การติดตามเฝ้าระวังแบบแผนความไวรับต่อยาปฏิชีวนะ และยีนดื้อยาของเชื้อ
เอสเชอริเชีย คอลัย ในระหว่างระบบการผลิตสุกร

นายกิตติธัช ลักษณ์สมยา

วิทยานิพนธ์นี้เป็นส่วนหนึ่งของการศึกษาตามหลักสูตรปริญญาวิทยาศาสตรดุษฎีบัณฑิต
สาขาวิชาพยาธิชีววิทยาทางสัตวแพทย์ ภาควิชาพยาธิวิทยา
คณะสัตวแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย
ปีการศึกษา 2560

Monitoring of antibiogram and resistance gene profiles among *Escherichia coli* in pig production system

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A Dissertation Submitted in Partial Fulfillment of the Requirements

for the Degree of Doctor of Philosophy Program in Veterinary Pathobiology

Department of Veterinary Pathology

Faculty of Veterinary Science

Chulalongkorn University

Academic Year 2017

Thesis Title	Monitoring of antibiogram and resistance gene profiles among *Escherichia coli* in pig production system
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กิตติธัช ลักษณ์สมยา : การติดตามเฝ้าระวังแบบแผนความไวรับต่อยาปฏิชีวนะ และยีนดื้อยาของเชื้อ*เอสเชอริเชีย คอลัย* ในระหว่างระบบการผลิตสุกร (Monitoring of antibiogram and resistance gene profiles among *Escherichia coli* in pig production system) อ.ที่ปรึกษาวิทยานิพนธ์หลัก: รศ. น.สพ. ดร. ณุวีร์ ประภัสระกูล, อ.ที่ปรึกษาวิทยานิพนธ์ร่วม: ศ. น.สพ. ดร. เผด็จ ธรรมรักษ์, ดร. ธนิษฐา ฉัตรสุวรรณ, หน้า.

เชื้อแบคทีเรียดื้อยาที่พบในฟาร์มสุกรเชื่อว่าเป็นแหล่งของการกระจายเชื้อดื้อยาที่สำคัญในทางสาธารณสุข ข้อมูลจากการศึกษาในฟาร์มสุกรทั้งระบบจะทำให้ทราบถึงชนิดของการดื้อยาและปัจจัยที่เกี่ยวข้องกับการเกิดการดื้อยา การศึกษานี้มีวัตถุประสงค์เพื่อสำรวจชนิดและคุณลักษณะทางฟีโนไทป์และพันธุกรรมเฉพาะของเชื้อดื้อยาต่อยาปฏิชีวนะ 18 ชนิด ในระบบการผลิตสุกรแบบ ณ ช่วงเวลาหนึ่ง (cross sectional study) และการศึกษาแบบติดตาม (longitudinal study) ตั้งแต่แรกเกิดจนถึงส่งโรงฆ่าสัตว์ รวมถึงผลของการใช้สารต้านจุลชีพชนิดฟลาโวมัยซินต่อระดับการดื้อยาในฟาร์ม โดยใช้เชื้อ*เอสเชอริเชีย คอลัย* ในอุจจาระเป็นตัวแทน ผลการศึกษาพบว่าเชื้อที่ได้จากสุกรระยะขุนมีอัตราการดื้อยาแบบหลายชนิด (multiple drug resistance) จากฟาร์มสุกรในประเทศไทยอยู่ในระดับที่สูงมาก แต่เมื่อพิจารณาจากชนิดของยาปฏิชีวนะที่เชื้อดื้อแบ่งได้เป็นสองแบบ คือ เชื้อดื้อยาที่พบได้ทั่วไป ได้แก่การดื้อต่อยากลุ่มแบต้าแลคแตม และเตตร้าซัยคลิน ซึ่งพบทั้งในฟาร์มที่ใช้ยาปฏิชีวนะผสมอาหาร (อะมอกซิซิลินและ ไทอะมูลิน)และฟาร์มที่ไม่ใช้ยา อีกแบบหนึ่งคือเชื้อดื้อยาแบบที่เกี่ยวข้องกับการใช้ยาปฏิชีวนะผสมอาหาร โดยเฉพาะเชื้อที่สร้างเอนไซม์ extended-spectrum beta-lactamase (ESBL) และการดื้อต่ออะมิโนไกลโคไซด์พบมากขึ้นอย่างมีนัยสำคัญ ในขณะที่ฟาร์มที่ใช้ยาปฏิชีวนะในรูปแบบการฉีดแต่ไม่ใช้ในการผสมอาหารไม่พบการเพิ่มขึ้นของการดื้อยากลุ่ม ESBL และอะมิโนไกลโคไซด์ รูปแบบการดื้อยา (antibiogram) ของเชื้อมีความสอดคล้องกับลักษณะทางพันธุกรรมจากยีนบนโครโมโซมและพลาสมิด โดยเฉพาะกลุ่ม ESBL จะพบยีน $bla_{CTX-M-1}$ และ/หรือ $bla_{CTX-M-9}$ การศึกษาแบบติดตามแบ่งเป็น 5 ระยะได้แก่ ระยะแรกเกิดถึงหย่านม ระยะหลังหย่านม ระยะอนุบาล ระยะขุน และเนื้อสุกรในโรงฆ่าสัตว์ การศึกษาแบบติดตามยืนยันว่าการใช้ยาปฏิชีวนะผสมอาหารชนิดอะมอกซิซิลินร่วมกับไทอะมูลินทำให้เกิดปริมาณและรูปแบบการดื้อยาเช่นเดียวกับการศึกษาแบบ ณ ช่วงเวลาหนึ่ง แต่พบเชื้อการดื้อยากลุ่ม ESBL และ อะมิโนไกลโคไซด์เพิ่มสูงขึ้นอย่างมีนัยสำคัญในช่วงระยะอนุบาลและระยะขุนช่วงต้น แต่อัตราการดื้อยานี้จะลดลงในช่วงก่อนเข้าโรงฆ่า ในขณะมีการดื้อต่อที่ยาปฏิชีวนะอื่นๆในระดับน้อยถึงปานกลางซึ่งไม่พบความสัมพันธ์กับระยะการเลี้ยง จากการติดตามชนิดของสายพันธุ์ (clone type) ด้วยวิธี multilocus sequence typing และ วิธี pulsed-field gel electrophoresis พบว่า เชื้อ*เอสเชอริเชีย คอลัย* สายพันธุ์ ST10 พบในฟาร์มมากที่สุดแต่ไม่พบในเนื้อสุกร ชนิดของสายพันธุ์จากเนื้อสุกรส่วนใหญ่แตกต่างจากที่พบในฟาร์ม มีเพียงสายพันธุ์ ST 44 117 638 ที่พบได้จากทั้งสุกรมีชีวิตและเนื้อสุกร แต่เชื้อเหล่านั้นไม่แสดงคุณลักษณะการดื้อต่อยากลุ่ม ESBLและอะมิโนไกลโคไซด์ ในฟาร์มที่ใช้ยาฟลาโวมัยซินเข้มข้น 10 พีพีเอ็ม ผสมอาหารพบการดื้อต่อยาในกลุ่ม ESBLP จะลดลงในระยะสุกรอนุบาลและสุกรขุนอย่างในระดับ 20.0-23.3% เมื่อเปรียบเทียบกับฟาร์มที่ให้ยาอะมอกซิซิลินและไทอะมูลินผสมอาหาร จากการศึกษาในห้องปฏิบัติการยืนยันได้ว่าฟลาโวมัยซินความเข้มข้น 8 และ 16 ไมโครกรัมต่อมิลลิลิตรสามารถลดการส่งผ่านของยีน $bla_{CTX-M-1}$ และ $bla_{CTX-M-9}$ ได้ 10 เท่า จากการศึกษาปริมาณและคุณลักษณะของเชื้อดื้อยาทั้งระบบการผลิตทำให้ทราบถึงปัจจัยการจัดการ ชนิดของยาผสมอาหาร และระยะการเลี้ยง มีผลต่อการเพิ่มขึ้นของเชื้อดื้อยา รวมถึงรูปแบบการดื้อยาจำเพาะที่ควรมีการเฝ้าระวังในฟาร์ม การศึกษานี้ไม่พบความสัมพันธ์ด้านการถ่ายทอดสายพันธุ์เชื้อดื้อยาที่สำคัญโดยเฉพาะเชื้อที่ดื้อต่อกลุ่มเซฟาโรสปอรินในรุ่นที่สาม และเชื้อที่ดื้อต่อยากลุ่มอะมิโนไกลโคไซด์จากสุกรมีชีวิตในฟาร์มไปสู่เนื้อสุกรในโรงฆ่าสัตว์

ภาควิชา พยาธิวิทยา	ลายมือชื่อนิสิต .....
สาขาวิชา พยาธิชีววิทยาทางสัตวแพทย์	ลายมือชื่อ อ.ที่ปรึกษาหลัก .....
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5575404131 : MAJOR VETERINARY PATHOBIOLOGY

KEYWORDS: ANTIMICROBIALS / ESCHERICHIA COLI / PIG FARM / RESISTANCE GENE

KITTITAT LUGSOMYA: Monitoring of antibiogram and resistance gene profiles among *Escherichia coli* in pig production system. ADVISOR: ASSOC. PROF. DR. NUVEE PRAPASARAKUL, D.V.M., Ph.D., D.T.B.V.P., CO-ADVISOR: PROF. DR. PADET TUMMARUK, D.V.M., Ph.D., D.T.B.T., INS. DR. TANITTHA CHATSUWAN, Ph.D., pp.

The antimicrobial resistant (AMR) bacteria in pig farms have been believed as an important source in food chain with public health concern. While some studies suggested transmission of AMR from pigs to humans may occur, but there was still needing to combine high resolution genomic data analysis with systematically collected epidemiological evidence to reconstruct patterns of AMR transmission between pigs and humans. The objectives of this study were to determine the occurrence and characterization the AMR phenotypes against 18 antimicrobials in pig producing system in both cross-sectional from fattening and longitudinal studies from newborn to slaughtering pigs and to evaluate the effect of flavomycin to reduce AMR rate in pig farms. The commensal enteric *Escherichia coli* were used as a proxy to estimate the overall extent of AMR. Altogether, Multiple Drug Resistance (MDR) *E. coli* were highly found from fattening pigs. Interestingly, some resistant phenotypes (β-lactam resistance and tetracycline resistance) were commonly detected in the isolates either from farm with and without antimicrobial usage in feed however extended-spectrum β-lactamase producing (ESBLP) and aminoglycosides resistance were detected in farms with antimicrobial usage in feed (amoxicillin and tiamulin) while farm using only antimicrobial by injection for therapeutic purpose had ESBLP and aminoglycosides resistance in very low rate. All ESBLP *E. coli* relatively possessed $bla_{CTX-M-1}$ and/or $bla_{CTX-M-9}$ genes. For longitudinal study, AMR situations were monitored through pigs producing system in 5 periods; pre-weaning, nursery, growing, fattening and slaughtering periods. ESBLP *E. coli* and aminoglycosides resistance significantly increased in nursery and growing periods in the farm with antimicrobial usage in feed (amoxicillin and tiamulin) but the resistant rate decreased in slaughtering. For clonal typing analysis, the most common clonal type of *E. coli* in live pigs was ST10 that were non-ESBLP strain and could not find in all meat samples. ST44, 117 and 638 shared between both live pigs and meats but none was ESBLP or aminoglycosides resistant strains. The resistant isolates recovered from pig meat largely differed from those detected in the feces of the same live pigs sampled during the production period. *In vitro,* flavomycin at 8 μg/ml and 16 μg/ml concentrations could reduce the conjugative rates of the plasmids carrying $bla_{CTX-M-1}$ and $bla_{CTX-M-9}$ 10 times. *In vivo*, use of flavomycin at 10 ppm in feed could reduce antimicrobial resistance rates of third generation cephalosporins resistance phenotypes and genotypes at 20.0-23.3% in nursery and growing periods. This study provided the insight of AMR type, distribution and their characteristics in pig farms in the relation of antimicrobial use and supported the indication of flavomycin mixed in feed. The molecular typing limited to identify a direct clonal relationship between critically important antimicrobial resistant strains, especially the third generation cephalosporins resistance and aminoglycosides resistance in meat and those found in the corresponding live animals during their production cycle. Further work is required to identify the source of resistant *E. coli* in pig meat following slaughter.

Department:	Veterinary Pathology	Student's Signature	.....
Field of Study:	Veterinary Pathobiology	Advisor's Signature	.....
Academic Year:	2017	Co-Advisor's Signature	.....
		Co-Advisor's Signature	.....

ACKNOWLEDGEMENTS

I would like to express my deepest gratitude to my principal advisor, Assoc. Professor Dr. Nuvee Prapasarakul, who provides many good opportunities in my Ph.D. study, scientific thinking, English writing skill and livelihood guidance with the good attitudes. This dissertation would have not been success without his assistance.

I would like to thank Professor David J. Hampson for supporting the scientific idea and teaching writing skill.

I gratefully thank Professor David J. Hampson, Dr Sam Abraham and all stuff in Murdoch University for invaluable and wonderful experiences in Australia.

I would like to acknowledge my co-advisors and committee members, Professor Padet Tummaruk, Dr. Tanittha Chatsuwan, Assistant Professor Channarong Rodkhum, Associate. Professor Kampon Kaeoket, Dr. Taradon Luangtongkum, Associate Professor Chanwit Tribuddharat for their suggestions and encouragements to fulfill this dissertation.

I would like to thank Associate. Professor Dr. Chanwit Tribuddharat, Mr. Thanakorn Watcharasupat and all Dr Chanwit's stuff for supporting 454 sequencing technique and high-through put multilocus sequence typing technique.

Thanks are due to Dr. Thanittha Chatsuwan and Mrs. Sirirat Luk-in for their helpful guides for blaCTX-M gene detection and plasmid conjugation technique.

Many thanks also go to all staffs of Department of Microbiology, all committees of Veterinary Pathobiology Program, Faculty of Veterinary Science, Chulalongkorn University for all assistance and recommendations.

Most of all, I would like to express my deepest gratitude to my parents and my brother who love me endlessly and always provide every good thing for my life. This doctoral dissertation is dedicated to my father and my mother.

Finally, I would like to acknowledge Graduate School, Chulalongkorn University for the Scholarships supporting this dissertation including the Chulalongkorn University 90th Year Anniversary Ratchdapisek Somphot Endowment Fund, Chulalongkorn University, Diagnosis and Monitoring of Animal Pathogen Research Unit (DMAP RU), Center of Excellence in Emerging and Re-emerging Infectious Diseases in Animals (EIDAs), Research and Researcher for Industrials Funds (PHD5610005), and Huvepharma (Thailand) Ltd.

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Chapter I: Introduction

Antimicrobial resistance (AMR) in bacteria is an important critical problem in veterinary public health. The major cause of emerging resistance is due to the abuse of antimicrobials in a shorter period and lower concentration than their recommendations (Paphitou, 2013). Regarding to pig farming, intensive use of antimicrobials is a common tool for reducing infectious diseases throughout production cycle except at late fattening period prior to slaughtering. Moreover, use of antimicrobials without prescription is also practically carried out by farmers and consultant decisions beneath their chemical company propaganda. Thus, increasing of resistance bacteria in pig farms has been reported, especially among gastrointestinal tract pathogens (Nogrady et al., 2006). These findings can enhance the chances of farm animal to human transmission (Szmolka and Nagy, 2013). Relation between bacteria from human and animal sources was based on genetic conserveness. For example, sharing of *E. coli* sequence type (ST) 7 [by multilocus sequence typing (MLST)] contained $bla_{CTX\text{-}M\text{-}1}$ located on plasmid incompatibility group II was recovered from pig and human feces (Leverstein-van Hall et al., 2011a). However, there has still been lack of data uncovering the background or concrete relationship between bacteria sourced from pigs and human.

