Hypervirulent *Klebsiella pneumoniae*: An update on epidemiology, detection and antibiotic resistance

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Received: November 10, 2023 • Accepted: November 20, 2023

ABSTRACT

*Klebsiella pneumoniae* is a major human pathogen as it is responsible for various infections. In the past years hypervirulent *K. pneumoniae* (hvKP) emerged and disseminated worldwide. In this review a summary will be given about epidemiology, detection and antibiotic resistance of hypervirulent *K. pneumoniae*. A common feature of hypervirulent *K. pneumoniae* is a combined expression of several virulence factors. A mucoviscous phenotype, certain capsulare serotypes (e.g.: K1, K2, K28, K47, K63) together with additional genetic markers namely, *magA*, *rmpA* or *iucABCD*, are needed in combinations to achieve the hypervirulent pathotype. Plasmid coded virulence determinants are also detected, that indicates horizontal gene transfer of hypervirulence factors in *K. pneumoniae*.

Interestingly, infections caused by hypervirulent *K. pneumoniae* occur usually in the community in otherwise healthy people, and during these infections multiple infection sites are detected. Clinical pictures include both invasive infections and local abscess formation. Pyogenic liver abscess is the most frequently reported clinical manifestation and abscess formation in brain, spleen and lung are also diagnosed. Additionally, meningitis, endophthalmitis, trombophlebitis, pneumonia can also develop.

In the early reports, hypervirulent *K. pneumoniae* strains exhibited enhanced virulence but these were susceptible to commonly used antibiotics. However, recently KPC, VIM, NDM and OXA-48 carbapenemase producing hypervirulent *K. pneumoniae* strains are increasingly reported, furthermore, well-known high-risk *K. pneumoniae* clones (e.g.: ST11, ST147, ST307) can develop hypervirulent pathotype, that poses an even more alarming challenge.

KEYWORDS

*Klebsiella pneumoniae*, hypervirulence, hypermucoviscosity, antibiotic resistance, hvKP

INTRODUCTION

*Klebsiella pneumoniae* is a member of the Enterobacteriaceae family and it is a normal inhabitant of human gastrointestinal tract, however, *K. pneumoniae* is also responsible for various human infections [1]. *K. pneumoniae* is divided into classical and hypervirulent pathotypes, based on phenotypic appearance and genetic markers. The classical *K. pneumoniae* pathotype occurs usually in healthcare settings, and it is a major nosocomial pathogen as it causes infections in hospitalized, older patients with certain level of immunosuppression. These infectious diseases include urinary tract infection, bloodstream infection, intra-abdominal infection and wound infection. By contrast, hypervirulent *K. pneumoniae* (hvKP) pathotype causes infections usually in the community, in otherwise healthy people, and multiple infection sites are typical for these infections. Interestingly, the main clinical appearance of these type of infections is abscess formation in liver, brain and spleen [2–4].

Genome of *K. pneumoniae* has a certain flexibility and variability to take up and to accumulate antibiotic resistance genes as well as virulence factor determinants [1]. Development of multiresistant strains has an ongoing tendency. It has been earlier...
established that *K. pneumoniae* belongs to ESKAPE pathogens, and this group comprises well-known bacterial pathogens that are capable to develop multidrug-resistance and can cause high-number of difficult-to-treat infections [5, 6].

Hypervirulent *K. pneumoniae* was first reported in the 1980s and 1990s in Taiwan and in other Asian countries, however, recently a broader dissemination is seen [3, 4]. In the early reports hypervirulent *K. pneumoniae* demonstrated an enhanced virulence and it was isolated mainly from pyogenic liver abscess. Later on, hypervirulent *K. pneumoniae* was identified in different clinical manifestations namely, in spleen abscess, community acquired meningitis and bacteremia [3].

A common feature of hypervirulent *K. pneumoniae* is a hypermucoviscous phenotype that develops through production of different virulence determinants namely, magA and rmpA. Interestingly, in the early reports of hypervirulent *K. pneumoniae* the virulence determinants were located chromosomally however, recently virulence plasmids were detected that harbour different hypervirulence determinants (e.g.: *rmpA*) [3]. This is an important feature, because hypervirulence determinants can spread through horizontal gene transfer, that results in further dissemination [3, 4].

