

1 WU and KI polyomaviruses in respiratory, blood and urine samples from renal transplant  
2 patients

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21 **Abstract**

22 *Background:* It is suggested that immunosuppression due to transplantation might be a risk  
23 for human polyomavirus KI (KIPyV) and WU (WUPyV) infection. Most of the publications  
24 report data about stem cell transplant patients, little is known about these virus infections in  
25 renal transplant patients.

26 *Objectives:* To study the presence of KIPyV and WUPyV in upper respiratory, plasma and  
27 urine samples from renal transplant patients. To analyse clinical and personal data.

28 *Study design:* 532 respiratory, 503 plasma and 464 urine samples were collected from 77  
29 renal transplant patients. KIPyV and WUPyV were detected by nested and quantitative real-  
30 time PCR. Patient and clinical data from medical records were analyzed.

31 *Results:* KIPyV was detected in respiratory, plasma and urine samples from 14.3 %, 3.9 %  
32 and 4.1 % of renal transplant patients. WUPyV was found in respiratory and plasma  
33 specimens from 9.1 % and 5.3 % of the patients. Significant association was revealed between  
34 the detection of KIPyV and WUPyV and the time of samples collection and the age of the  
35 patients. KIPyV was presented in respiratory and plasma sample at the same time. KIPyV was  
36 detected in plasma samples from two patients and in urine samples of three other patients  
37 providing also KIPyV positive respiratory samples at the same time. No clinical consequences  
38 of KIPyV or WUPyV infection were found.

39 *Conclusion:* Although no clinical consequences of KIPyV and WUPyV infections were found  
40 in renal transplant patients, it is suggested that renal transplantation might result in higher  
41 susceptibility or reactivation of these infection.

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## 46 **1. Background**

47 KI and WU polyomaviruses (KIPyV and WUPyV) were discovered in respiratory  
48 samples from children suffering from respiratory symptoms in 2007 [1, 2]. Subsequent  
49 studies using PCR methods revealed the presence of viral DNA in many different samples:  
50 respiratory, blood, stool, cerebrospinal fluid, lymphoid tissue, lung [3] and urine samples [4,

51 5]. Seroepidemiological studies showed that both viruses are widespread, the seropositivity of  
52 KIPyV and WUPyV are 55-100 % in the adult population [6-9]. Since similarly high  
53 seropositivity was also found among young children, it is suggested that primary infections by  
54 KI and WU viruses occur during childhood [6, 7]. This is also strengthened by the finding that  
55 the viruses were detected more frequently in samples from children [10]. Respiratory and/or  
56 fecal-oral transmission is suggested for both KI and WU viruses [11]. Despite the numerous  
57 prevalence data published, little is known about the pathogenesis of KIPyV and WUPyV, and  
58 none of them is associated with any disease. Primary infection by WU and KI viruses might  
59 be mild or asymptomatic. The higher prevalence in samples (respiratory samples, lymphoid  
60 tissues, blood and urine) from immunocompromised patients suggests that  
61 immunosuppression might result in higher susceptibility or reactivation of KIPyV and  
62 WUPyV [3-5, 12-15]. Transplantation associated with immunosuppression might be a risk for  
63 these infections, since higher frequencies of these viruses were found in stem cell transplant  
64 patients compared with other immunocompromised groups. Increased pathogenic potential  
65 was also suggested, even if not higher prevalence, but higher viral loads were detected in  
66 immunocompromised patients [16]. Most of these studies focused on haematopoietic cell  
67 transplant patients, and we published previously data about renal transplant patients [4].

68 In this study we detected KIPyV in respiratory, blood and urine samples from renal  
69 transplant patients, frequency was 14.3, 3.9 and 4.1%. WUPyV was found in 9.1% of  
70 respiratory and 5.3% of blood samples. No clinical consequence was found.

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## 72 **2. Objectives**

73 Based on the above mentioned data it is suggested that renal transplant patients  
74 receiving immunosuppressive therapy might be more susceptible for these infections or  
75 reactivation of these viruses may occur. To examine the prevalence of these viruses in renal

76 transplant patients, to find potential site of viral replication and/or latency the presence of  
77 KIPyV and WUPyV was studied by PCR in respiratory, blood and urine samples from renal  
78 transplant patients from transplantation until 18 month. Clinical and personal data of the  
79 patients were analyzed to study the potential pathogenic role of KIPyV and WUPyV.