By susceptibility determination surveillance, the most gastrointestinal bacteria resistant to first generation antibiotics such as beta-lactam, quinolone and tetracycline group used in production cycle, and fecal carriage of multiple drug resistant (MDR) bacteria seem to be very common in our areas (Prapasarakul et al., 2010). Thus, whether the researchers are going to a right solution or not if they still find the problems by report of ambiguous resistant bacteria. Although all parameters in farms have been investigated their associations to the crisis, there was still lack of a specific parameter benefit to strategic management which reduces emerging of bacteria presenting multidrug resistance. Numerous reports have still reported rising of MDR in livestock and meats without evident connection (Fernandes et al., 2016; Rehman et al., 2017; Strom et al.,

2018). Pig production cycle in Thailand is a major section where antibiotics are highly used for both therapeutic and feed additive indication. Previously, reports described the critical AMR situation were investigated in certain particular period of production such as piglets (Prapasarakul et al., 2010) or fattening (Lugsomya et al., 2017) and these were randomized sampling in any farms in the area of study. This study hypothesized that some antimicrobial resistance traits might be a consequence of routine use of antimicrobials and inappropriate management but some of them became the common traits in our country. However, in one production cycle, type and frequency of AMR bacteria in fecal carriage may vary due to duration of consumption and growing period. We believe that some resistance strains were able to be maintained their crisis AMR traits and transferable neither to slaughter period nor to the next batch of pig cycles. By contrast, certain reported resistance isolates may reconsider as a bacterial persistent, wild type-liked, that was able to be something blindfold for monitoring and surveillance. In the study, we attempt to fully investigates antimicrobial situation through pig cycles by using phenotype and genotype characterization following by longitudinal monitoring (newborn piglets to slaughtering) of antimicrobial resistance genes by using fecal carriage *Escherichia coli* in healthy in standard pig farms with the historical different antibiotic use.

Chapter II: Literature reviews

Escherichia coli (E. coli)

Escherichia coli (*E. coli*) is a rod bacterial member in family *Enterobacteriaceae,* phylum *Gamma Proteobacteria.* These facultative anaerobic bacteria are commonly found along alimentary tract which act as both commensal and pathogen in human and animals. Pathogenic *E. coli* are tentatively characterized by hemolysin together with particular genes encoding enterotoxins such as STa ,STb, Stx2e (Souza et al., 2010) causing colibacillosis or edema diseases in piglets to nursery pigs, respectively (Prapasarakul et al., 2010). The pigs infected with enterotoxigenic *E. coli* (ETEC) and enterohemorrhagic *E. coli* (EHEC) generally suffer with mild diarrhea to bloody diarrhea (Casey and Bosworth, 2009). In pig industries, the diseases can be controlled by oral antimicrobial use such as colistin, amoxicillin, tetracycline, or enrofloxacin in the recommendation of farm consultants or empirical use (Straw et al., 2006). Not only the problem in veterinary field but the emerging of resistance and new genotype *E. coli* contaminated in food chain has been sporadically reported with high mortality and cause of kidney failure and death in human (Mehrgan and Rahbar, 2008).

The antimicrobial resistance mechanisms

Four general mechanisms of antibiotic resistance in *E. coli* are controlled by the expression of specific genes, all of which are listed as below,

1. Enzymatic modification

Antibiotic target modification is reported as two major mechanisms dividing as inactivation and modification enzymes. β-lactamases is an inactivate enzymes gain by chromosomes and plasmids, which hydrolyze beta-lactam structure molecule of antibiotic. β-lactamases can be gained from the particular genetic element called “transposons”. One of the most importance of beta-lactamase is metallo-β-lactamases (MBLs), which is the inactivate enzyme for resistance to imipenem, new-generation

cephalosporins and penicillins. MBLs also inactivates the inhibitors of β-lactamases (clavulanic acid) but is still sensitive to aztreonam (Thomson and Bonomo, 2005; Vatopoulos, 2008). Esterase is another hydrolysis enzyme which inactivates to erythromycin via esterase II protein expressed from *ereB* gene by hydrolyse lactone ring of erythromycin A and oleandomycin (Kim et al., 2002).

On the other hand, the group of modification enzymes called "transferase" can resist to streptogramin, macrolides, or rifampicin by binding adenylyl, phosphoryl, or acetyl groups of the antibiotic molecules. In addition, aminoglycosides are overwhelmed by the enzymes; phosphoryltransferases (APHs), adenylyltransferases (ANTs), and acetyltransferases (AACs). These enzymes reduce affinity of the drug at 30S ribosomal binding site (Strateva and Yordanov, 2009). Genes encoding antimicrobial resistance phenotypes are transferred by transposons (Martinez and Baquero, 2002). For chloramphenicol resistance, bacteria produce chloramphenicol transacetylase that acetylates hydroxyl groups of chloramphenicol cause a low affinity at ribosomal 50S subunit binding (Tolmasky, 2000).

2. Active drug efflux

Efflux pump is a function for exporting antibiotics away from cell resulting in of antibiotic uptake (Hawkey, 1998). The mechanisms are associated with interactions between the inner and outer membrane components of the tri-partite multidrug efflux pump AcrAB-TolC. Especially, the efflux pump of *E. coli* is AcrAB-Tolc, which associates with fluoroquinolones, β-lactams, tetracycline, chloramphenicol and trimethoprim resistance (Walsh, 2004).

3. Outer membrane (OM) permeability alteration.

In general, the outer membrane of gram-negative bacteria contains phospholipids layer and a lipid A layer. Some part of outer membrane composition reduces drug uptake to a cell and transfer through the OmpF in *E. coli.* In low outer membrane permeability, small hydrophilic molecules (β-lactams and quinolones) can cross the OM only through

porins but aminoglycosides and colistin cannot be transferred to the cell through porins. Therefore, self-promoted uptake to bacterial cell is initiated by binding to lipopolysaccharides of the outer side of the OM (Lambert, 2012). Acquired resistance is characteristic of high resistance to almost all aminoglycosides (especially to tobramycin and gentamicin) (Walsh, 2004).

4. Deoxyribonucleic acid (DNA) synthesis interference.

Enzymatic modifications during DNA synthesis are the major mechanism of resistance by DNA interference. There are two common enzyme groups, which co-operate during DNA synthesis, DNA gyrase (topoisomerase II : *gyrA* and *gyrB*) (Levy, 1998) and topoisomerase IV : *parC* and parE). Mutations in genes *gyrA* and *parC* cause replication failure resulting low affinity of fluoroquinolones to bacteria DNA modified-DNA complex. The most common resistance mechanism is mutation on *E. coli gyrA.* (Martinez-Martinez et al., 1998).

The emerging and acquiring of resistance gene

Management error in veterinary field is an important inducer for bacterial resistance. Use of subtherapeutic antibiotic in farm or inappropriate administration time can induce emerging of AMR bacteria. In general, the emerging and acquiring of antibiotic resistance are induced from two mechanisms (Normark and Normark, 2002).

1. Alteration in the genome via mutation

In common, a mutation usually affect only one class of antibiotics (Roberts, 2003) except the mutation of efflux and impermeability associated genes. The mutation of chromosomal genes associating antibiotic resistance are showed in Table 1.

Table 1 Antibiotics and their target gene of mutation

Antibiotics	Target of mutations	References
Aminoglycosides	16S rRNA	(Melancon et al., 1988)
Fluoroquinolones and quinolones	DNA gyrase DNA topoisomerase IV	(Fluit et al., 2001) (Drlica and Zhao, 1997)
Macrolide	23S rRNA	(Sigmund et al., 1984)
Rifampicin	β-subunit of RNA polymerase	(Telenti et al., 1997)
Sulfonamide	Dihydropteroate synthetase (*dhps* gene)	(Huovinen et al., 1995)
Tetracyclines	16S rRNA	(Ross et al., 1998)
Trimethoprim	dihydrofolate reductase (*dhfr* gene)	(Powell et al., 1991)
β-lactams	Penicillin binding protein (PBP)	(Fluit et al., 2001)

2. Acquisition resistance gene from mobile genetic element (Horizontal transfer)

Horizontal transfer gains by mobile genetic element (MGE) set as a free interchangeable unit between the intraspecies and interspecies bacteria in microenvironmental system (Roberts, 2003). The horizontal transfer can be occurred via self-replicating plasmids, bacteriophages, transposons, integrons and pathogenicity islands (Fluit et al., 2001; Salyers and Amabile-Cuevas, 1997) (Table 2).

Table 2 Mobile genetic element and their specific characters

Mobile genetic elements	Characters of mobile genetic element	Role in resistance gene spreading
Integron	DNA segment composes 3 components: integrase, promoter and integration site of gene	Form the cassette of different resistance gene (gene in cassette can use promoter for expression)
Self-transmissible conjugative plasmid	Self-replicating element which carries the gene for conjugal transfer	Transfer resistance gene
Mobilizable plasmid	Self-replicating element which can use conjugal apparatus from self-transmissible plasmid for transferring	Transfer resistance gene
Transposon	Mobile from DNA segment to another place in the same cell	Transfer resistance gene between chromosome and plasmid
Bacteriophage	Viral vector for DNA transferring	Transfer resistance gene

Horizontal gene transfer in *E. coli*

Generally, most of genes encoding resistance traits can be transferred horizontally cause wide-spreading of the genetic element transfect to other bacteria in microenvironment (Angulo et al., 2004a; Angulo et al., 2004b; Deng et al., 2011; Yao et al., 2011). By horizontal transmission, the extra-intestinal *E. coli* in farm animals possess class 1 integrons and *iss, tsh* and *colV* gene via using conjugative process that encode

certain virulence and resistance as well as those of commensal *E. coli.* (Nogrady et al., 2006). Thus, occurrence of integron and certain other MGE have usually been investigated for explanation of AMR situation between food animals and consumers.

Integrons are genetic elements that participate in site-specific recombination system of bacterial DNA. The elements majorly play a role in spreading antibiotic resistance genes in a clinical and animal farm setting. Integrons function as gene caption and expression system containing three main structures; (i) integration site (*attI*) for site specific insertion of gene cassettes (Partridge et al., 2000), (ii) integrase encoding gene for excising and arranging the gene cassette (Collis et al., 1993) and (iii) promoter session for expression (Levesque et al., 1995). Integrons are highly variable in type, number and gene function. (Recchia and Hall, 1995).

Class 1 integron is widely disseminated in bacterial strains within family *Enterobacteriaceae*. In animals and human. Class I integrons is composed of specific recombination site *attI1*, integrase gene *intI1* and the promoter Pc. At each end of class 1 integron gene possesses CSs site. 5´-CS with *intI1* gene (Hall and Vockler, 1987) and promoter towards to integration site and also 3´-CS with *qacE*Δ*1 (Paulsen et al., 1993)*(Paulsen et al., 1993) *sul1, orf5* (Stokes and Hall, 1989).

E. coli identification and strain typing

E. coli has much potential to harbor in wide-range host and environment. As its role to host is both resident and transient microorganism. It is needed not only to identify species but realize its role is also a need for controlling and management. *E. coli* strain typing is helpful for classify strength of enteric enemy and their violent situation, for example; toxin gene screening (Ostroff et al., 1989), plasmid profiling (Ostroff et al., 1989), phage typing (Ahmed et al., 1987), restriction fragment length polymorphism with bacteriophage (Paros et al., 1993), ribotyping (Dalla-Costa et al., 1998), pulse-field gel electrophoresis (PFGE) (Bohm and Karch, 1992), PCR using randomly amplified fragment length polymorphism analysis (RFLP) (Zhao et al., 2004), enterobacterial repetitive intergenic consensus sequence polymerase chain reaction (ERIC-PCR) (Versalovic et al.,

1991) and multilocus sequence typing (MLST) (Tartof et al., 2005). A variety of *E. coli* subpopulation in microenvironment is much feasible, thus major clone during investigation should be distinguished by number of clone together with approved genetic evidence. The MLST method is a gold standard to discriminate the strain typing (Maiden et al., 1998) especially from *E. coli* in term of molecular epidemiological study but MLST are relatively expensive when compare with other protocol because of laborious process of DNA sequencing. Nowaday, there are the cost effective high-throughput MLST (HiMLST) is performed by next-generation sequencing (NGS) to generate the sequence data. The HiMLST consists by 4 steps as follow target gene amplifling, individual barcode Incorporating, PCR Sample Pooling. Emulsion PCR and *454* Sequencing (Boers et al., 2012).

The concordance of animal production in the emergence of antimicrobial resistance

There was many roughly evidence to support the view that the emergence of antimicrobial resistant bacteria in livestock populations was connected to the emergence of AMR in bacterial populations that colonized and infect humans (EFSA, 2015; Singer et al., 2003), For example, a AMR review found that “proportion of human extra-intestinal extended-spectrum β-lactamases producing *Escherichia coli* (ESBLP) infections originate from food-producing animals”, with poultry as a probable source (Lazarus et al., 2015; Leverstein-van Hall et al., 2011a). Regardless of this other recent study claim that most of the emergence of AMR in bacteria in humanity appears to originate from AMU (Antimicrobial usage) in humans, while the majority of AMR bacteria in livestock seem to originate from AMU in livestock. For example, whole genome sequence analysis of other *Enterobacteriaceae* bacteria, *Salmonella enterica* serovar Typhimurium phage type (PT) or definitive type (DT) 104 in human and livestock productions i has shown a greater diversity of AMR genes in human *S.* Typhimurium DT104, by comparison with those isolated in local livestock populations (Mather, 2013; Mather et al., 2013). The indication was that there were contributing sources other than foods of animal origin or livestock (Mather et al., 2013). In addition, a systematic review reported that usage of critically important antimicrobials for human treatment (such as fluoroquinolones and third- and

fourth-generation cephalosporins) was higher in humans than in food-production animals (EFSA, 2015). Commonly, in both animals and humans, a positive association was found between the volume of antimicrobial consumption and prevalence of resistance in the exposed bacterial populations (Burow et al., 2014; EFSA, 2015). Nonetheless, there was consensus within the scientific literature that there are routes for spillover of AMR between the bacterial populations of human and food-producing animals in both directions (Lazarus et al., 2015; Leverstein-van Hall et al., 2011a). The most recently mentioned route is via AMR bacteria passed through food distribution and consumption, the majority of which were colonizing bacteria of the host gastrointestinal tract (Lazarus et al., 2015). Such bacteria might be commensal in animals but pathogenic in humans, or may be commensal in both (Singer et al., 2003). Basically, the reflection of crossover events, in terms of human disease, appeared to be the outbreak form (Mather et al., 2013), although this apparent pattern might be a result of reporting bias, as a result of a relatively high abundance of research into the evidence generated through outbreak investigations. The strong and direct evidence for AMR transmission via food was stilled limited (Lazarus et al., 2015). For example, a study in The Netherland reported increased levels of (extended-spectrum β-lactamases producing) ESBLP bacterial isolates with similar resistance genes in poultry meat and humans (Overdevest et al., 2011a; Overdevest et al., 2011b). Moreover, there was evidence of AMR occurrence not only in foodstuffs from animals (Duong et al., 2006; EFSA, 2015; Muriuki et al., 2001; Raufu et al., 2014; Thai et al., 2012) but also in other types of foodstuffs like vegetable (Allen et al., 2013).The recent detection of colistin resistance in food-borne pathogens in humans, livestock, pork and vegetables in many countries raises the issue of the potential role of the global travel and trade in the transboundary dissemination of resistance genes (Doumith et al., 2016; Liu et al., 2016; Skov and Monnet, 2016; Zurfuh et al., 2016).

