the mechanisms of pathogenesis differ between these 2 diseases.

References