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### 81 **3. Study design**

#### 82 *3.1. Patients and samples*

83 The study was approved by Regional and Institutional Ethics Committee of University  
84 of Debrecen.

85 Throat swab, plasma (from EDTA blood samples) and urine samples were collected  
86 from 77 renal transplant patients receiving kidney between September 2008 and September  
87 2012 at University of Debrecen as described previously [4]. Samples were collected from  
88 patients visiting the outpatient clinic of the renal transplant centre at University of Debrecen.  
89 Table 1. summarizes the number of samples and the data of the patients. It was preferred to  
90 collect blood and urine sample together with respiratory samples at the same time, but beside  
91 532 respiratory samples, 503 plasma and 464 urine samples were provided. Samples were  
92 collected 1-16 times from the following number of patients: 2, 10, 9, 3, 5, 3, 6, 9, 11, 9, 4, 2,  
93 1, 2, 1 patients at the 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, 13, 14 or 16 time points.

94 Immediately after collection, nucleic acid was isolated from 200  $\mu$ L of each sample  
95 using High Pure Viral Nucleic Acid Kit (Roche, Switzerland) according to the manufacturer's  
96 instructions. DNA eluted in 50  $\mu$ L volume was stored at -20 °C until use.

97 Clinical and laboratory data were obtained from medical records.

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#### 99 *3.2. Nested and real-time PCR for WUPyV and KIPyV*

100 Nested PCR was performed as described previously [17]. For quantitative detection of  
101 KIPyV and WUPyV DNA, multiplex real-time PCR (qPCR) was carried out with primers and  
102 probes published previously [14] in a final volume of 50  $\mu$ L using TaqMan Universal PCR  
103 Master Mix in an Applied Biosystem 7500 real-time PCR instrument (Applied Biosystems,  
104 USA). Standard curves generated by PCR amplification of 10-fold dilutions of plasmids  
105 containing WU and KI polyomavirus DNA were used for absolute quantification. All samples  
106 were tested by nested and qPCR, 10  $\mu$ L nucleic acid was amplified in each PCR. Nested PCR  
107 positivity was confirmed by sequencing if qPCR was negative.

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### 109 3.3. Statistical analysis

110 Difference in frequency for categorical variables was analysed by Fisher's exact and  $\chi^2$   
111 test. For continuous variables Mann-Whitney test was applied. Logistic regression was used  
112 to analyze the relationship between KIPyV and/or WUPyV detection and different variables.  
113 Cox regression models were performed to analyse the risk factors for detection of KI or WU  
114 viruses, including age, month of examination, sex, CMV reactivation, respiratory symptoms,  
115 leukopaenia, monocytopenia, neutropenia, leukocytosis, monocytosis and neutrophilia.  
116 Analyses were performed using SigmaPlot version 10.0 (Systat Software, Inc., USA) and  
117 XLSTAT 2014 software (Addinsoft, USA) , p value < 0.05 was considered significant.

118

## 119 4. Results

120 In our study group 20.7 % of the renal transplant patients (16/77) provided KIPyV  
121 and/or WUPyV positive respiratory samples during the examination period. KIPyV DNA was  
122 detected in 17 respiratory samples (17/532; 3.2 %) taken from 11 patients (11/77; 14.3 %).  
123 Three patients had two or three positive respiratory samples within 7-21 days, and one patient  
124 provided a KIPyV positive sample 158 days after his first positive sample. WUPyV was

125 found in 8 respiratory samples (8/532; 1.5%) from 7 patients (7/77; 9.1%). Two WUPyV  
126 positive samples were collected within 14 days from one patient. Both viruses was detected in  
127 one sample at the same time (Table 2.). The frequency of KIPyV was significantly higher  
128 than that of WUPyV in positive respiratory samples (17/24 vs. 8/24;  $p=0.019$ ). Only three  
129 respiratory samples were qPCR negative and nested PCR positive, but sequencing proved the  
130 presence of the polyomaviruses. Additionally, one of these patients had KIPyV qPCR positive  
131 plasma, and one provided KIPyV positive urine at the same time. Although KIPyV viral loads  
132 were higher in respiratory samples (range:  $2.2E+02$ - $1.04E+07$  copies/mL; median:  $1.39E+03$   
133 copies/mL), it was not significant difference ( $p=0.056$ ) comparing with WUPyV viral loads  
134 (range:  $4.6E+02$ - $2.18E+04$  copies/mL; median:  $7.31E+02$  copies/mL). Significant difference  
135 was found regarding the sample collection time points between KI and/or WU viral DNA  
136 positive and negative samples ( $p=0.001$ ; Table 2.). Although the number of positive  
137 samples/patients is low, patients provided WUPyV positive respiratory samples mainly during  
138 summer (6/7), seasonality was not apparent for KIPyV (Figure 1.). Logistic regression  
139 showed significant relationship between the presence of KIPyV and/or WUPyV in respiratory  
140 samples and the age of the patients (odds ratio 0.963, 95% CI 0.935-0.991,  $p = 0.01$ ) Age was  
141 a significant risk factor for detecting KIPyV and/or WUPyV (hazard ratio 0.963 CI 0.936-  
142 0.991  $p=0.009$ ). KIPyV was detected in samples from patients 20-59 years of age, while  
143 WUPyV was found also in respiratory samples from a patient < 20 years of age. All patients >  
144 60 years of age provided respiratory samples negative for KI and WU virus, respectively  
145 (Figure 2.). Most of the samples positive for KIPyV and WUPyV were collected 1-5 months  
146 after transplantation (Figure 3A), logistic regression significant relationship between the  
147 presence of KIPyV and/or WUPyV in respiratory samples and the time of sampling (odds  
148 ratio 0.992, 95% CI 0.988-0.996,  $p<0.001$ )..