The pressure which influence AMR emergence in animal production

Even though, antimicrobial resistance occurs basically as a consequence of selective pressure which placed on susceptible microbes by the usage of antimicrobial agents

(Dione et al., 2009; Glynn et al., 2004; Koningstein et al., 2010), a diversity of other factors also affected to the emergence and spread of resistance. Standards such as restricted comingling, vaccination, house managing, adequate ventilation, temperature controls, biosecurity and appropriate nutrition were basically used in the new-fashion animal production to reduce the risk of introduction and spread on bacterial infection in animal herds. But this risk assessment required strongly and hugely financial investment, as well as practicing and incentivizing staff. Regardless, these standards were implemented suitably, a residual disease risk would continue (Adelaide et al., 2008; Cerniglia and Kotarski, 2005). The antimicrobials were regularly used in livestock production as a kind of insurance in addition to animal diseases risk-management standard. The tetracyclines, penicillins and sulfonamides resistance had been regularly observed in chicken and swine bacterial isolates in *Enterobacteriaceae* family, and MDR had been reported as significantly higher in these isolates than those from cattle. The intensively growing conditions under which pigs and chickens were often housed might be associated with higher disease potential and therefore a greater AMU in order to control sub-clinical infection (Duff and Galyean, 2007). In some developing countries, antimicrobials were widely utilized by swine farmers without veterinary recommendation due to their low prize and ready availability for sale over the counter (Laxminarayan et al., 2013). In some African countries, 55 percent of tetracyclines accounting used in food animals (Mitema et al., 2001). Harmonizingly, oxytetracycline was commonly used in small-scale livestock farm, while antimicrobial used included fluoroquinolones, erythromycin, sulfonamides and trimethoprim in Kenya (Kariuki et al., 2013). Antimicrobials were mostly purchased over the counter or from animal health assistants, without veterinarian advice. Some of drug quality was also an issue, as approximately one third of the drugs failed quality tests carried out by the National Quality Control Laboratory. The enteric bacterial isolates detected in food producing animals and meat were commonly resistant to ampicillin, tetracycline, sulfamethoxazole-trimethoprim and streptomycin with notable additional resistance to quinolones and third-generation cephalosporins, which were critically important in human medicine (FAO, 2016). Food from animal's meat was likely to still be

an important fomite for transmission of AMR bacteria from animals to humans. Other factors that could drive AMR include environmental contamination with antimicrobials from feces or their metabolites, residue concentrations of antimicrobials in eatable tissues, and direct zoonotic transmission (Aarestrup et al., 2006; Marshall and Levy, 2011; Padungtod and Kaneene, 2006; Padungtod et al., 2006). The ruin of effective antimicrobials to treat unhealthy animals undergo effects of livestock production (Cerniglia and Kotarski, 2005). Harmonizingly, there was risk for anyone involved in the meat production chain which exposed to resistant bacteria (Garcia-Alvarez et al., 2012; Lewis et al., 2008).

Relation between of AMU in animal production and AMR detection

Globalizing of antimicrobial use was considered to be the major factor associated with resistance in bacterial populations (Aarestrup et al., 2008; Acar and Moulin, 2012). The antimicrobial usage in health care, agriculture, aquaculture and industries had an impact on the expression, persistence, selection and transfer of resistance phenotypes and genotypes in bacterial populations (Aminov and Mackie, 2007; Courvalin, 2008; Mathew et al., 2007). Redundant use and misuse of antimicrobials were commonly recognized as two of the main factors for acquired AMR, both directly and indirectly, due to the selection pressure placed on human and animal microbiota (Martinez and Baquero, 2009; Novo et al., 2013; WHO, 2014) and on environmental bacteria (Martinez and Baquero, 2009). In European Union (EU) and Southeast Asian countries, many of the antimicrobial drugs licensed for veterinary use matched to antimicrobial classes or groups routinely used in humans (FAO, 2016). Third-generation cephalosporins (e.g. ceftiofur), regarded as critically important antimicrobials in humans (WHO, 2016) had been associated with the selection of co-resistance to different antimicrobials such as tetracycline and chloramphenicol in *E. coli* isolated from feces (Lowrance et al., 2007). This situation had been observed in health care unit, farms, effluent and sewage environments and in the intestinal tract of treated animals and humans (Martinez and Baquero, 2009). The constancy of antimicrobial residues in animal feed and animal waste contaminant also affected the aquatic and environmental microbiota (You and Silbergeld, 2014). Colistin was good example for drug which had controversial issue between veterinary medicine

and human medicine. Colistin which was the polymyxin E had been use in veterinary medicine for several decades and was used across several food producing animal species (e.g. pigs, poultry, sheep, goats, calves and adult cattle) including farming fish. Indications for use ranged from gastrointestinal tract infections by bacteria in *Enterobacteriaceae* family to topical treatment of mastitis. Colistin was regularly utilized in feed and water in intensive growing systems, not only for therapeutic purposes but also for prophylactic and metaphylactic purposes in flock of animals (Catry et al., 2015). Colistin was also often used in human medicine for the cure of infections caused by MDR carbapenemases producing Gram-negative bacteria, in combination with tigecycline, which had brought colistin to be re-justified as a highly important antimicrobial by WHO (Catry et al., 2015; WHO, 2011). In 2016, the detection of mobilized colistin resistance in food-borne pathogens in animals (Liu et al., 2016), foods and humans (associated with infection), observed worldwide, raises serious and urgent public health awareness (Skov and Monnet, 2016). At now, there were at least five allotypes of plasmid borne genes associated with this resistant phenotype (Borowiak et al., 2017; Carattoli et al., 2017; Liu et al., 2016; Xavier et al., 2016; Yin et al., 2017). It was strictly recommended that, for veterinary purposes, colistin should only be used for treatment (Catry et al., 2015; EMA, 2015). Nevertheless, there were currently confined data on the range and pattern of antimicrobial usage from food-producing animals, particularly in Southeast Asian countries. Only a few countries in Europe (e.g. Denmark, Sweden and Netherlands) regularly conducted systemic surveillance of AMU and AMR in humans, animals and food products of animal origin. At European Union level, the ESVAC (European Surveillance of Veterinary Antimicrobial Consumption) program assessed antimicrobial sales, adjusted by biomass of livestock populations, across different European countries (ESVAC, 2015). The emergence of AMR bacterial strains was dependent on various factors relating to the antimicrobial (e.g. amount, dosage, frequency and duration of selection pressure) and the bacterial organism (e.g. appearance of genes conferring resistance to that antimicrobial substance, and advantage gained by the expression of these to the survival of the bacteria) (McEwen, 2006). Usage of antimicrobials might unblock gene expression,

affecting the development of resistance genes in bacteria (Courvalin, 2008; Lambert, 2012) or inducing the occurrence of mutation (Limoli et al., 2014). Type, frequency and duration of antimicrobial therapy were the important factor in dissemination of resistance. Commonly, it had been estimated that more than 75 percent of antimicrobials used in livestock were excreted, mostly unmetabolized (Marshall and Levy, 2011). The antimicrobial residues in these farm environments was likely to be high. This was a crucial risk factor for the emergence of AMR. AMU also impacted on the competition for nutrients between bacterial populations in ecosystems through the elimination of susceptible bacteria (Aarestrup et al., 2008). In recently previous study, plasmid-mediated resistance to third generation cephalosporin in *E. coli* in livestock was affected by the reduction of numbers of susceptible bacteria in the gut microbiome (Volkova et al., 2012). Antimicrobial usage also appeared to reduce the infective dose required by resistant pathogens to cause infection, causing a serious risk for hosts exposed to, these bacteria (da Costa et al., 2013). Even though, there was evidence of prevalence of resistance in gut commensal bacteria (especially *E. coli*) in food-producing animals and foods of animal origin (Chantziaras et al., 2014). there were currently limited data on the role of these bacteria as potential sources of resistance genes for human. This same issues had been discussed in humans carrying resistant strains and undertaking antimicrobial therapy (da Costa et al., 2013). In *E. coli*, transfer of resistance determinants between bacteria had been mainly caused by the selection pressure imposed by AMU (da Costa et al., 2013). Usage of third-generation cephalosporins in livestock had been associated with emergence and spread of ESBL P Gram-negative bacteria, which caused a serious risk to public health (Aarestrup et al., 2008). The occasional isolation of carbapenem-resistant Gram-negative bacteria in livestock animals was also believed as a serious risk to public health, as carbapenems were considered “last-resource” β-lactam antimicrobials for cure of life-threatening infections in humans. Carbapenems were not regularly used in food-producing animals and were predominantly used in human hospital settings. Nevertheless, there could be a risk of co-resistance through usage of other antimicrobials in agriculture or through horizontal transferred from human pathogens.

Transfer of resistance traits within the bacterial cells could be induced by AMU and had been detected in macrolides in bacteria in Enterobacteriaceae family. Erythromycin promoted the transposition of erythromycin-resistant genes from a non-conjugative to a conjugative plasmid, which can then became mobile between bacteria (Courvalin, 2008). Controversially, in countries where use of particular antimicrobials (e.g. fluoroquinolones) were not allowed in livestock, low levels of, or no resistance to, these antimicrobials were observed in foodborne zoonotic bacteria (Aarestrup et al., 2008). Antimicrobials at low dosages (i.e. residual levels, sub-therapeutic dosages) were also contributing to resistance as they promoted genetic and phenotypic variability in exposed bacteria (Andersson and Hughes, 2014; Martinez, 2008; You and Silbergeld, 2014), however they were less likely to kill susceptible bacteria leading to selection bias than antimicrobials administered at higher dosages. In addition, sub-lethal doses also appeared to increase gene expression, formation of biofilms that were also indirectly responsible for resistance due to the close proximity of bacteria, which might favour the horizontal transfer of mobile resistance determinants (Andersson and Hughes, 2014; Lupo et al., 2012). Soil (Forsberg et al., 2012; Mathew et al., 2007) and water bacteria (Lupo et al., 2012) had been reported as reservoirs for resistance genes, and were exposed to antimicrobial residues derived from human, industrial, and agricultural use (Forsberg et al., 2012). Existence of antimicrobial residues derived from human, industrial and agricultural usage in the aquatic and terrestrial environments also contributed to selective pressure on environmental bacteria (Forsberg et al., 2012; Lupo et al., 2012; You and Silbergeld, 2014) and commensals and pathogens presented in the gut microbiome of farm animals (You and Silbergeld, 2014). It should be noted that antimicrobials differed in how efficiently they were processed in animal guts (and thus in the amount of residue excreted) (Kemper, 2008)and in how long the residues remain bioavailability in the environment (e.g. how long they were adsorbed to soil) (Kemper, 2008; Kumar et al., 2005). Therefore different type of antimicrobials caused different levels of public health risk (ASM, 2009). For example, sulfonamides did not strongly adsorb to soil, thus remaining bioavailability in the environment for long periods (Wegst-Uhrich et al., 2014). Excretion rates were dependent

on the type of antimicrobial, mode of administration, animal species and period since administration. Excretion rates for tetracyclines and sulfonamides might vary between 40 and 90 percent when they were comparing (Kemper, 2008). There was currently a lack of data on concentrations of antimicrobials in soil, manure and surface water, perhaps due to insufficiently sensitive analytical methods (Thanner et al., 2016). Importantly, antimicrobials which were concentration-dependent, such as aminoglycosides, were more likely to rapidly exert selection pressure on bacteria in soil or water before they were diluted, in comparison to time-dependent antimicrobials (such as β-lactams) which required sustained high concentrations in order to had an effect on bacterial viability (Amábile-Cuevas, 2016).The growing occurrence of MDR organisms enabled co-selection, which reduced the removal of all antimicrobials in order to achieve a useful reduction in the prevalence of resistance. However, reduction of numbers of resistant bacteria might only be possible if these were outnumbered by susceptible bacteria in an antimicrobial-free environment in which only a small number of individuals had been exposed to antimicrobials, or in the presence of a limited “selective density”(Levy and Marshall, 2004). This would not be the case in high-selective-density environments such as hospitals and conventional intensive farms (Levy and Marshall, 2004; PHE, 2014).

Categories of AMU in animal production in relation AMR detection

Antimicrobial growth promoters (AGPs)

Exposure of bacteria to subtherapeutic concentrations of antimicrobials was important role in AMR evolution (Andersson and Hughes, 2014). The usage of AGPs as feed additives in intensively produced animals had been found to alter the gut microbiome of treated animals and promoted resistance transfer within the animal and the environmental microbiome (You and Silbergeld, 2014). AGPs were administered at subtherapeutic dosages to groups of animals via drinking water or feed for prolonged periods to improve growth rates (Capita and Alonso-Calleja, 2013; Castanon, 2007; Wielinga et al., 2014). AGPs were sold and used in many countries without veterinary recommendations or supervision (Laxminarayan et al., 2013). There was still contrasting evidence, however, as to whether the improvement in animal production due to the use of

AGPs was significant, and the mechanism behind any such effect was still largely unknown (Lee et al., 2012). It was important to state that the impact of AGPs on productivity could be as little less than 1 percent, if nutrition, hygienic practices and health care of the herd or flock was also improved (Laxminarayan et al., 2015). However, such necessary improvements might not always be easy to achieve, especially in developing countries, where money were limited. The banning of AGPs in Europe in 2006 (EC, 2006) led to a reduction in the levels of vancomycin-resistant *enterococci* (VRE) previously observed in chicken in Denmark (Singer et al., 2003). Vancomycin was not licensed for use in poultry, but their resistance had emerged as result of the use of avoparcin (also in glycopeptides group) as an AGP in poultry production (Singer et al., 2003; Wielinga et al., 2014).Even though sub-therapeutic dosages had been linked to the emergence of antimicrobial resistance, AGPs continued to be used in many non-EU countries in intensive animal production, although the extent of this was currently unknown (Capita and Alonso-Calleja, 2013; Castanon, 2007; Singer et al., 2003). There had been a recent move in the United States to reduce their use. Animal feed is supplemented by other, non-antimicrobial compounds, which might, in turn, affected microorganisms. Sepiolite, for example, had been used as an additive in animal feed since 1990 in the EU. It slowed the passage of food through the intestinal tract, enabling a better absorption of nutrients. Sepiolite was not an antimicrobial, nor does it exerted any antimicrobial effect, but it promoted the horizontal transfer of resistance plasmids between bacteria, which could be aggravated if there was concomitant presence of AGPs (Rodriguez-Beltran et al., 2013).

Prophylaxis

This was defined as the antimicrobial usage to susceptible but healthy animals to prevent the occurrence of infectious disease. A regular example was the infiltration of the mammary glands of dairy cattle with antimicrobials such as penicillins, cephalosporins, or other lactams after cessation of lactation (Capita and Alonso-Calleja, 2013; Landers et al., 2012). Such AMU was likely to have a similar effect to that of growth promoters, although therapeutic levels of dosing, if adhered to, should be less likely to induce resistance in exposed bacterial populations. Nonetheless, it might not be the case when

the administration occurred in animal groups through water and feed (e.g. pigs, chicken) due to the variations in consumption by individual animals and the number of animals exposed. It was noted that particularly in countries where antimicrobial production and storage chains were inadequate (due to environmental or infrastructure-related issues) antimicrobials might be susceptible to degradation through oxidation-reduction reactions, hydrolysis, biodegradation or photodegradation (Osei, 2014). These antimicrobial preparations might reduce concentration and bactericidal activity when used, allowing for the survival of exposed bacteria and the generation of resistance (Osei, 2014).

Metaphylaxis

Defined as the administration of an antimicrobial at therapeutic doses to all animals within a group in which some individuals exhibited infection. Metaphylaxis acted both as a treatment for those animals currently infected and a preventive measure against infection in those animals who are healthy but risk becoming infected. The administration of oxytetracycline in the flock water supplied, as treatment and prevention against *Mycoplasma* infections in poultry, was a common example. The number of animals exposed to metaphylaxis was often large: in poultry production, medicated water or feed could be used to treat more than 30,000 poultry in the same flock. In addition, even if precise dosing is used for example where antimicrobials were administered to all members of a herd in injectable form such widespread AMU inevitably increased the risk of resistance emergence, due to the increased probability of bacteria with natural resistance which encountered the antimicrobial and potentially selected for within the affected microbiome (FAO, 2016).