Another interesting feature of hypervirulent *K. pneumoniae* is the antibiotic susceptibility. During the early reports hypervirulent *K. pneumoniae* strains were susceptible to commonly used antibiotics, but this has changed recently. The majority of currently isolated hypervirulent *K. pneumoniae* strains show multidrug resistance, that is explained mainly by acquired resistance determinants (e.g.: beta-lactamases) [2–4].

Generally, antibiotic resistance in *K. pneumoniae* is well described, as resistance to beta-lactams, fluoroquinolones and aminoglycosides are reported from all over the World [1]. Resistance to beta-lactams in *K. pneumoniae* is explained mainly by production of different beta-lactamases namely, extended-spectrum beta-lactamases (ESBLs) and carbapenemases. Among ESBLs the most frequently reported enzymes are TEM, SHV, CTX-M types, while among carbapenemases *K. pneumoniae* carbapenemase (KPC) from Ambler class A, metallo-beta-lactamases (e.g.: VIM, NDM, IMP types) from Ambler class B and OXA types (e.g.: OXA-48, and OXA-48-like enzymes) from Ambler class D are detected in *K. pneumoniae* [7–11]. Fluoroquinolone resistance in *K. pneumoniae* is explained by chromosomal mutations in gyrA and topoisomerase IV coding genes namely, in gyrA, gyrB, parC and parE. Additionally, plasmid-mediated quinolone resistance determinants (PMQRs) have been also reported, that enhance development of fluoroquinolone resistance. PMQRs include Qnr determinants, Aminoglycoside acetyltransferase (6’)-Ib-cr variant enzyme [AAC(6′)-Ib-cr], QepA and OqxAB efflux pumps [12–15]. Resistance to aminoglycosides develops mainly through enzymatic modifications by aminoglycoside acetyltransferase, aminoglycoside adenylation transferase, and aminoglycoside phosphotransferase. Another mechanism in aminoglycoside resistance is the modification of ribosomal binding site through 16s rRNA methylase [16].

The aim of this review is to summarize current epidemiology, detection, clinical features and antibiotic resistance of hypervirulent *K. pneumoniae*.

**VIRULENCE DETERMINANTS ASSOCIATED WITH HYPERVIRULENT *K. PNEUMONIAE***

Phenotypic and genetic markers are detected in hypermucoviscous and hypervirulent *K. pneumoniae*. Hypermucoviscosity can be detected by string test however, a hypervirulent type requires additional markers [17].

**String test**

The string test is a well-known test to detect mucoviscous phenotype of *K. pneumoniae*. This routine method is performed on fresh colonies from an overnight culture on blood agar with a loop. Detection of viscous string greater than 5 mm in length, when the colony was taken with a loop wire is considered a positive result [17–19].

**Capsular polysaccharide serotype**

Capsule polysaccharide is a virulence determinant of *K. pneumoniae* and at least 78 capsular serotypes are already described [20, 21]. Interestingly, serotype K1 and K2 are the most frequently associated with hypervirulent *K. pneumoniae*. Notably, invasive infections like pyogenic liver abscess were associated with K2 serotype in 42.9% of cases while K1 serotype was present in 23.8% of cases [22]. K2 serotype was detected in 68.75% of *K. pneumoniae* strains exhibiting hypermucoviscosity in China [23]. In South Korea, *K. pneumoniae* strains showing hypermucoviscosity were commonly detected as serotype K1 and K2 [24].

Interestingly, K1 and K2 serotypes demonstrate more resistance to phagocytosis and intracellular killing by macrophages, compared to other serotypes of *K. pneumoniae*, that contribute to higher virulence [25–27]. During early detection of hypervirulent *K. pneumoniae* strains, the K1 and K2 serotypes were the most frequently reported however, recently the hypervirulent *K. pneumoniae* is associated with other capsular serotypes namely, K5, K16, K20, K28, K54, K57, K63 [3] (Table 1).