149 Relationship between detection of KI and/or WU viruses in respiratory samples and  
150 sex, CMV reactivation, leukopaenia, monocytopenia, neutropenia, leukocytosis,  
151 monocytosis, neutrophilia was not revealed. Mild, upper respiratory symptoms were recorded  
152 only for three patients provided KIPyV/WUPyV positive respiratory samples, but similar  
153 symptoms were also existed when 26 negative samples were collected from patients (3/24 vs.  
154 26/508;  $p=0.11$ ).

155 Plasma samples were also collected together with the 19 respiratory samples positive  
156 for KIPyV and/or WUPyV from 13 patients. In two out of these 18 plasma samples KIPyV,  
157 and in one sample WUPyV was also detected, while the respiratory samples of these three  
158 patients were KIPyV positive. Therewith, in three additional plasmas out of the 503 samples  
159 WUPyV DNA was found, and in one of them KIPyV was also detected at the same time. The  
160 respiratory samples of these three patients were negative for viral DNA at that time point.  
161 Altogether, 7.9 % of the patients (6/76) had viraemia, the frequency of KIPyV and WUPyV  
162 was 0.6% and 0.8%, respectively (3/503 and 4/503). The viral loads were low,  $\leq 260$  copies  
163 /mL. Significant difference was found regarding the sample collection time points between KI  
164 and/or WU viral DNA positive and negative samples ( $p=0.006$ ; Table 2.). Figure 3B shows  
165 the time points of sampling for KIPyV and WUPyv positive samples. KIPyV and/or WUPyV  
166 viraemia was significantly more frequent in patients providing also positive respiratory  
167 sample at the same time (3/19 vs. 3/484;  $p=0.0009$ ). Logistic regression model found  
168 association between KIPyV/WUPyV viraemia and the presence of these viruses in respiratory  
169 samples at the same time (odds ratio 0.008 95 % CI 0.015-0.441,  $p=0.004$ ). Not any other  
170 parameter (age, sex, CMV reactivation, leukopaenia, monocytopenia, neutropenia,  
171 leukocytosis, monocytosis, neutrophilia) was associated with KIPyv/WUPyV viraemia based  
172 on Cox regression model.

173 At the time point of WU or KI viral DNA positive respiratory samples 6 patients  
174 provided also urine samples, out of which KIPyV was detected in three samples (3/11). All  
175 other urine samples were negative for KIPyV and WUPyV, the frequency of KIPyV in urine  
176 samples was 0.6 % (3/464; Table 2.). Positivity was confirmed with sequencing of the nested  
177 PCR product. Viruria was revealed in 4.1 % of the patients, and viraemia was not detected at  
178 that time point. Figure 3C shows the time points of sampling.

179

## 180 **5. Discussion**

181 Previous studies with haematopoietic transplant patients suggest that  
182 immunosuppression related to transplantation may result in higher frequencies of KIPyV and  
183 WUPyV infection [14, 16, 18, 19]. At the same time, little is known about renal transplant  
184 patients [4]. In this study the presence of KIPyV and WUPyV was studied in upper  
185 respiratory tract, plasma and urine samples from 77 renal transplant patients from  
186 transplantation until 18 months after it. KIPyV was revealed in all sample types, WUPyV was  
187 found in respiratory and plasma samples. KIPyV was detected in respiratory samples from  
188 14.3 % of the patients, while 9.1 % of the patients provided WUPyV positive respiratory  
189 sample during this study. The frequency in respiratory samples was 3.2 % and 1.5 % for  
190 KIPyV and WUPyV, respectively. KIPyV was detected in 0.8-17.8 % of respiratory  
191 specimens from stem cell transplant patients meaning 0.8-22 % KIPyV prevalence in these  
192 patients. Lower prevalence of WUPyV was reported, 0-7 % in stem cell transplant patients.  
193 [14, 16, 18, 19]. Our data from renal transplant patients are in accordance with these  
194 publications.