Therapeutic use

This described treatment of active bacterial infection in a single animal, or a group, via antimicrobial administration. Whereas even a single dose of antimicrobial administered to a single animal had the propensity to generate AMR within bacterial populations resident in that animal, the repeated and continued usage of antimicrobials, for example to treat recurrent infections, compounds this risk (Harada and Asai, 2010; Usui et al.,

2014). Commonly, broad-spectrum antimicrobials were used in livestock before, or in place of, a confirmed diagnosis (for example before undertaking any antimicrobial susceptibility testing) due to economic considerations. The administration of macrolide antimicrobials such as erythromycin to pigs (Harada and Asai, 2010). The duration of systemic treatment should only be long enough to ensure elimination of infection in the affected animal or animal populations as this could result in further selective pressure on the gut microbiota (EMA, 2015). Correct dosing was very important for the reasons stated above. In addition, for antimicrobial substances that had been licensed for veterinary use for many years, recommended dosages by manufacturers in the Summaries of Products Characteristics (SPCs) might not be adequate as these may had not been calculated in accordance with updated pharmacokinetics and pharmacodynamics principles, or might not had taken account of the evolution of antimicrobial susceptibility in bacterial populations (EMA, 2015). It was important to note that when antimicrobials were administered via largely unregulated vehicles such as feed or water, whether for therapeutic, metaphylactic or prophylactic purposes, the exact intake of individual animals would be hard to ensure and define, and suboptimal dosing might occur (particularly of sick animals within a group housing and/or an ad lib feed and water system), which increased the risk of AMR emergence.

Biocide use

These were substances which, through chemical or biological action, hinder the activity of a broad spectrum of microorganisms (IFT, 2006; SCENIHR, 2009). Not only were they commonly used in agricultural settings. Their use was also frequent in human health-care systems and at community level. They might lead to emergence of AMR through cross-resistance, co-resistance and mutating mechanisms, and by activating an SOS response in bacteria leading to the repair and integration of DNA, some of which might include resistance genes (Capita and Alonso-Calleja, 2013). Biocide usage within the agricultural industry could be divided into two broad categories: a) animal feed preservatives and b) disinfectants and antiseptics. Within the food-production industry, biocides might also be used as food preservatives or decontaminants. For examples

including sulfites, lactic acid, trisodium phosphate or acidified sodium chlorate. Such compounds inhibited the growth of microorganisms in, or on, foodstuffs and produce (Capita and Alonso-Calleja, 2013). Lower susceptibility and resistance to biocides has been reported in bacterial populations since the 1950s, plasmids, transposons and integrons often also carry genes which conferred resistance to biocides (e.g. disinfectants and antiseptics) and to heavy metals, provided an evolutionary advantage to the resistant bacteria even in the absence of antimicrobial pressure (Acar and Moulin, 2012; Martinez and Baquero, 2009).Resistance mechanisms were similar for biocides and antimicrobial substances: selective pressure from biocide use in food production, industrial, agricultural and human health care settings, and water and wastewater treatment facilities. They could result in cross- or co-selection for AMR (SCENIHR, 2009). Biocides and antimicrobial substances might share common target sites and could be located closely together in mobile units (e.g. plasmids), leaded to co-resistance (Levy and Marshall, 2004). Efflux pumps coded at chromosomal level was involved in resistance to both antimicrobials and biocides (e.g. quaternary ammonium compound) due to their non-specific mechanism. Resistance to biocides had been associated with stress responses in bacteria, particularly when in the presence of sub-lethal doses but also in the presence of other stressors in the environment (e.g. osmotic and oxidative pressure, pH, nutrient availability) (IFT, 2006). Non-compliance with recommended dilution, preparation and storage of biocides might explain the increased tolerance to these products at low or sub-lethal concentrations and changed in phenotypic expression (e.g. membrane permeability, changes in membrane charge, efflux pumps and biofilm formation) of exposed bacterial populations (SCENIHR, 2009). There was currently a paucity of data relating to the extent of biocide use, presence of environmental residues and environmental stability (SCENIHR, 2009). Although risk assessment for AMR occurrence due to exposure to biocides was now a mandatory requirement for registration and licensing of these substances in European countries (ECHA, 2014), there was still little information on the correlation between biocides and antimicrobial resistance (Oggioni et al., 2015). Quaternary ammonium compounds or ethanol were used to destroy or inhibit microorganisms in animal husbandry and food

production and processing facilities like abattoir. In a recent study, *Salmonella enterica* strains exhibited reduced susceptibility to chlorine dioxide and peroxyacids when exposed to increase concentrations of these chemicals over time. In addition, the resistance of these bacterial species to various antimicrobials also increased after disinfectant exposure. Prior exposure to acidic disinfectants also increased the percentage of bacteria surviving subsequent acid treatments (SCENIHR, 2009). Although several existing studies provided evidence of a role of biocides in the emergence of AMR, exceptions exist where only weak or moderate correlations were observed between phenotypic biocide resistance and AMR in some bacteria (Oggioni et al., 2015). Therefore, further research was needed to assess the impact of biocides on pathogens relevant to public health. Since such substances were used ubiquitously and in large quantities throughout the food chain, it might be surmised that their relative impact on AMR emergence within agriculture and food industries might be important. Regardless, in order to quantify further the repercussions of biocide usage on the emergence of AMR within and outside the agricultural industry, further in-field surveillance of biocide use, and research into potential causal associations, was warranted (Fraise, 2002). It was noted that the usage of biocides was very widespread in many industries, and the disease burden to humans and domestic animals without their use would need to be weighed against any potential benefits from their reduced use.

Animal feed preservatives

Preservatives such as citric acid or sodium benzoate protect animal feed against decay caused by microorganisms. Such organic acids when ingested by food-producing animals might induce a selective pressure on gut bacteria (SCENIHR, 2009). In addition, these preservatives were often added in large quantities to feed such as silage, an increasing trend globally. This silage, if stored in such a manner that effluent could contaminate the environment, might potentially extend selective pressure to environmental bacteria.

Heavy Metals

Heavy metals might be used in agriculture as part of livestock feed supplements, and in a East Asia study were detected in manure from pig farms (Zhu et al., 2013). Heavy metals associated with the emergence and spread of AMR in environmental bacteria due to co-selection. The presence of heavy metals associated with the reduction of susceptibility of bacterial populations in soil (Aminov and Mackie, 2007) and commensal bacteria (e.g. *Enterococci*) (Werner et al., 2013) to antimicrobials. Heavy metals in soil could be derived from mining and industrial activities but also from agriculture and health care (Aminov and Mackie, 2007). AGPs use in livestock production could also contain heavy metals as trace elements (e.g. copper, zinc), or medication (e.g. arsenic in coccidiostatics) (You and Silbergeld, 2014). These metals could co-select for AMR not only in the gut microbiota but also in the environment through their persistence in animal waste (You and Silbergeld, 2014). Commensals and pathogens in the gut microbiota of animals could also be exposed to heavy metals through contaminated feed (e.g. mercury in fishmeal) (Defra, 2014; You and Silbergeld, 2014).

Other environmental factor which associated resistance emergence and maintenance

The stress and resistance genes in the bacterial genome were located closely together, which would promote their co-expression under stressful conditions, even in the absence of AMU (Mathew et al., 2007). Stressors identified as associated with emergence and transfer of resistance include extreme temperatures and variations on osmotic pressure and pH that could have an impact on the integrity of the DNA and affect bacterial survival (Aarestrup et al., 2008). Lack of biodiversity in ecosystems seemed to drive the emergence of resistance determinants and bacteria (da Costa et al., 2013). Transfer of resistant bacterial clones to hosts (i.e. humans and animals) was dependent on the age and health status of the host, and the frequency of contacts between the host and the environment, and/or between humans and animals (Martinez and Baquero, 2009; Mathew et al., 2007). Host stressors such as weaning was described as influencing the occurrence of AMR as they might have an impact on the gut environment, either by

enhancing uptake of resistance genes by bacteria or by favoring the survival of resistant strains (Mathew et al., 2007). Finally, a number of stress conditions in urban areas, especially those in developing countries, was related to the selection or maintenance of AMR genes in potentially pathogenic bacteria. Conditions as apparently unrelated to antimicrobials as air pollution might foster the resistance of airborne bacteria to antimicrobials (Jimenez-Arribas et al., 2001).

Risks of agricultural antimicrobial usage, other than AMR selection

Antimicrobial usage of any kind implies a risk for AMR selection and spread. We currently lacked adequate risk-assessment models for exploring the impact of agricultural AMU, simply because we had a poor understanding of the complex processes that lead to the emergence and spread of AMR. Many such mechanisms, e.g. mutations and horizontal transfer between distantly-related bacteria, occurred at very low rates, often below our detection capabilities. Regardless, as bacterial populations were enormous and many of them still unknown (we had been able to culture less than 10 percent of the species of the human microbiome, and less than 1 percent of the soil microbiome), most of these very rare phenomena at individual organism level occurred frequently at population level. In addition, there were other unpredictable implications of AMU in livestock. The acceptable levels of oxytetracycline and erythromycin in meat, followed usage in food-producing animals, could disrupt the fermentation process of sausages, as they were able to inhibit microbial starter cultures, but might allow the growth of pathogens such as *Salmonella* Typhimurium and *Escherichia coli* serotype O157:H7 (Kjeldgaard et al., 2012). The use of antimicrobials in food-producing animals might lead to food-related outbreaks through unexpected pathways.

AMR emergence and AMU within different animal production systems

Intensive systems

The intensification of livestock production (e.g. large numbers of animals kept at high density and usually indoors) associated with the use of antimicrobials as prophylaxis

against infectious disease, often for prolonged periods and for large populations of animals. Pro- and metaphylactic usage of antimicrobials at different stages of livestock production could also had an impact on the emergence of resistance (Chang et al., 2015). In Eastern European countries, higher levels of resistance had been reported in *E. coli* isolates in piglets. This contrasted with the predominant *E. coli* isolates with susceptible phenotypes and genotypes reported in sows in the same study (Mazurek et al., 2013). It was associated with the prophylactic use of antimicrobials in younger animals to prevent and contain the spread of respiratory and gastrointestinal infectious diseases (Mazurek et al., 2013). Animals bred for intensive production also tended to have reduced variability in their microbiota and a similar susceptibility to colonization with particular bacterial species (Schokker et al., 2014). This, coupled with the close proximity of animals in such systems, could result in amplification of any resistant population(s) of bacteria, which might outcompete other bacterial populations. Due to the factors above, an intensive system processed with poor biosecurity and herd/flock health might run a high risk of being colonized by pathogenic strains of bacteria (Zhu et al., 2013). The poor animal health within such situations also necessitate the increased use of antimicrobials, this was likely to support the development of AMR (FAO, 2013b). Given ever-growing global demand for livestock products, it was expected that intensive production would continue to expand in the future. It might be hypothesized, however, that intensive systems with high biosecurity might, in fact, reducing requirements for AMU and thus reducing the risk of AMR emergence. Intensive farms might also be able to take practical steps to mitigate AMR transfer into and out of the system. But as the authors could find little evidence to substantiate these theories in the literature, further research was warranted. It was important to note that, while hypotheses could be made about the effect of agricultural practices on the emergence of AMR in food animals, biological factors needed to be considered in relation to the potential for transmission of resistance to human bacterial populations. The results from previous study, it found that livestock animals appeared to be a more likely source for a proportion of human infections than other food-producing animals (Lazarus et al., 2015). Genomic data demonstrated that human extraintestinal

pathogenic *E. coli* and avian pathogenic *E. coli* shared numerous virulence factors, and resistant strains that were able to infect avian sources were also more likely to possess the cellular machinery required to infect humans (Lazarus et al., 2015). Such findings were relevant from a public health perspective since the fractional proportion of poultry products consumed globally currently outstretches any other protein source, and was projected to continue to do so (due both to increasing global demand and the efficiency of poultry feed conversion, which surpasses that of other livestock) (FAO, 2013a).

Extensive systems

Extensive livestock farming systems, typically characterized by low inputs generating low out-puts (the converse of intensive systems) might potentially require lower inputs of antimicrobials, and thus by default, result in lower rates of AMR emergence. However, by comparison with intensive systems, extensive systems required higher animal numbers for the same output (FAO, 2013b). Extensive systems involving free-roaming animals in large numbers might exhibit high commensal and pathogenic bacterial transmission rates and exposure to multiple bacterial species (including environmental species such as soil bacteria) which might not be as prevalent in intensive systems (FAO, 2013a). These factors might result in promoting the generation and transmission of AMR genetic material and bacterial populations.

Organic systems

Organic production systems in different countries could vary in the level of antimicrobial therapies allowed. In Europe, restrictions existed in the number of therapeutic courses allowed and the duration of withdrawal periods (Anon, 2007). Pro- and metaphylactic use of antimicrobials was prohibited. Alternative therapeutic plans were encouraged and use of antimicrobials was only permitted when necessary. Use of vaccines for disease prevention was permitted and encouraged (Anon, 2007). Recent studies comparing AMR levels in livestock reared in organic versus conventional production systems showed higher concentrations in the latter (Cui et al., 2005; Holtcamp, 2011; Mazurek et al., 2013). In Eastern European countries, Mazurek et al. (2013) reported

that resistant *E. coli* isolates were mainly observed in livestock animals raised in barns in conventional farms rather than in animals having access to pasture and raised organically, with lower exposure to antimicrobials (Mazurek et al., 2013). In another study in the United States, MDR enteric bacteria spp. were detected in both antimicrobial-free and conventional pig farms. This was likely due to environmental reservoirs that could be a source of resistance genes and resistant bacteria (Quintana-Hayashi and Thakur, 2012). These results conformed to the reports from pig farms in Thailand (Lugsomya et al., 2017). A poorly managed organic system, the drive to reduce AMU might lead to the administration of doses of antimicrobials below the minimum inhibitory concentration (MIC), leading to an increased selection pressure for AMR bacteria and/or recurrent infections or extensive onward transmission, required repeating treatment of single or multiple animals and instigating selection pressure for AMR. In addition, in organic systems where livestock production was integrated with an extensive and/or a free-range or outdoor farming model, access to AMR genes or bacterial populations via soil bacteria and effluent might result in a propensity for organic/extensively-produced livestock to harbor AMR comparable with conventionally produced or indoor animals. More comparative research was required on this topic, though it might be suggested that high biosecurity, high herd/flock health and indoor, organic systems might potentially induce and harboured relatively less AMR than others. Despite this, it should be noted that biocide treatment of organically-produced animal feed and human foods might still potentially induce AMR in the food chain. The indiscriminate use of biocides should therefore be discouraged (Davin-Regli and Pagès, 2012; Fuentes et al., 2014).

Pig production cycle and management in Thailand

The majority of pig farms in Thailand are raised on either an open or a closed housing system. An evaporative cooling system is defined as closed houses by literally wrapping up with plastic sheets. Pig farming systems can be classified based on period of animal ages; breeder, one site, two sites and multisite farm systems. In breeder farms, this produces only piglets and supplies among weaned pigs to fattening farms. In general,

piglets are weaned at 18–28 days of age, and the sows are moved to mating unit on the same day. Sows are prepared in their quarantined-pen for 2 weeks. A week prior to delivery, sows are moved to farrowing units with well hygienic management, which are administrated with antibiotics at least two days after delivery for disease prevention and reduce stress. All-in-all-out system, in which all pigs enter and leave from the facility together at the same time, is applied in mating, farrowing, and nursery units. Animals raised under the same management system are considered to have a similar disease status. Therefore, any production compartments can claim freedom of disease for the animals produced regardless of where the animals actually are.

Risk factors for the emergence of AMR in agriculture at national and international level

It was important to highlight the fact that the extent and patterns of AMU in agriculture and other industries were likely to vary considerably between and within countries, due to the influence of various factors (e.g. legislative framework and governance, financial status and stability, degree of international imports and exports, human resources: population size, education and expertise, culture, structure and organization of the various agricultural production systems in use nationally). In many countries, particularly developing countries, there had been dramatic changes in agricultural systems in recent years, driven by both increasing local demand and new and emerging trade opportunities (FAO, 2013a, b; Otte et al., 2007; Rushton, 2010; Stiftung, 2014). A growing global population and increasing wealth in emerging economies, for example in China and India , had stimulated demand for animal protein and the development of global value chains (Otte et al., 2007). For example, new export opportunities for African countries (USDA, 2018) led to increased production and intensification of agricultural systems in the region (FAOSTAT, 2017). Globally, poultry production was growing this century at around 3 percent per year and seemed set to continue to grow as global diets and consumption patterns shift (FAO, 2013b). Changes to agricultural systems as a result of intensification

involved changes in livestock/fish numbers, feed type and quantity used, husbandry methods, and animal density. All of these factors could influence disease dynamics (Otte et al., 2007), which in turn might drive changes in AMU. The extent of the impact on AMU depended on the attitude of veterinary practitioners and farmers towards use of antimicrobials within particular legislative and governance frameworks, and alternative methods for maximizing animal productivity.