**RmpA (regulator of the mucoid phenotype A)**

RmpA is a major regulator of capsule production, that leads to hypermucoviscosity and it is associated with enhanced virulence [28]. Altogether three *rmpA* genes were described in hypervirulent *K. pneumoniae* strains namely, *p-rmpA* and *p-rmpA2* (plasmid located) and c-*rmpA* (chromosomal located) [29, 30]. Based on whole genome sequence data, several reports demonstrated that hypervirulent *K. pneumoniae* strains commonly harbour *rmpA* gene. Interestingly, this feature was found in different countries and in different infection sites [31–34].
MagA (mucoviscosity associated gene A)

MagA was described as a major factor in hypercapsular phenotype in *K. pneumoniae*, however, it has been clarified, that *magA* is a K1 serotype allele of *wzy* gene in *cps* gene cluster, therefore, *magA* is named as *wzy* K1 [17, 35]. A strong correlation (83% of detected *K. pneumoniae* strains) was found between *magA* gene positivity and hypervirulent *K. pneumoniae* strains obtained from pyogenic liver abscess [36]. Moreover, significant association was found between K1 capsular serotype and *magA* gene positivity in hypervirulent *K. pneumoniae* [22].

Siderophores

*K. pneumoniae* produces several siderophores (aerobactin, salmochelin, enterobactin, and yersiniabactin) that take up iron from the environment. Iron is an important metal, that plays a cardinal role in growth and virulence of *K. pneumoniae* [37, 38]. It has been demonstrated that hypervirulent *K. pneumoniae* exhibits a 6-10 fold enhanced siderophore activity compared to that of classical *K. pneumoniae* strains [37, 39].

Aerobactin, salmochelin and yersiniabactin are plasmid coded determinants and can be transferred through horizontal gene transfer [31, 40, 41]. Interestingly, hypervirulent *K. pneumoniae* mainly produces aerobactin, that was found in over 90% siderophore activity of investigated strains [37]. An *iucABCD* operon encodes aerobactin and this determinant was found in high prevalence among hypervirulent *K. pneumoniae* [22, 33, 34, 37, 42–47] (Table 1) (Fig. 1).

### ANTIBIOTIC RESISTANCE IN HYPERVIRULENT *K. PNEUMONIAE*

During early reports of hypervirulent *K. pneumoniae* antibiotic susceptible strains were isolated [3, 48]. However, recently several studies have detected antibiotic resistance in

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**Table 1. Markers and virulence determinants associated with hypervirulent *K. pneumoniae***

<table>
<thead>
<tr>
<th>Virulence determinants</th>
<th>Gene</th>
<th>Marker</th>
<th>Reference</th>
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<tr>
<td><strong>Hypermucoviscosity</strong></td>
<td>capsular types: K1, K2, K5, K16, K20, K28, K54, K57, K63</td>
<td>[3]</td>
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<td><em>magA</em></td>
<td>mucoviscosity associated gene A</td>
<td>[64]</td>
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<tr>
<td><em>rmpA</em></td>
<td>regulator of the mucoid phenotype A gene</td>
<td>[64]</td>
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<tr>
<td><em>rmpA2</em></td>
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<td>[64]</td>
<td></td>
</tr>
<tr>
<td><strong>Siderophores</strong></td>
<td><em>iucABCD</em></td>
<td>aerobactin siderophore biosynthesis</td>
<td>[64]</td>
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<td><em>peg344</em></td>
<td>putative transporter</td>
<td>[64]</td>
<td></td>
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<tr>
<td><em>peg-589</em></td>
<td>putative carboxymuconolactone decarboxylase family</td>
<td>[64]</td>
<td></td>
</tr>
<tr>
<td><em>iroB</em></td>
<td>salmochelin siderophore biosynthesis</td>
<td>[64]</td>
<td></td>
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<tr>
<td><em>irp2</em></td>
<td>yersiniabactin siderophore biosynthesis</td>
<td>[64]</td>
<td></td>
</tr>
<tr>
<td><em>ybt</em></td>
<td>yersiniabactin</td>
<td>[3]</td>
<td></td>
</tr>
<tr>
<td><strong>Other</strong></td>
<td><em>terB</em></td>
<td>tellurit resistance</td>
<td>[64]</td>
</tr>
</tbody>
</table>

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**Fig. 1. Virulence determinants of hypervirulent *K. pneumoniae***
different sequence types (STs) of hypervirulent K. pneumoniae. The majority of multidrug-resistant and hypervirulent K. pneumoniae strains are identified in China however, in the past years a worldwide dissemination was seen [49, 50]. K. pneumoniae has several multidrug-resistant and high-risk clones that has been reported in the past decades namely, ST11, ST147, ST307. However, hypervirulent pathotype has just been recently detected in these high-risk clones [49]. Interestingly, the earlier detected hypervirulent and antibiotic susceptible K. pneumoniae STs (e.g. ST23, ST1265, ST1797) can take up resistance plasmids, therefore multidrug-resistant and hypervirulent strains can evolve also in this process [49] (Fig. 2).