195 We found that most of the KIPyV and WUPyV positive respiratory samples were  
196 provided 1-5 month after transplantation. The detection of the viruses was significantly  
197 associated with the time point of sample collection. All KIPyV positive respiratory samples



198 were collected from patients at 21-44 years of age (median 29 years), while WUPyV was  
199 detected mainly in patients at 12-68 years of age (median 45 years), the difference was not  
200 statistically significant ( $p=0.058$ ). Seroprevalence studies found high KIPyV and WUPyV  
201 seropositivity in young children similar to that of the adults suggesting that primary infections  
202 occur mainly during childhood [6, 7]. Additionally, in most of the publications higher PCR  
203 prevalence data for KIPyV and WUPyV were reported in respiratory samples compared with  
204 adults [3, 20, 21]. These findings suggest that the detected KIPyV and WUPyV infections  
205 might be reactivations or re-infections.

206         Seasonality of KIPyV in respiratory samples was not found in this study. Although the  
207 number of the positive samples is low, most of the WUPyV positive samples were provided  
208 during summer. A previous study also found a small peak of WUPyV detection during  
209 summer, however they found WUPyV throughout the year [22]. Further studies are required  
210 to determine whether seasonality of WUPyV occurs.

211         In our study group respiratory symptoms were not associated with the detection of  
212 KIPyV and/or WUPyV in the upper respiratory specimens. Only three patients provided  
213 KIPyV (1 patient) or WUPyV (2 patients) positive respiratory samples had mild, upper  
214 respiratory symptom (coughing, sore throat without fever). Not any risk factor was associated  
215 with KIPyV/WUPyV detection, although the available clinical data were analysed (CMV  
216 reactivation, haematologic clinical data, symptoms of patients).

217         In a study high viral loads of KIPyV in respiratory samples from paediatric stem cell  
218 transplant patients were found suggesting higher pathological role for KI virus in these  
219 patients, but not for WUPyV [16]. In our study KIPyV was not only more frequent, but the  
220 viral loads also were higher than that of WUPyV in respiratory samples. Although the KIPyV  
221 viral loads in respiratory samples from our patients were lower than was detected in paediatric  
222 patients [16], it may refer to active viral replication.

223 We detected KIPyV and WUPyV in plasma samples from 3.9 % and 5.2 % of patients.  
224 Previous publications also reported that KIPyV and WUPyV can be detected in blood [22]. At  
225 the same time, in a study with stem cell transplant patients KIPyV and WUPyV was not  
226 detected in plasma samples suggesting that these viruses have no effect on post-transplant  
227 clinical course [23]. Recently, detection of KIPyV antigen in lung and spleen tissues has been  
228 published. It was also determined that CD68+ cells in lung harbour KIPyV antigens, hence  
229 alveolar macrophages may be infected by KIPyV [24]. In our study we found co-existence of  
230 KIPyV in respiratory and blood specimen from two patients, and one patient provided KIPyV  
231 positive respiratory and WUPyV positive blood samples at the same time point. The viral  
232 loads in all plasma specimens were low, there is no evidence for active virus replication in  
233 blood circulation, but it also cannot be excluded. Blood cells might be the site of active  
234 infection, non productive infection, the site of latency, as it was hypothesized for CD68+ cells  
235 [24]. We detected KIPyV in urine samples from three patients provided also KIPyV positive  
236 respiratory specimen at the same time. Analysis of clinical data did not reveal any risk factor  
237 or association with clinical parameters.

238 Different hypotheses arise from the co-detection of KIPyV in respiratory and blood or  
239 urine specimens. The respiratory tract infection may enter the blood circulation directly or by  
240 circulating blood derived cells, as was suggested [24]. By the blood circulation the virus can  
241 reach other organs, maybe kidney, urinary tract. It cannot be excluded that KIPyV/WUpyV  
242 detected in respiratory, blood and urine samples from adult, renal transplant patients  
243 originated from reactivation of latency, or latent infections (blood, urinary tract) were found,  
244 if these viruses are able to establish latency.

245 In conclusion, we detected KIPyV and WUPyV in respiratory specimens from renal  
246 transplant patients with a frequency similar to published data about other  
247 immunocompromised patients. Age and the time point of sample collection were associated

248 with detection. KIPyV and WUPyV were also found in plasma samples, co-occurrence of  
249 KIPyV in blood and respiratory specimens were detected. Presence of KIPyV in urine  
250 samples were also revealed, these patients had KIPyV positive respiratory samples at the  
251 same time. No clinical consequences of KIPyV or WUPyV infection were found. Further  
252 studies are required to clarify many questions in connection with these viruses.

253

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259

#### 260 **Competing interests:**

261 The authors have no competing interest.

#### 262 **Ethical approval :**

263 The study was approved by Regional and Institutional Ethics Committee of University of  
264 Debrecen (number: 2740-2008, 2917-2009, IX-R-052/00876-2/2012).

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