Route of spread of antimicrobial resistance between animals and humans

Both pathogenic and non-pathogenic resistant bacteria could be transmitted from livestock to humans via food consumption, or via direct contact with animals or their waste in the environment (Marshall and Levy, 2011). Fomites could also play an important role in the local and wider spread of resistant bacteria. In Denmark, farm-to-farm spread of multidrug-resistant *Salmonella enterica* serovar Typhimurium DT204 had been closely studied, and shared farm equipment (e.g. machinery) was identified as an important route (Aarestrup et al., 2006). The mechanism that helped spread bacteria had the potential to transfer resistant bacteria. Resistance might also be conferred by the exchange of genetic elements between bacteria of the same or different strain or species, and such transfer could occur in any environment where resistant bacteria had the opportunity to mix with a susceptible bacterial population, such as in the human or animal gut, in slurry spread on agricultural soil, or in aquatic environments (Aarestrup et al., 2006; Martinez and Baquero, 2002). If resistance developed in environmental bacteria, this could create an animal or human health problem when such bacteria contaminated water, food crops or animal feed, introducing the opportunity for bacterial mixing with commensal or pathogenic species in the animal or human gut (Aarestrup et al., 2006; Finley et al., 2013; Marti et al., 2013).

Risk pathways for the spread of AMR via the environment

Many antimicrobial preparations used for livestock were given orally so that antimicrobial residues excreted in animal faeces have the potential to exert selection pressure on bacterial populations in soil or water (AAM, 2009; Woolridge, 2012). However,

evidence was scarce as to how important this mechanism was in transferring resistance (Hong et al., 2011; McEwen, 2006; Novo et al., 2013) and different antimicrobials had different fates in the environment (AAM, 2009; Kemper, 2008; Kumar et al., 2005). The residues resulted from human treatment with antimicrobials or from pharmaceutical manufacturing could also exert selection pressure on environmental bacteria (Baquero et al., 2008; Finley et al., 2013; Igbinosa et al., 2011; Novo et al., 2013; Wellington et al., 2013). Indeed, effluent from drug manufacturing was found to contain extremely high concentrations of antimicrobial residues, as previously reported in countries with large pharmaceutical industries such as India (Larsson et al., 2007; Sim et al., 2011). Water, including that treated for human consumption, was an important vehicle for the spread of AMR. Water was not only directly consumed by humans and animals but is used for irrigation of crops which were then consumed by humans or used as animal feed (Finley et al., 2013). Water spread antimicrobial residues, resistant bacteria and resistance genes far and wide through the flow of natural water bodies and anthropogenic influences such as irrigation. This was a significant concern in developing countries, where water was shown to be a major route for transmission of pathogenic bacteria to humans (Wellington et al., 2013). Recreational water use was linked to exposure to AMR bacteria (Leonard et al., 2015).

Resistant *E. coli* transmissible chance from pig to human

There were certain reports on resistance *E. coli* isolates from pigs and pork had some genotyping characteristics such as *E. coli* O2:HNM isolated from human which is the extra-intestinal pathogenic *E. coli* (ExPEC) had a genetic identical to that in pig feces in Spain. These pig and human isolates were the same sequence type (ST10) by MLST but differed when compared by pulsatypes by PFGE (Cortes et al., 2010). In addition, there were sharing of *E. coli* sequence type 7 by MLST contained $bla_{\text{CTX-M-1}}$ located on plasmid incompatibility group from pig and human feces (Leverstein-van Hall et al., 2011a). In term of pig and pig associated farmer, only 11 strains (1.5%) of *E. coli* isolated from animals were identical clones to strains isolated from healthy farmers. These data suggest that the transmission of animal clones to livestock farmers or vice versa is less common (Cortes

et al., 2010). Additionally, *qnrS1* gene, the specific quinolones-mediated resistance plasmid, was independently presented in *E. coli* from human and farm animals with 99% similarity (Szmolka et al., 2011). In Thailand, the distribution of similar bacterial clone among diarrheal, healthy pigs and Thai farmers was noticed by existence of resistance cassette gene conferring MDR comprising tetracycline, ciprofloxacin, gentamicin, nalidixic acid, sulfamethoxazole-trimethoprim, sulfamethoxazole, kanamycin, ampicillin and streptomycin (Phongpaichit et al., 2007). However, this could not be consensual on direction of transmission, as long as those cross-section study did explain only existence in specific time and host investigation. There has been still lack of data uncovering a background and a concrete relationship between bacteria originated from pigs, pork and consumers. To date, there has not been defined why and when they initially emerge and whether they persisted or resisted during farming system.

Resistance of *E. coli* in pigs in the foreign country

In China, the high prevalence of MDR *E. coli* had the main antibiogram profile to ampicillin, ceftazidime, ciprofloxacin, sulfamethoxazole, chloramphenicol and tetracyclines (Xu et al., 2014). In European pig industrials, MDR appearance could not be concluded but the resistance patterns showed against penicillin, streptomycin, spectinomycin, doxycycline and sulfamethoxazole/trimethoprim (Schwaiger et al., 2012). Therefore, MDR incidences distributing in pig industrial area seem to be usual situation or certain strains might not be persistent resistance but a wild type.

Resistance of *E. coli* in pigs in Thailand

From the previous studies, role of both of porcine pathogenic *E. coli* and commensal *E. coli* are likely as reservoirs of antimicrobial resistance. Up to 98% *E. coli* isolates from normal healthy pigs are multidrug resistance (MDR) *E. coli.* Most of porcine fecal *E. coli* strains are resistance to tetracyclines and ampicillin. A total of 73 % commensal *E. coli* isolates carried class 1 integron. The most prevalence of inserted-gene cassette was

aadA2 (aminoglycoside adenyltransferase) gene. In term of pathogenic *E. coli*, porcine enterotoxigenic *E. coli* (ETEC) and enterohemorrhagic *E .coli* (EHEC) resisted to amoxicillin (100%) chlortetracycline (100%), streptomycin (100%), tetracycline (100%), sulfamethoxazole/ trimethoprim (100%) eventually in colistin (51.3%) and enrofloxacin (87.8%) (Prapasarakul et al., 2010). Moreover, the high incidence of resistance *E. coli* to streptomycin, sulfamethoxazole, amoxicillin, gentamicin and tetracycline was also reported in pork retail market in Khonkaen province, Thailand (Angkititrakul et al., 2005).

Effect of flavomycin to resistance *E. coli* in pigs

flavomycin is the phosphoglycolipid antimicrobial group. This flavophospholipol inhibits bacterial cell wall synthesis by interfere transglycosylase activity of the penicillin-binding proteins (Butaye et al., 2003). Flavomycin diminishes frequency of transferable plasmid conjugation encoding drug resistance among target pathogens, *in vitro* (Butaye et al., 2003). *In vivo* experiment, flavomycin at 9 mg/kg added to feed of fattening pigs could prevent either overgrowth by resistance strains or transmission of plasmids carrying resistance genes among intestinal *E. coli* of the pigs (van den Bogaard et al., 2002). By using molecular evidence, this can be explained by presence of sex pili promotes target of flavophospholipol into the bacterial cell (Pfaller, 2006). However, minimal inhibitory concentration (MIC) of *E. coli* direct against flavophospholipol was 64 ug/ml, but MICs range of these plasmid-bearing strains became 0.125 to 5 ug/ml (van den Bogaard et al., 2002).

CHAPTER III: Research question, Hypothesis, objectives, keywords and conceptual framework

Research questions

1: What is the situation of antimicrobial resistance (AMR) problems of enteric *E. coli* isolated from pig in Thai industrial farm during 2012-2014?

2: How does the antimicrobials using protocol affect the AMR problems in pig growing period and could they affect their pork at the end process at abattoir or not?

3: Can flavomycin reduce AMR rate in *E. coli* in pig production cycles?

Hypothesis

1. High rate of AMR in porcine fecal *E. coli* are commonly found in Thai pig production cycle at the time of investigation.

2. Phenotype and genotype associated AMR of fecal *E. coli* in pigs are different in each period of observation and can be altered by routine antibiotic used.

3. Use of flavomycin as feed additives in pig production cycle can reduce rate of AMR *E. coli* in term of genotype and phenotypes.

Objectives of the study

1. To determine AMR profile of porcine fecal *E. coli* isolated from pigs with routine antibiotic uses in Thai industrial farms.

2. To longitudinally monitor and compare AMR characteristics of porcine fecal *E. coli* difference between non-antibiotic uses and routine antibiotic uses in feed farms from creeping to fattening periods and their pork.

3. To determine efficacy of flavomycin using as feed additive to reduce AMR profile of porcine enteric *E. coli* in pig production cycles.

Keywords (Thai) : ยาต้านจุลชีพ เชื้อ*เอสเชอริเชีย คอลัย* ฟลาโวมัยซิน ฟาร์มสุกร ยีนดื้อยา

Keywords (English) : antimicrobials, *Escherichia coli*, flavomycin, pig farm, resistance gene

Conceptual framework

Chapter IV: Materials and methods

Part 1. Distribution of resistance characteristics of *E. coli* isolated from pigs in different antibiotic using status in Thailand

1.1 Study area and animal selection

Samples were collected from 28 farms across five Provinces of Thailand. Farms were selected based on their antimicrobial usage patterns. Twelve farms routinely used prophylactic antimicrobials (prophylactic antimicrobials: PA); two only used antimicrobials therapeutically (therapeutic antimicrobials: TA); and 11 small farms had never used antimicrobials (no antimicrobials: NA). The PA and TA farms had similar management systems, including open housing, the same vaccine program and multi-site production. They all had over 1,000 sows, had no pig replacement from outside sources and had consistent management for at least 2 years in terms of antimicrobial use, and sanitary and biosecurity measures taken. They were well managed and kept good records of production and antimicrobial use. The NA farms were all small (<50 sows) village-based enterprises, where the houses were open sided and contained pigs of different ages and stages of production. Feedstuffs were locally sourced. For the 12 PA farms a routine in-feed antimicrobial use program had been followed for at least 2 years for endemic bacterial disease prophylaxis. This included incorporating tiamulin-fumarate at 100 parts per million (ppm) and amoxicillin at 250 ppm in the feed in the nursery (6-8 weeks) and grower (8-16 weeks) phases of production. Where individual pigs became sick they were isolated and treated with therapeutic antimicrobials as appropriate. A total of 102 fecal samples from individual 18-20 week-old fattening pigs with a normal appearance and no recent history of enteric or respiratory disease or therapeutic antimicrobial treatment were obtained from the farms (7-10 samples per farm). A total of 70 fecal samples were obtained from similar fattening pigs on two TA farms that did not use prophylactic antimicrobials and only used the injectable antimicrobials enrofloxacin or gentamicin under veterinary prescription for individual treatment of specific clinical cases where the

sick pigs were kept in isolation. Another 67 samples were collected from fattening pigs on the 11 NA farms, where there was no access to or use of antimicrobials or vaccination.

1.2 Sample collection and bacterial identification

The sampling protocol was approved by the Chulalongkorn University Animal Care and Use Committee (permit number 58/2558). For creeping and nursery pigs, rectal swabs were directly used and kept in Clary-Blair transport medium. For growing and finishing, at least 25 g of rectal feces were collected into sterile plastic container. For carcass, at least 25 g of pork at thigh area were cut by sterilized blade and kept into sterilized container. All samples were delivered to the laboratory at 4°C within 24 hours. The rectal swabs were soaked in 0.85% sodium chloride solution (normal saline solution:NSS) and directly spread on the Eosine Methylene Blue (EMB) (Oxoid, UK) agar (Renoux and Terdjman, 1951). For rectal feces, at least 5 g were diluted 10-fold dilution till 10^{-4} and spread the solution EMB agar. (Demarco and Lim, 2002; Stampi et al., 2004; USDA, 2017). Colony presented metallic sheen on EMB plates to select the major population of bacteria. *E. coli* colonies were identified by their IMViC (Oxoid) biochemical reactions, comprising an indole test (+), a methyl red test (+), a Voges-Proskauer test (-), and a citrate test (-) (Clark et al., 1957). A representative pure colony from the highest dilution plate was randomly selected for further investigation.

1.3 Phylogenetic grouping

All isolates were characterized into their phylogroups using a published multiplex polymerase chain reaction (PCR)-based method that identifies eight phylogroups (A, B1, B2, C, D, E, F, and cryptic clades).*E. coli* ATCC 25922 and *E. fergusonii* CUVET427 were used as the control strains (Clermont et al., 2013).

Table 3 Primer list for Phylogenetic grouping

Target sites	Names of primer	Nucleotides sequences	PCR product (bp)
chuA	chuA.1b	ATGGTACCGGACGAACCAAC	288

	chuA.2	TGCCGCCAGTACCAAAGACA	
yjaA	yjaA.1b	CAAACGTGAAGTGTCAGGAG	211
	yjaA.2b	AATGCGTTCCTCAACCTGTG	
TspE4.C2	TspE4C2.1b	CACTATTCGTAAGGTCATCC	152
	TspE4C2.2b	AGTTTATCGCTGCGGGTCGC	
arpA1	AceK.f	AACGCTATTCGCCAGCTTGC	400
	ArpA1.r	TCTCCCCATACCGTACGCTA	
arpA2	ArpAgpE.f	GATTCCATCTTGTCAAAATATGCC	301
	ArpAgpE.r	GAAAAGAAAAAGAATTCCCAAGAG	
trpA1	trpAgpC.1	AGTTTTATGCCCAGTGCGAG	219
	trpAgpC.2	TCTGCGCCGGTCACGCCC	
trpA2	trpBA.f	CGGCGATAAAGACATCTTCAC	489
	trpBA.r	GCAACGCGGCCTGGCGGAAG	

1.4 Phenotypic resistance characterization and ESBLP confirmation

The minimal inhibitory concentration (MIC) of antimicrobials for the *E. coli* isolates was determined using the AST-GN 38 test kit in the Vitek2 compact automated susceptibility level detection apparatus (BioMérieux, France), excepted for tiamulin (Sigma-Aldrich, USA) where susceptibility testing was performed using an agar dilution test with results interpreted according to CLSI standards. The 19 antimicrobials or antimicrobial combinations tested were amikacin (AK), amoxicillin (AMX), amoxicillin-clavulanic acid (AMC), ampicillin (AMP), cefpirome (CPR), cefpodoxime (CPD), cefalexin (CEX), ceftiofur (XNL), chloramphenicol (C), enrofloxacin (ENR), gentamicin (GM), imipenem (IMP), marbofloxacin (MBR), nitrofurantoin (NIT), piperacillin (PIP), tetracycline (TET), tiamulin (TI), tobramycin (TM) and trimethoprim/Sulfamethoxazole (SXT). In the Vitek2 machine the set of antimicrobial arrays were harmonized to match with veterinary guidelines (Plumb, 2015b), including some drugs that were allowed for use in veterinary practice in the past (MoAC, 1999) *E. coli* ATCC 25922, *Pseudomonas aeruginosa* ATCC

27853 and *Staphylococcus aureus* ATCC 25913 were used as the control strains. The interpretation of susceptibility levels for AMP, CPD, XNL, GM, ENR, MBR, TET, C and TI followed the CLSI standards for antimicrobial disks and testing for bacteria isolated from animals Isi(VET1-0S3) (CLSI, 2015a), and interpretation for AMX, PIP, AMC, CEX, CPR, IMP, AK, TM, NT, SXT followed the CLSI standards for antimicrobial susceptibility testing (M100-S25) (CLSI, 2015b).

1.5 ESBL phenotypic screening and confirmatory test

The *E. coli* isolates were screen for ESBL production using the Vitek2 machine (BioMérieux, France) (Espinar et al., 2011). As a confirmation test for ESBLs the combination disk test (CDT) was performed for all isolates, as recommended (CLSI, 2015b).

1.6 bla_{CTX-M} gene detection.