ST11 is among the most frequently detected multidrug-resistant and hypervirulent K. pneumoniae. ST11 is a well-known antibiotic resistant high-risk clone and its expression of hypervirulence has been also reported. In a study in China colistin resistant hypervirulent K. pneumoniae ST11 strains were isolated. In these strains colistin resistance was mediated mainly by mgrB mutation (ISKpn26, or Q30Stop), phoQ mutation (D150G), pmrD mutation (D80G), pmrB mutations (R256G, D313N) [50].

In 2016 during an outbreak in China, hypervirulent carbapenem resistant K. pneumoniae caused ventilator-associated pneumonia. The detected five strains belonged to ST11 clone, and each carried approx. 170 kbp pLVPK-like virulence plasmid [51].

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In a study in China a hypervirulent carbapenem resistant K. pneumoniae ST23 was detected. It was isolated from a sputum sample of patient, who was treated in an intensive care unit in Sichuan province, China. This K. pneumoniae strain harboured blaKPC-2 on a plasmid [52].

A carbapenem resistant K. pneumoniae ST23 was detected from bloodstream infection of a patient treated at an intensive care unit in Sichuan province, China. This K. pneumoniae strain was further investigated, and it carried blaKPC-2 Resistance gene and rmpA virulence determinant. According to multilocus sequence typing of this strain, it belonged to ST36 [53].

Altogether 163 K. pneumoniae-induced pyogenic liver abscess cases were investigated in China between 2016 and 2017. Twelve K. pneumoniae strains exhibited multidrug-resistance. K1 and K2 serotypes were detected in 40.5% and 19% of strains, whereas 30.7% of strains showed mucoviscosity. Diverse virulence determinants were detected in hypervirulent K. pneumoniae strains namely, iroN (24.5%), magA (45.4%), genes encoding for aerobactin, rmpA, ybtA, (68.7–100%). Among tested K. pneumoniae strains ST23 was the most frequently detected, however, additional STs (e.g.: ST3507, ST3508 and ST3509) were also reported [54].

A NDM-5 carbapenemase producing hypermucoviscous K. pneumoniae was isolated from sputum sample in China. This K. pneumoniae harboured a pVirSCNJ1 plasmid, that carried rmpA virulence gene, that determines hypervirulent pathotype. Interestingly, this strain belonged to ST29, and the blaNDM-5 was located on a IncX3 conjugative plasmid [55].

Two carbapenem resistant IMP-4 producing hypervirulent K. pneumoniae were isolated from blood and sputum samples in an intensive care unit in China. Both K. pneumoniae strains belonged to ST65 and K2 capsular serotype. Virulence factors indicating hypervirulence pathotype, namely, rmpA/rmpA2, iucA, and iroN were also detected in these strains. One K. pneumoniae strain demonstrated tigecycline non-susceptibility, that was conferred by frame-shift mutation in the TetR/AcrR transcriptional regulator [56].

A carbapenem resistant hypervirulent K. pneumoniae was recoved from a sputum sample of patient, who was treated in an intensive care unit in China. This K. pneumoniae strain belonged to ST592 and had K57 capsular serotype. Interestingly, this strain harboured a plasmid that was carrying blaKPC-2. This carbapenem resistant K. pneumoniae strain harboured a virulence plasmid that possessed IncFIBK, IncHI1B replications and carried rmpADC, rmpA2, iucABC-iutA and iroBCDN virulence determinants [57].

A multidrug-resistant hypervirulent K. pneumoniae was isolated from a lung infection in China. This strain belonged to ST25 and K2 serotype. This K. pneumoniae strain harboured a plasmid carrying iucABC-iutA operon, that codes

![Fig. 2. Multidrug-resistant and hypervirulent K. pneumoniae clones](Image)
aerobactin virulence determinant. Interestingly, this strain harboured another plasmid, that was carrying blaKPC-2. During conjugation assays it has been demonstrated that both plasmids can be transferred, indicating a possible joint horizontal genetranfer of blaKPC-2 carbapenemase and hypervirulence determinant [58].