The gene bla_{CTX-M} comprising variants $bla_{CTX-M-1}$ group, $bla_{CTX-M-2}$ group, $bla_{CTX-M-9}$ group, $bla_{CTX-M-8}$ group and $bla_{CTX-M25/26}$ group were detected by multiplex PCR in all ESBLP *E. coli* strains (Xu et al., 2005). The identity of representative PCR amplicons was confirmed by DNA-sequencing and analyzed using BioEdit version 7.0.0 (Ibis Biosciences, Australia), with comparisons made to the GenBank database.

Table 4 Primer lists for bla_{CTX-M} genes detection

Target sites	Names of primer	Nucleotides sequences	PCR product (bp)
bla_{CTX-M} Group 1	CTXM7	GCGTGATACCACTTCACCTC	540 –559
	CTXM8	TGAAGTAAGTGACCAGAATC	780 –779
bla_{CTX-M} Group 2	CTXM17	TGATACCACCACGCCGCTC	543 –561
	CTXM18	TATTGCATCAGAAACCGTGGG	863 –883
bla_{CTX-M} Groups 8 and 25/26	CTXM19	CAATCTGACGTTGGGCAATG	582 –601
	CTXM20	ATAACCGTCGGTGACAATT	855 –873
bla_{CTX-M} Group 9	CTXM11	ATCAAGCCTGCCGATCTGGTTA	298 –319

CTXM12	GTAAGCTGACGCAACGTCTGC	570 –590

1.7 bla_{CTX-M} conjugative assay

To determine whether bla_{CTX-M} genes were located on transmissible plasmids, a conjugation assay was performed using the broth mating technique, as recommended. The two selected donors used were *E. coli* PCU1 (positive for $bla_{CTX-M-1}$ and a single F replicon) and *E. coli* PCU2 (positive for $bla_{CTX-M-9}$ and a single FIB replicon). The recipient strain *E. coli* J53 was resistant to sodium azide: $NaN_3{}^{r}$. Transconjugants were selected on Luria Bertani (LB) agar (Oxoid, UK) supplemented with cefotaxime (2 μg ml^{-1}) and sodium azide (100 μg ml^{-1}) (Oxoid, UK). Antimicrobial susceptibility, a confirmatory test for ESBL P phenotype, PCR detection, and DNA sequencing of bla_{CTX-M} genes were performed on the transconjugants.

1.8 Genes encoding antimicrobial resistance

Bacterial DNA was extracted using a Wizard® Genomic DNA Purification Kit (Promega, Germany). A total of 16 pairs of primers specific for resistance genes in the *Enterobacteriaceae* family were generated (First Base Laboratories Sdn Bhd, Malaysia) and PCR thermal cycling conditions followed previous recommendations (Chuanchuen et al., 2010). Amplified genes included bla_{TEM} and bla_{PSE-1} for ampicillin, amoxicillin and piperacillin resistance, *aadA1* and *aadA2* for streptomycin resistance, *aadB* for tobramycin and gentamicin resistance, *tetA* and *tetB* for tetracycline resistance, *dfrA1*, *dfrA10* and *dfrA12* for trimethoprim resistance, *sul1*, *sul2* and *sul3* for sulfomanide resistance and *catA*, *catB* and *cmlA* for chloramphenicol resistance. A representative positive PCR amplicon for each gene was submitted for DNA-sequencing and analyzed by Bioedit version 7.0.0 (Ibis Biosciences, Australia) using the GenBank database.

Table 5 Primer lists for resistance gene detection

Target genes	Names of primer	Nucleotide Sequences	Product sizes
aadA1	aadA1-F	CTCCGCAGTGGATGGCGG	631
	aadA1-R	GATCTGCGCGCGAGGCCA	

Target genes	Names of primer	Nucleotide Sequences	Product sizes
aadA2	aadA2-F	CATTGAGCGCCATCTGGAAT	500
	aadA2-R	ACATTTCGCTCATCGCCGGC	
aadB	aadB-F	CTAGCTGCGGCAGATGAGC	300
	aadB-R	CTCAGCCGCCTCTGGGCA	
bla_{PSE-1}	blaPSE1-F	GCAAGTAGGGCAGGCAATCA	422
	blaPSE1-R	GAGCTAGATAGATGCTCACAA	
bla_{TEM}	blaTEM-F	ATCAGTTGGGTGCACGAGTG	608
	blaTEM-R	ACGCTCACCGGCTCCAGA	
catA	catA-F	CCAGACCGTTCAGCTGGATA	454
	catA-R	CATCAGCACCTTGTCGCCT	
catB	catB-F	CGGATTCAGCCTGACCACC	461
	catB-R	ATACGCGGTCACCTTCCTG	
cmlA	cmlA-F	TGGACCGCTATCGGACCG	641
	cmlA-R	CGCAAGACACTTGGGCTGC	
dfrA1	dfrA1-F	CAATGGCTGTTGGTTGGAC	254
	dfrA1-R	CCGGCTCGATGTCTATTGT	
dfrA10	dfrA10-F	TCAAGGCAAATTACCTTGGC	432
	dfrA10-R	ATCTATTGGATCACCTACCC	
dfrA12	dfrA12-F	TTCGCAGACTCACTGAGGG	330
	dfrA12-R	CGGTTGAGACAAGCTCGAAT	
sul1	sul1-F	CGGACGCGAGGCCTGTATC	591
	sul1-R	GGGTGCGGACGTAGTCAGC	
sul2	sul2-F	GCGCAGGCGCGTAAGCTGAT	514
	sul2-R	CGAAGCGCAGCCGCAATTC	
sul3	sul3-F	GGGAGCCGCTTCCAGTAAT	500
	sul3-R	CCGTGACACTGCAATCATTA	
tetA	tetA-F	GCTGTCGGATCGTTTCGG	658
	tetA-R	CATTCCGAGCATGAGTGCC	

Target genes	Names of primer	Nucleotide Sequences	Product sizes
tetB	tetB-F	CTGTCGCGGCATCGGTCAT	615
	tetB-R	CAGGTAAAGCGATCCCACC	

1.9 Plasmid replicon typing and intI detection

The presence of 18 plasmid replicons that are widespread in *Enterobacteriaceae,* comprising IncF (IncFIA, IncFIB, IncFIC, IncFIIA, IncF), IncI1-Iγ, IncN, IncP, IncW, IncHI1, IncHI2, IncL/M, IncT, IncA/C, IncK, IncB/O, IncX and IncY was investigated using five multiplex and three simplex polymerase chain reaction (PCR) tests. The PCR conditions and thermal cycles were performed according to a previous study (Carattoli et al., 2005). The *intI* gene representing integron I cassettes was detected by a simplex PCR following a previously published method (Goldstein et al., 2001). Representative positive PCR amplicons for each replicon were submitted for DNA-sequencing and analyzed as described above.

Table 6 Primer lists for characterization of plasmid replicons

Target sites	Names of primers	Nucleotide Sequences	Product sizes
IncHI1(parA-parB)	HI1 FW	GGAGCGATGGATTACTTCAGTAC	471
	HI1 RV	TGCCGTTTCACCTCGTGAGTA	
IncHI2 (iterons)	HI2 FW	TTTCTCCTGAGTCACCTGTTAACAC	644
	HI2 RV	GGCTCACTACCGTTGTCATCCT	
IncI1 (RNAI)	I1 FW	CGAAAGCCGGACGGCAGAA	139
	I1 RV	TCGTCGTTCCGCCAAGTTCGT	
IncX (ori γ)	X FW	AACCTTAGAGGCTATTTAAGTTGCTGAT	376

Target sites	Names of primers	Nucleotide Sequences	Product sizes
	X RV	TGAGAGTCAATTTTTATCTCATGTTTTAGC	
IncL/M (repA,B,C)	L/M FW	GGATGAAAACTATCAGCATCTGAAG	785
	L/M RV	CTGCAGGGGCGATTCTTTAGG	
IncN (repA)	N FW	GTCTAACGAGCTTACCGAAG	559
	N RV	GTTTCAACTCTGCCAAGTTC	
IncFIA (iterons)	FIA FW	CCATGCTGGTTCTAGAGAAGGTG	462
	FIA RV	GTATATCCTTACTGGCTTCCGCAG	
IncFIB (ori γ)	FIB FW	GGAGTTCTGACACACGATTTTCTG	702
	FIB RV	CTCCCGTCGCTTCAGGGCATT	
IncW (repA)	W FW	CCTAAGAACAACAAAGCCCCCG	242
	W RV	GGTGCGCGGCATAGAACCGT	
IncY (repA)	Y FW	AATTCAAACAACACTGTGCAGCCTG	765
	Y FW	GCGAGAATGGACGATTACAAAACTTT	
IncP (iterons)	P FW	CTATGGCCCTGCAAACGCGCCAGAAA	534
	P RV	TCACGCGCCAGGGCGCAGCC	
IncFIC (repA2)	FIC FW	GTGAACTGGCAGATGAGGAAGG	262
	FIC RV	TTCTCCTCGTCGCCAAACTAGAT	
IncA/C (repA)	A/C FW	GAGAACCAAAGACAAAGACCTGGA	465

Target sites	Names of primers	Nucleotide Sequences	Product sizes
IncT (repA)	A/C RV	ACGACAAACCTGAATTGCCTCCT T	750
	T FW	TTGGCCTGTTTGTGCCTAAACCA T	
	T RV	CGTTGATTACACTTAGCTTTGGAC	
IncFIIs (repA)	FIIs FW	CTGTCGTAAGCTGATGGC	270
	FIIs RV	CTCTGCCACAAACTTCAGC	
IncFrepB (RNAI/repA)	FrepB FW	TGATCGTTTAAGGAATTTTG	270
	FrepB RV	GAAGATCAGTCACACCATCC	
IncK/B (RNAI)	K/B FW	GCGGTCCGGAAAGCCAGAAAAC	160
	F/B RV	TCTTTCACGAGCCCGCCAAA	
IncB/O (RNAI)	B/O FW	GCGGTCCGGAAAGCCAGAAAAC	159
	B/O RV	TGCGTTCCGCCAAGTTCGA	

Table 7 Primer list for characterization of cassette in integron 1.

Target sites	Names of primers	Sequences	Product size
intI1	intI1F	CCTCCCGCACGATGATC	280
	intI1R	TCCACGCATCGTCAGGC	

1.10 Data analysis

Resistance rates, replicon positivity rates, and resistance gene profiles were presented as percentages, and the antimicrobial resistance profiles were reported in antibiogram patterns for the three categories of farms. SPSS version 17.0 (IBM, USA) was used to calculate Odds ratios and 95% confidence intervals (CI) for each resistance phenotype and each replicon type, and each resistance gene between isolates from the three different categories of farm.

Part 2. Longitudinal monitoring of molecular resistance genotypic of *E. coli* monitoring from pig in the production cycle

2.1 Farm selection criteria

The two multisite industrial pig farms were chosen from Chainart and Nakhon Pathom provinces where run beneath Thai standard livestock farm criteria ruled by Department of Livestock Development. In Nakhon Pathom, farm with antimicrobial use in feed (AF) was screened by the farm routinely used combination of tiamulin and amoxicillin at 100 (part per million ppm and 250 ppm) respectively, mixed in feed during nursery and growing periods. Farm with non-antimicrobial use in feed (NF) in Chainart indicated by the farm had never used antimicrobials in feed additive over ten years at least. At creeping period, enrofloxacin injection was used in case of infection with apparent symptom however all treated pigs were excluded from this study. The selected farms had over 1,000 sows, had no pig replacement from outside sources, all in all out system, and had consistent management for at least 2 years in terms of antimicrobial use and sanitary and biosecurity measures taken. They were well managed and kept good records of production and antimicrobial use. The withdraw period of antimicrobial use was done before at least 30 days of slaughtering.

2.2 Animal selection

Ten clinical healthy pigs per farm from different litters were included and continuously observed at five periods: creeping period (0 to 28 days), nursery (>28days to 70 days) growing period (>70 days to 112 days) finishing (112 days until slaughtering) and slaughters were collected from thigh area of carcass at standard abattoirs.

2.3 Sample collection and bacterial identification

The sampling protocol was approved by the Chulalongkorn University Animal Care and Use Committee (permit number 58/2558). For creeping and nursery pigs, rectal swabs were directly used and kept in Clary-Blair transport medium. For growing and finishing, at least 25 g of rectal feces were collected into sterile plastic container. For

carcass, at least 25 g of pork at thigh area were cut by sterilized blade and kept into sterilized container. All samples were delivered to the laboratory at 4°C within 24 hours. The rectal swabs were soaked in 0.85% sodium chloride solution (normal saline solution:NSS) and directly spread on the Eosine Methylene Blue (EMB) (Oxoid, UK) agar (Renoux and Terdjman, 1951). For rectal feces, at least 5 g were diluted 10-fold dilution till 10^{-4} and spread the solution EMB agar. After grinding process, one gram of harmonized pork was drowned in 9 ml of sterile normal saline. After vigorously checking, the suspensions were spread on EMB agar at first dilution (Demarco and Lim, 2002; Stampi et al., 2004; USDA, 2017). Colony presented metallic sheen on EMB plates to select the major population of bacteria. *E. coli* colonies were identified by their IMViC (Oxoid) biochemical reactions, comprising an indole test (+), a methyl red test (+), a Voges-Proskauer test (-), and a citrate test (-) (Clark et al., 1957). A representative pure colony from the highest dilution plate was randomly selected for further investigation.

2.4 Antibiogram and extended spectrum beta-lactamase phenotype confirmation

The minimal inhibitory concentration of antimicrobials for *E. coli* were determined using AST-GN 3 8 test kit in the Vitek2 compact automated susceptibility level detection apparatus (BioMérieux, France). The 19 antimicrobials or combination of antimicrobials were amikacin (AK), amoxicillin (AMX), ampicillin (AMP), cefalexin (CEX), cefpirome (CPR), cefpodoxime (CPD), ceftiofur (XNL), chloramphenicol (C), enrofloxacin (ENR), gentamicin (GEN), imipenem (IMP), marbofloxacin (MBR), nitrofurantoin (NIT), piperacillin (PIP), tiamulin (TI), tetracycline (TET), tobramycin (TOB) and trimethoprim/ Sulfamethoxazole (SXT) (Lugsomya et al., 2017).The set of antimicrobial array in card were harmonized to match with veterinary drug guidelines (Plumb, 2015a). *E. coli* ATCC 25922, *Pseudomonas aeruginosa* ATCC 27853 and *Staphylococcus aureus* ATCC 25913 were used as the control strains. The interpretation of the susceptibility levels for AMP, CPD, CPR, XNL, GEN, ENR, MBR, TE were performed following Clinical Laboratory Standards (CLSI) for antimicrobial disks and testing for bacteria isolated from animals (VET2-0S3)(CLSI, 2015a), and interpretation for AMX, PIP, AMC, CEX, IMP, AK, TM, NT,

SXT were followed the CLSI standards for antimicrobial susceptibility testing (M100-S25) (CLSI, 2015b).

2.5 Extended Spectrum Beta-lactamase (ESBL) phenotypic screening and confirmatory test

ESBL producing (ESBLP) *E. coli* was first screened by Vitek2 machine (BioMérieux, France) (Espinar et al., 2011) and confirmed by the combination disk test (CDT) by CLSI standard recommendation (CLSI, 2015b). The bla_{CTX-M} genes comprising variant $bla_{CTX-M-1}$, $bla_{CTX-M-2,}$ $bla_{CTX-M-8,}$ $bla_{CTX-M-9}$, and $bla_{CTX-M-25/26}$.were detected by multiplex PCR in all ESBLP strains (Xu et al., 2005). The identity of representative PCR amplicons was confirmed by DNA-sequencing and analyzed using Mega 7.0 (Kumar et al., 2016) with comparisons made to the GenBank database to confirm whether bla_{CTX-M} genes were located on transmissible plasmids, a conjugation assay was performed using the broth mating technique, as previous studies (Gray et al., 2006). The three representative selected donor clones, *E. coli* PCU12-4 (positive for $bla_{CTX-M-1}$group with single IncI1-Iγ replicon), *E. coli* PCU12-5 (positive for $bla_{CTX-M-1}$group with single IncHI2 replicon), PCU12-6 (positive for $bla_{CTX-M-9}$ group with single IncHI2 replicon) were selected. The recipient strain *E. coli* J53 was resistant to sodium azide: Azi^r and susceptible to cefotaxime. The transconjugants were selected on Luria Bertani (LB) agar (Oxiod, UK) supplemented with cefotaxime (2 μg ml^{-1}) and sodium azide (100 μg ml^{-1}) (Oxoid, UK). Antimicrobial susceptibility, ESBL confirmatory phenotype, PCR detection and DNA sequencing of bla_{CTX-M} genes were performed on the transconjugants (Gray et al., 2006).