A multidrug-resistant K. pneumoniae was isolated from a blood sample of a haematological patient in China. This strain belonged to ST464 and co-produced NDM-1 and KPC-2 carbapenemases, and additionally it exhibited resistance to tetracyclin, fluoroquinolones, tigecycline and fosfomycin. Hypervirulent pathotype of this K. pneumoniae strain was confirmed by serum resistance, and in a Galleria mellonella infection model [59].

A hypermucoviscousus K. pneumoniae was isolated from a neck abscess in China. This strain belonged to ST1049 and co-produced NDM-1 and OXA-48 and expressed different virulence markers indi-

A survey in Qatar revealed carbapenem resistant and hypervirulent K. pneumoniae. Ten strains carried rmpA and/ or truncated rmpA2. Strains carrying carbapenem resistance and hypervirulence determinants belonged to ST231 and ST383. A K. pneumoniae ST383 strain carried blaNDM on IncHI1B plasmid, that co-harboured rmpA, rmpA2, aerobactin (iucABCD-iutA) virulence determinants of hypervirulent pathotype [60].

A study in Germany detected two hypervirulent K. pneumoniae ST420 strains that were isolated from a swab and a bloodculture of a patient. In these strains a chromosomally integrated virulence plasmid was detected and increased siderophore production as well as hyper-
mucoviscosity were also seen. Interestingly, both genomes of K. pneumoniae ST420 strains carried the “yersinia-
bactin lineage 9” that was associated with K. pneumoniae integrative conjugative element 3 (ICEKp3). According to bioinformatic analysis insertion of the hypervirulence plasmid into K. pneumoniae chromosome was mediated by IS5 family sub-group IS903, designated as IKKpn74 [62].

A 2 year survey in Ireland detected twenty-eight carba-
penem resistant hypervirulent K. pneumoniae ST23 strains. All these K. pneumoniae strains were isolated from different clinical samples, including rectal swab, faeces (as colonizer) as well as from blood culture samples (from invasive in-
fec
tions). All hypervirulent K. pneumoniae strains produced OXA-48 and expressed different virulence markers indi-
cating the hypervirulent pathotype namely, rmpA2, iuc, iro, ybt [63].

Several other reports demonstrate multidrug-resistant and hypervirulent K. pneumoniae in different countries, namely, in India, France, Germany, Italy and Iran [70–76]. The most frequently reported antibiotic resistance de-

CHARACTERISTICS OF INFECTIONS CAUSED BY HYPERVIRULENT K. PNEUMONIAE

First infections caused by hypervirulent K. pneumoniae were reported in 1980s and 1990s in Southeast Asia [4]. These early reports described a special clinical picture that was dominated by community acquired pyogenic liver abscess, and it was associated with severe complications such as meningitis and endophthalmitis [3, 77–80]. Later on, several other reports from Asia described role of hypervirulent K. pneumoniae in different infections. A report from China demonstrated that 90.9% of the pathogens causing pyogenic liver abscesses were hypervirulent K. pneumoniae [43]. In different studies conducted in South Korea and Taiwan a high prevalence of hypermucoviscousus and hypervirulent K. pneumoniae strains were isolated from invasive infections, namely, 42.2% of K. pneumoniae strains isolated from bacteremia had hypermucoviscosity phenotype in South Korea [81], while 88.8% of K. pneumoniae isolates from community acquired extrahepatic abscesses exhibited hypermucoviscosity phenotype in Taiwan [82] and addition-

ally, 41.5% of community acquired bacteriaemia caused by K. pneumoniae belonged to hypervirulent K. pneumoniae in Taiwan [83].

During infections caused by hypervirulent K. pneu-

moniae multiple infection sites and metastatic spread are detected [2, 84]. These interesting and specific features are characteristics of hypervirulent K. pneumoniae, that are normally not seen in infections of classical K. pneumoniae. Pathomechanism of multiple infection sites can be explained by the local invasion of hypervirulent K. pneumoniae, breaking into different tissues or another explana-
tion can be a primary infection site in the human body that serves as a source for subsequent bacteriaemia and distant infections. This later type can be called as a metastatic spread [2].

The most frequently reported infection is pyogenic liver abscess that has been described mainly in Taiwan and China, however, recently several reports from USA, and from European countries have revealed its clinical signifi-
cance [85–96].

Some specific features of pyogenic liver abscess caused by hypervirulent K. pneumoniae have been described: it is almost always monomicrobial infection, while in case of a non-hypervirulent K. pneumoniae infection a pyogenic liver abscess is almost always polymicrobial [97, 98]. During an infection caused by hypervirulent K. pneumoniae metastatic complications occur more frequently compared to that of other K. pneumoniae infections [45, 79]. Interestingly, pyogenic liver abscess through hypervirulent K. pneumoniae develops in patients with normal hepatic and biliary func-
tions, by contrast, other liver infections are linked to hepatic impairment and to different pathological disorders. [45]. Regional thrombophlebitis is reported in aprox. 30% of pyogenic liver abscess infections. Additional intra-
abdominal infection caused by hypervirulent \textit{K. pneumoniae} includes splenic abscess \cite{99}.

Pneumonia is a typical infection caused by classical \textit{K. pneumoniae} among hospitalized patients and it usually occurs as ventilator-associated pneumonia. However, recent reports demonstrated that hypervirulent \textit{K. pneumoniae} is a major causative agent of community acquired pneumonia in Taiwan, and in these cases certain complications and clinical pictures develop namely, empyema, lung abscess, respiratory failuer, bilateral lobar involvement and septic schock \cite{100}.

Endogenous endophthalmitis develops in patients after a bacteraemia caused by hypervirulent \textit{K. pneumoniae}. It is a very severe infection of eyes, and bilateral involvement can occur between 13 and 25\% of cases. Furthermore, this infection can lead to irreversible blindness \cite{101}.

Among central nervous system infections, hypervirulent \textit{K. pneumoniae} is a major pathogen in community acquired meningitis, and certain complications (e.g.: septic shock) can develop. Further central nervous system infections caused by hypervirulent \textit{K. pneumoniae} include brain abscess, subdural empyema and epidural abscess \cite{102}.

**CONCLUSION**

In the past years hypervirulent \textit{K. pneumoniae} strains emerged and disseminated worldwide. Hypervirulent \textit{K. pneumoniae} causes infections usually in healthy people and multiple infection sites are typical. The clinical picture is very diverse, however, the most frequent clinical presentation is abscess formation in liver, spleen and brain. Additionilly, meningitis, endophthalmitis and other invasive infections (e.g.: bacteraemia, sepsis) can also develop. Noteworthy to mention, that classical infections of \textit{K. pneumoniae} (e.g.: urinary tract infection, ventilator-associated pneumonia, catheter associated infections) are caused less frequently by hypervirulent \textit{K. pneumoniae}.

During the early detection of hypervirulent \textit{K. pneumoniae}, it has been demonstrated that these strains pose enhanced virulence alongside with an antibiotic susceptible feature. However, recently multidrug-resistant hypervirulent \textit{K. pneumoniae} strains evolved through the acquisition of different resistance genes (\textit{bla}_{VIM}, \textit{bla}_{NDM}, \textit{bla}_{OXA-48}, \textit{bla}_{CTX-M-15}). Furthermore, multidrug-resistant high-risk \textit{K. pneumoniae} clones, namely ST11, ST15, ST147, ST307, are also capable to take up virulence plasmids that carry \textit{rmpA}, ybt.

<table>
<thead>
<tr>
<th><strong>Table 2. Sequence types, virulence determinants and antibiotic resistance associated with hypervirulent \textit{K. pneumoniae}</strong></th>
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<tbody>
<tr>
<td><strong>Country</strong></td>
</tr>
<tr>
<td>China</td>
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<tr>
<td>ST11, ST23, ST65, ST86, ST437</td>
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<td>ST11</td>
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<td>ST147</td>
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<td>ST893</td>
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hypervirulence determinants, therefore development of hypervirulent pathotype is possible in these high-risk *K. pneumoniae* clones.

**Conflict of interest:** The author is a member of the Editorial Board of the journal. Therefore he did not take part in the review process in any capacity and the submission was handled by a different member of the editorial board.

**ACKNOWLEDGEMENT**

B.K. was supported by Janos Bolyai Scholarship (BO/00286/22/5) of the Hungarian Academy of Sciences.

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