2.6 Gene encoding antimicrobial resistance detection

Bacterial DNA was extracted using a Wizard ® Genomic DNA Purification Kit (Promega, Germany). A total of 16 pairs of primers set specific for resistance genes in the bacteria in superfamily *Enterobacteriaceae* were generated (First Base Laboratoies. Sdn Bhd, Malaysia) and PCR thermal cycling conditions performed following the previous recommendations. The resistance genes were included bla_{TEM} and bla_{PSE-1} for ampicillin, amoxicillin and piperacillin resistance, *aadA1* and *aadA2* for streptomycin resistance, *aadB* for tobramycin and gentamicin resistance, *tet*(A) and *tet*(B) for tetracycline

resistance, *dfrA1*, *dfrA10* and *dfrA12* for trimethoprim resistance, *sul1*, *sul2*, *sul3* for sulfonamide resistance and *catA*, *catB* and *cmlA* for chloramphenicol resistance (Chuanchuen et al., 2010). A representative positive PCR amplicon for each gene was submitted for DNA-sequencing and analyzed by MEGA 7.0 (Kumar et al., 2016).

2.7 Plasmid replicon characterization

The plasmid replicons in *Enterobacteriaceae* composing IncF (IncFIA, IncFIB, IncFIC, IncFrep), IncI1-Iɣ, IncN, IncP, IncW, IncHI1, IncHI2, IncL/M, IncT, IncA/C, IncK, IncB/O, IncX and IncY was detected using five multiplexes and three simplexes PCR tests. The primers, PCR conditions and thermal cycles were proceeded as the previous studies (Carattoli et al., 2005). Representative positive PCR amplicons for each replicon were submitted for DNA sequencing and analyzed as described above.

2.8 Molecular strain typing by pulse field gel electrophoresis (PFGE)

Pulse field gel electrophoresis was performed following Centers of Diseases Control and Prevention (CDC) standard protocol (CDC, 2013). Briefly, whole *E. coli* DNA was randomly cut by X*ba*I restriction enzymes (Sibenzyme, Russia). Gel electrophoresis was run in 200 V field which included Angle: 120° for 18-19 hours and *Salmonella* serovar Braenderup H9812 were used as standard for proceeding protocol. The DNA band patterns were visualized by ethidium bromide staining. The dendrograms were computed by GeneTool program (Syngene, India) and analyzed by GeneDirectory program (Syngene, India).

Table 8 Primer lists for *E. coli* HiMLST

Target sites	Names of primers	Sequences	Product size
adk	adk_eF_univ_F	GACACTATAGATTCTGCTTGGCGCTCCGGG	604 bp
	adk_eR_univ_R	CACTATAGGGCCGTCAACTTTCGCGTATTT	
fumC	fumC_eF_univ_F	GACACTATAGGCGGCAAAAGTTAATGAAGA	580 bp
	fumC_eR_univ_R	CACTATAGGGTCCGGATGGGTATTTAGTCC	

Target sites	Names of primers	Sequences	Product size
gyrB	gyrB_eF_univ_F	GACACTATAGAGTGATCATGACCGTTCTGC	567 bp
	gyrB_eR_univ_R	CACTATAGGGCGGAATGTTGTTGGTAAAGC	
icd	icd_eF_univ_F	GACACTATAGAAGGTGATGGAATCGGTGTA	627 bp
	icd_eR_univ_R	CACTATAGGGTTCATGATGTTGCCTTTGTG	
mdh	mdh_eF_univ_F	GACACTATAGGCGCAGATGTCGTTCTTATC	526 bp
	mdh_eR_univ_R	CACTATAGGGAGACAGACCAAAACGTGCAG	
purA	purA_eF_univ_F	GACACTATAGATGTCCGCTGATCCTTGAT	605 bp
	purA_eR_univ_R	CACTATAGGGAATTCGTTACCCTGCTTGC	
recA	recA_eF_univ_F	GACACTATAGGTTTATCGATGCTGAACACG	594 bp
	recA_eR_univ_R	CACTATAGGGCTTCTCTTTTACGCCCAGGT	

2.9 Molecular strain typing by Multilocus sequence typing (MLST)

DNA sequence of 7 housekeeping genes [Adenylate kinase (*adk*), Adenylosuccinate dehydrogenase (*purA*), ATP/GTP binding motif (*recA*), DNA gyrase (*gyrB*), Fumarate hydratase (*fumC*), Isocitrate/isopropylmalate dehydrogenase (*icd*), Malate dehydrogenase (*mdh*)] were assigned to allelic profile for sequence type (STs) of *E. coli* (Adiri et al., 2003). By using HiMLST with 454 sequencing and multiplex identifier (MID), *E. coli* 300 isolates were sequenced and MLST profile of each individual isolate was generated by unique MID (Roche, Switzerland). The target genes were amplified by two-step PCR using primer sequence of target genes includes universal tail sequence primer at 5' end and MID sequence primer with *454* sequencing-specific nucleotides. The PCR products were pooled, clonally amplified by emulsion PCR (emPCR) (Roche, Switzerland) and sequencing using GS junior (Roche, Switzerland). Allele and sequence type (STs) were assigned at the publicly accessible *E. coli* MLST database at http://mlst.warwick.ac.uk/mlst/dbs/Ecoli.

Part 3 The evaluation of flavomycin to reduce resistance characteristics and plasmid transfer. in vitro and in vivo.

3.1 in vitro reduction of plasmid transferability test

3.1.1 bacterial donor strains

20 Bacterial strains were selected ESBL P producing *E. coli* which confirmed the *bla* $_{CTX-M-1}$group and plasmid replicon type existence by PCR (Carattoli et al., 2005; Xu et al., 2005).

Table 9 Bacterial strains which were used in in vitro reduction of plasmid transferability test

No	Strains Name	Resistance gene	Plasmid replicon types
1	*E. coli* PCU13	*bla*$_{CTX-M-1}$group	single IncHI2
2	*E. coli* PCU14	*bla*$_{CTX-M-1}$group	single Inc HI2
3	*E. coli* PCU15	*bla*$_{CTX-M-1}$group	single Inc HI2
4	*E. coli* PCU16	*bla*$_{CTX-M-1}$group	single IncHI2
5	*E. coli* PCU17	*bla*$_{CTX-M-1}$group	single IncHI2
6	*E. coli* PCU18	*bla* $_{CTX-M-1}$group	single IncHI2
7	*E. coli* PCU19	*bla*$_{CTX-M-1}$group	single Inc HI2
8	*E. coli* PCU20	*bla*$_{CTX-M-1}$group	single Inc HI2
9	*E. coli* PCU21	*bla*$_{CTX-M-1}$group	single IncHI2
10	*E. coli* PCU22	*bla*$_{CTX-M-1}$group	single IncHI2
11	*E. coli* PCU23	*bla*$_{CTX-M-9}$group	single IncHI2
12	*E. coli* PCU24	*bla*$_{CTX-M-9}$group	single Inc HI2
13	*E. coli* PCU25	*bla*$_{CTX-M-9}$group	single Inc HI2
14	*E. coli* PCU26	*bla*$_{CTX-M-9}$group	single IncHI2
15	*E. coli* PCU27	*bla*$_{CTX-M-9}$group	single IncHI2
16	*E. coli* PCU28	*bla*$_{CTX-M-9}$group	single IncHI2

17	*E. coli* PCU29	$bla_{CTX-M-9}$group	single Inc HI2
18	*E. coli* PCU30	$bla_{CTX-M-9}$group	single Inc HI2
19	*E. coli* PCU31	$bla_{CTX-M-9}$group	single IncHI2
20	*E. coli* PCU32	$bla_{CTX-M-9}$group	single IncHI2

3.1.2 bacterial recipient strains

E. coli strain J53 were induced to resisted sodium azide (MIC level >512) were used as the recipient. The recipients was confirmed that the susceptible to colistin ($\leq$2 ug/ml), cefatazidime ($\leq$2 ug/ml), and cefotaxime ($\leq$2 ug/ml), by agar dilution test

3.1.3 Resistance gene transfer

The donor and recipient bacteria mixed together in Luria Bertani (LB) broth (Oxoid, UK) with Flavomycin® 8 μg/ml, (experimental group I) 16 μg/ml (experimental group II) and non-added up (control group) and incubate for 24 hours in 37 ^{o}C The transconjugants will be selected on Bertani (LB) agar (Oxoid, UK) supplemented with cefotaxime (2 μg/ml)+sodium azide (100 μg/ml) bla_{CTX-M} plasmids (Gray et al., 2006). The number of colonies which were appeared on agar were counted and randomly sampled for confirmed the transferability by PCR method (Xu et al., 2005) The resistance gene transferability comparison between experimental group (group 1 : 10 *E. coli* strains which was detected $bla_{CTX-M-1}$ group genes and group 2 : 10 *E. coli* strains which was detected $bla_{CTX-M-9}$ group genes) and control group The resistance gene transferable ability were determined by detecting the efficacy of conjugation between experimental group and control group.

$$\text{Conjugation efficiency} = \frac{\textbf{Numbers of transcojugants of selectve media}}{\textbf{Numbers od Donors}}$$

3.1.4 Data analysis

The number of transconjugants colonies were compared between control group and two of experimental groups will be statistical analyze by Wilcoxon signed ranked test. The transconjugation efficiency were reported by descriptive analysis.

3.2 in vivo reduction of plasmid transferability test

3.2.1 Animal selection

A total of 10 pigs from AF farm in part II were selected for longitudinally monitored at at four periods: creeping period (0 to 28 days), nursery (>28days to 70 days) growing period(>70 days to 112 days) and finishing (112 days before slaughtering). Bambermycin (flavomycin, trade name : Flavomycin®) was mixed in feed at 10 ppm feed also added the routine antimicrobials formula at nursery and grower periods (combination of tiamulin and amoxicillin at 100 ppm and 250 ppm).

3.2.2 Sample collection and bacterial identification

The sampling protocol was approved by the Chulalongkorn University Animal Care and Use Committee (permit number 58/2558). For creeping and nursery pigs, rectal swabs were directly used and kept in Clary-Blair transport medium. For growing and finishing, at least 25 g of rectal feces were collected into sterile plastic container. The rectal swabs were soaked in 0.85% sodium chloride solution (NSS) and directly spread on the Eosine Methylene Blue (EMB) (Oxoid, UK) agar (Renoux and Terdjman, 1951). For rectal feces, at least 5 g were diluted 10-fold dilution till 10^{-4} and spread the solution EMB agar. After grinding process, one gram of harmonized pork was drowned in 9 ml of sterile normal saline. After vigorously checking, the suspensions were spread on EMB agar at first dilution (Demarco and Lim, 2002; Stampi et al., 2004; USDA, 2017). Colony presented metallic sheen on EMB plates to select the major population of bacteria. *E. coli* colonies were identified by their IMViC (Oxoid) biochemical reactions, comprising an indole test (+), a methyl red test (+), a Voges-Proskauer test (-), and a citrate test (-) (Clark et al., 1957). A representative pure colony from the highest dilution plate was randomly selected for further investigation.

3.2.3 Antibiogram and extended spectrum beta-lactamase phenotype confirmation

The minimal inhibitory concentration of antimicrobials for *E. coli* were determined using AST-GN 38 test kit in the Vitek2 compact automated susceptibility level

detection apparatus (BioMérieux, France). The 18 antimicrobials were amikacin (AK), amoxicillin (AMX), ampicillin (AMP), cefalexin (CEX), cefpirome (CPR), cefpodoxime (CPD), ceftiofur (XNL), chloramphenicol (C), enrofloxacin (ENR), gentamicin (GEN), imipenem (IMP), marbofloxacin (MBR), nitrofurantoin (NIT), piperacillin (PIP), tetracycline (T), tobramycin (TOB) and trimethoprim/ Sulfamethoxazole (SXT) (Lugsomya et al., 2017).The set of antimicrobial array in card were harmonized to match with veterinary drug guidelines (Plumb, 2015a). *E. coli* ATCC 25922, *Pseudomonas aeruginosa* ATCC 27853 and *Staphylococcus aureus* ATCC 29513 were used as the control strains. The interpretation of the susceptibility levels for AMP, CPD, CPR, XNL, GEN, ENR, MBR, TE were performed following Clinical Laboratory Standards (CLSI) for antimicrobial disks and testing for bacteria isolated from animals (VET2-0S3)(CLSI, 2015a), and interpretation for AMX, PIP, AMC, CEX, IMP, AK, TM, NT, SXT were followed the CLSI standards for antimicrobial susceptibility testing (M100-S25) (CLSI, 2015b).

3.2.4 Extended Spectrum Beta-lactamase (ESBL) phenotypic screening and confirmatory test

ESBL producing (ESBLP) *E. coli* was first screened by Vitek2 machine (BioMérieux, France) (Espinar et al., 2011) and confirmed by the combination disk test (CDT) by CLSI standard recommendation (CLSI, 2015b). The bla_{CTX-M} genes comprising variant $bla_{CTX-M-1}$, $bla_{CTX-M-2,}$ $bla_{CTX-M-8,}$ $bla_{CTX-M-9}$, and $bla_{CTX-M-25/26}$.were detected by multiplex PCR in all ESBLP strains (Xu et al., 2005). The identity of representative PCR amplicons was confirmed by DNA-sequencing and analyzed using Mega 7.0 (Kumar et al., 2016) with comparisons made to the GenBank database To confirm whether bla_{CTX-M} genes were located on transmissible plasmids, a conjugation assay was performed using the broth mating technique, as previous studies (Gray et al., 2006).

3.2.5 Gene encoding antimicrobial resistance detection

Bacterial DNA was extracted using a Wizard ® Genomic DNA Purification Kit (Promega, Germany). A total of 16 pairs of primers set specific for resistance genes in the bacteria in superfamily *Enterobacteriaceae* were generated (First Base Laboratoies.

Sdn Bhd, Malaysia) and PCR thermal cycling conditions performed following the previous recommendations. The resistance genes were included bla_{TEM} and bla_{PSE-1} for ampicillin, amoxicillin and piperacillin resistance, *aadA1* and *aadA2* for streptomycin resistance, *aadB* for tobramycin and gentamicin resistance, *tet*(A) and *tet*(B) for tetracycline resistance, *dfrA1*, *dfrA10* and *dfrA12* for trimethoprim resistance, *sul1*, *sul2*, *sul3* for sulfonamide resistance and *catA*, *catB* and *cmlA* for chloramphenicol resistance (Chuanchuen et al., 2010). A representative positive PCR amplicon for each gene was submitted for DNA-sequencing and analyzed by MEGA 7.0 (Kumar et al., 2016).

3.2.6 Plasmid replicon characterization

The plasmid replicons in *Enterobacteriaceae* composing IncF (IncFIA, IncFIB, IncFIC, IncFrep), IncI1-Iγ, IncN, IncP, IncW, IncHI1, IncHI2, IncL/M, IncT, IncA/C, IncK, IncB/O, IncX and IncY was detected using five multiplexes and three simplexes PCR tests. The primers, PCR conditions and thermal cycles were proceeded as the previous studies (Carattoli et al., 2005). Representative positive PCR amplicons for each replicon were submitted for DNA sequencing and analyzed as described above.

3.2.7 Data analysis

Association of the resistance rates and genetic characteristics between pairs (Flavomycin® added up in feed) FF groups and NF group (non Flavomycin® added up in feed) data was retrieved in part II were compared between each growing period by Chi square test.

CHAPTER V: Results

Part 1. Distribution of resistance characteristics of *E. coli* isolated from pigs in different antibiotic using status in Thailand

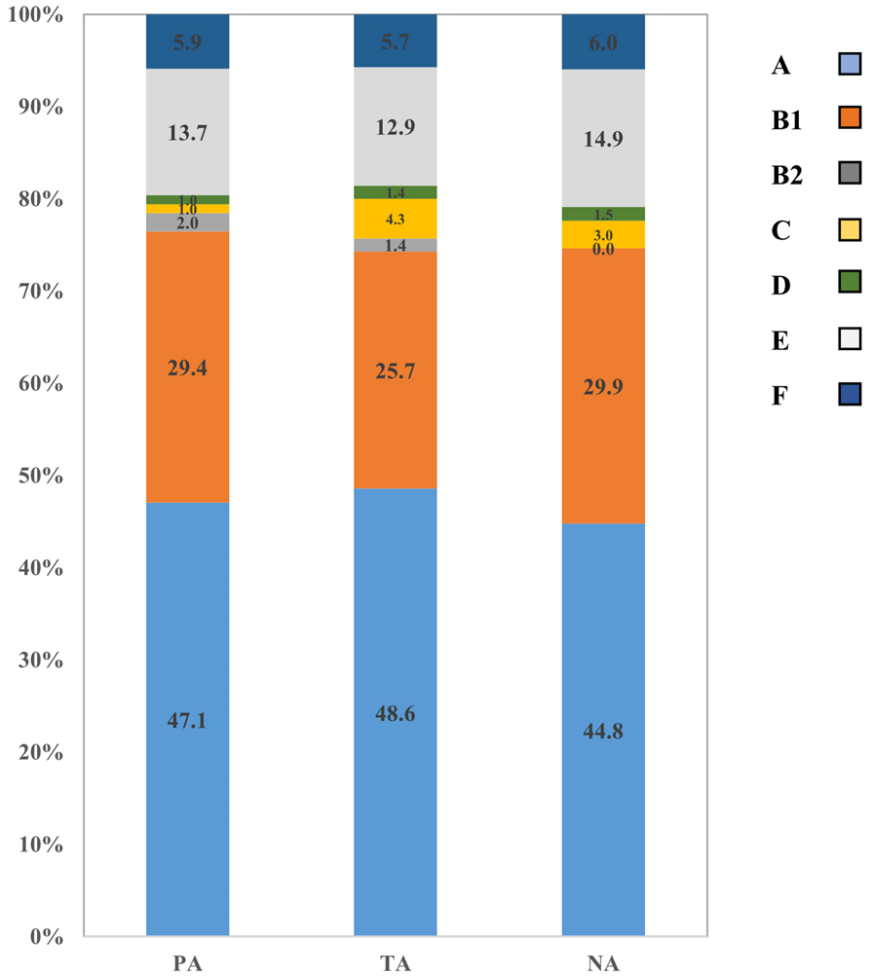
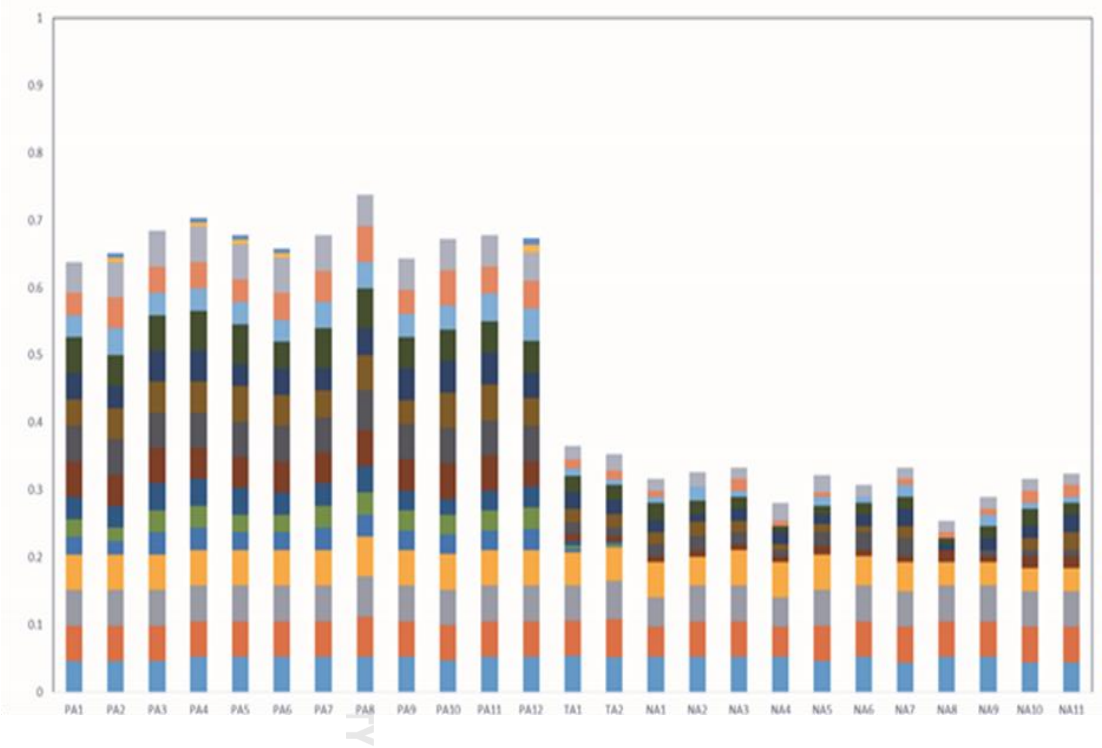


Figure 1 The relative abundance of phylogroup in porcine commensal *E. coli* from the three categories of farms. Vertical bar, PA, TA, and NA. PAs, prophylactic antimicrobials; TAs, therapeutic antimicrobials and NAs, no antimicrobials; .

1.1 Distribution of phylogroups

The distribution of phylogroups of the commensal *E. coli* tested from the three categories of farms is shown in Figure 1. The types and proportion of phylogroups of the selected *E. coli* colonies were quite similar in all farms (p = 0.894). Phylogroup A (PA:

47.1%, TA: 48.6%, NA:44.8%), B1 (PA: 29.4%, TA: 25.7%, NA: 29.9%), and E (PA: 13.7%, TA: 12.9%, NA: 14.9%) were the most commonly detected groups. Phylogroups B2, C, D, and F were minor components and in total were detected at <14% in each farm category (PA: 9.8%, TA: 12.9%, and NA:10.4%). Representatives of the cryptic clade group were not found.



	P value		
	PA	TA	NA
amikacin	-	-	-
imipnem	-	-	-
amoxicillin /clavulonic acid	0.22	-	-
cefpirome	0.22	-	-
tiamulin	0.70	0.78	0.50
gentamicin	0.80	0.89	0.88
tobramycin	0.98	0.46	0.73
enrofloxacin	0.74	0.36	0.98
marbofloxacin	0.27	0.36	0.98
nitrofurantoin	0.44	0.57	0.53
chloramphenicol	1.00	0.50	0.57
trimethroprim /sulfamethoxazole	0.66	0.36	-
cefalexin	0.99	0.60	-
cefpodoxime	0.99	0.60	-
ceftiofur	0.00	0.60	-
tetracycline	0.44	0.93	0.44
amoxicillin	1.00	0.30	0.43
ampicillin	1.00	0.30	0.43
piperacillin	1.00	0.95	0.75

Figure 2 The relative abundance of antibiotic resistance rate in each farms and comparison resistance rate between farms in the same group using the Kruskal-Wallis test.

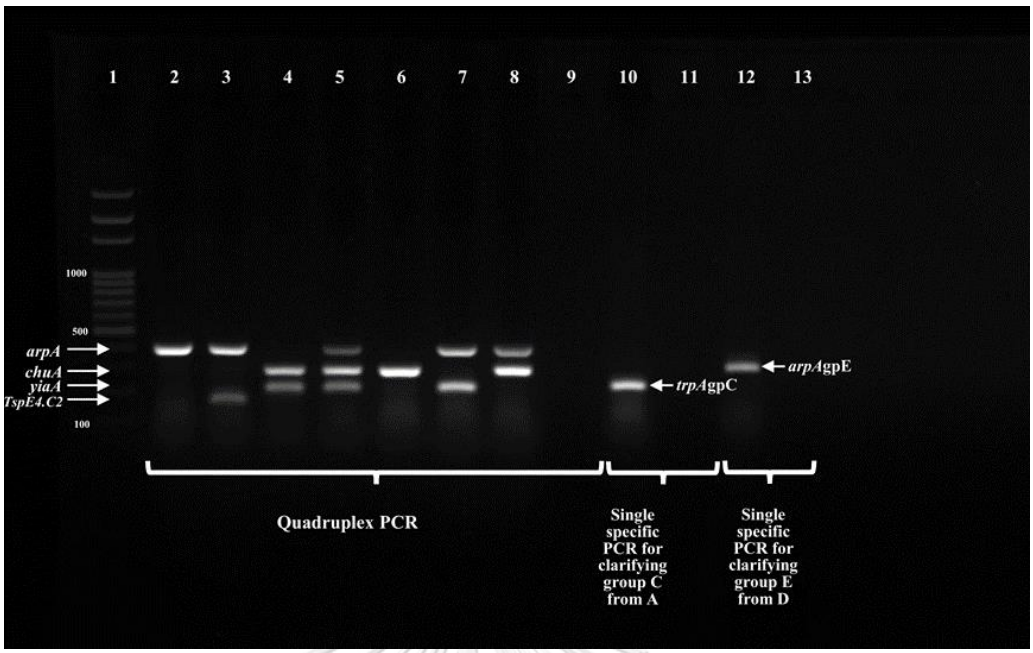


Figure 3 Phylogenetic grouping PCR product in 1% agarose gel electrophoresis. Legend : lane 1, 100 base pair marker (Solis BioDyne, Estonia) ; [lane 2-9 : quadruplex pcr]: lane 2, group A : (+ - - -) ; lane 3, group B1 : (+ - - +) ; lane 4, group B2 : (- + + -) ; lane 5, group E : (+ + + -) ; lane 6, group F : (- + - -) ; lane 7, group A or C : (+ - + -) ; lane 8, group E or D : (+ + - -) ; lane 9, Escherichia fergusonii strain CU MIC427 (negative control) : (- - - -) , [lane 10,11 : specific pcr for clarifying between group A and C] : lane 10, group C : (+) ; lane 11, group A : (-) , [lane 12,13 : specific pcr for clarifying between group E and D] : lane 12, group E : (+) ; lane 13, group D : (-)

1.2 Distribution of phenotypic AMR rates

The distribution of phenotypic resistance rates for the commensal *E. coli* against the antimicrobials tested in the three categories of farm is shown in Figure 4 Amikacin and imipenem resistant *E. coli* were not found in any of the farms, and resistance to amoxicillin/clavulanic and cefpirome was found only in the PA farms and was uncommon. The isolates from the three categories of farms all had very high rates of resistance to tetracycline, amoxicillin, ampicillin and piperacillin (over 83.6%), and these rates did not differ significantly between the farm categories. The isolates from the PA farms had

resistance rates to the other antimicrobials tested that varied from 52.9 to 100%, whilst rates were much lower (0 to 42.3%) for the various antimicrobials in the other two farm categories. The rates in the PA farms were statistically significantly higher than those in both the TA and NA farms but were not significantly different between the latter two categories. Within each of the three categories of farm, resistance rates for each of the drugs between farms did not reveal any significant farm related differences (Figure 2). Over 50% (64/102) of the PA isolates were resistant to gentamicin, tobramycin, enrofloxacin, marbofloxacin, nitrofurantoin, chloramphenicol and sulfamethoxazole/ trimethoprim. The antibiogram AMX-AMP-PIP-CEX-CPD-XNL-GM-TM-ENR-MBR-TET-NT-C-SXT-TI was the most prevalent type found (n=28, 27.5%) in the isolates from the PA farms. On the other hand, 18.6-41.4% of isolates from the TA farms were resistant to gentamicin, tobramycin, nitrofurantoin, enrofloxacin, marbofloxacin, chloramphenicol and sulfamethoxazole/ trimethoprim. The antibiogram AMX-AMP-PIP-ENR-MBR-TET-NT-C-SXT-TI was the most prevalent type found (n= 12, 17.1%) in *E. coli* from TA farms, whilst AMX-AMP-PIP-TE-C was the most prevalent type found in isolates from the NA farms (n=10, 14.9%).

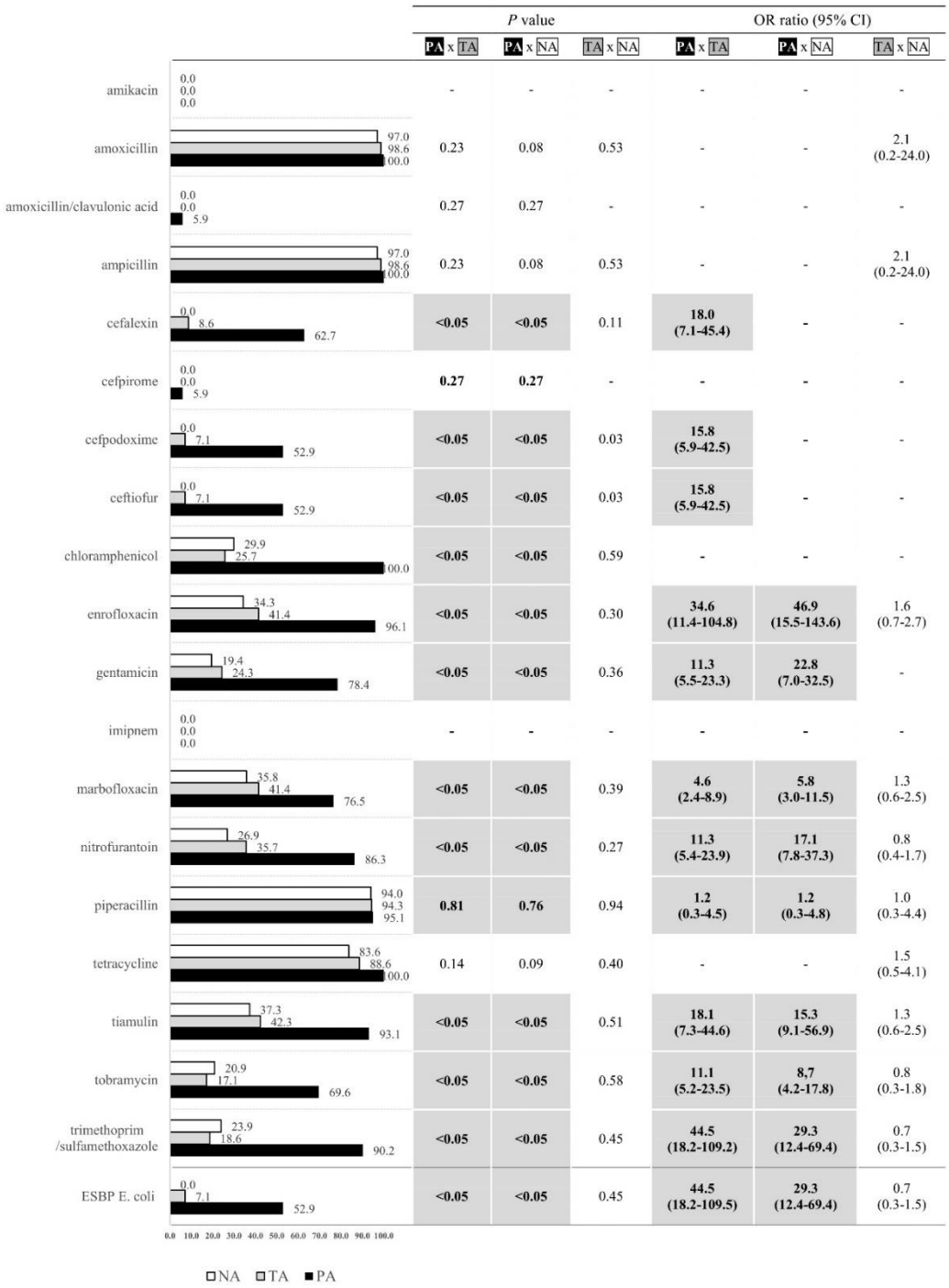
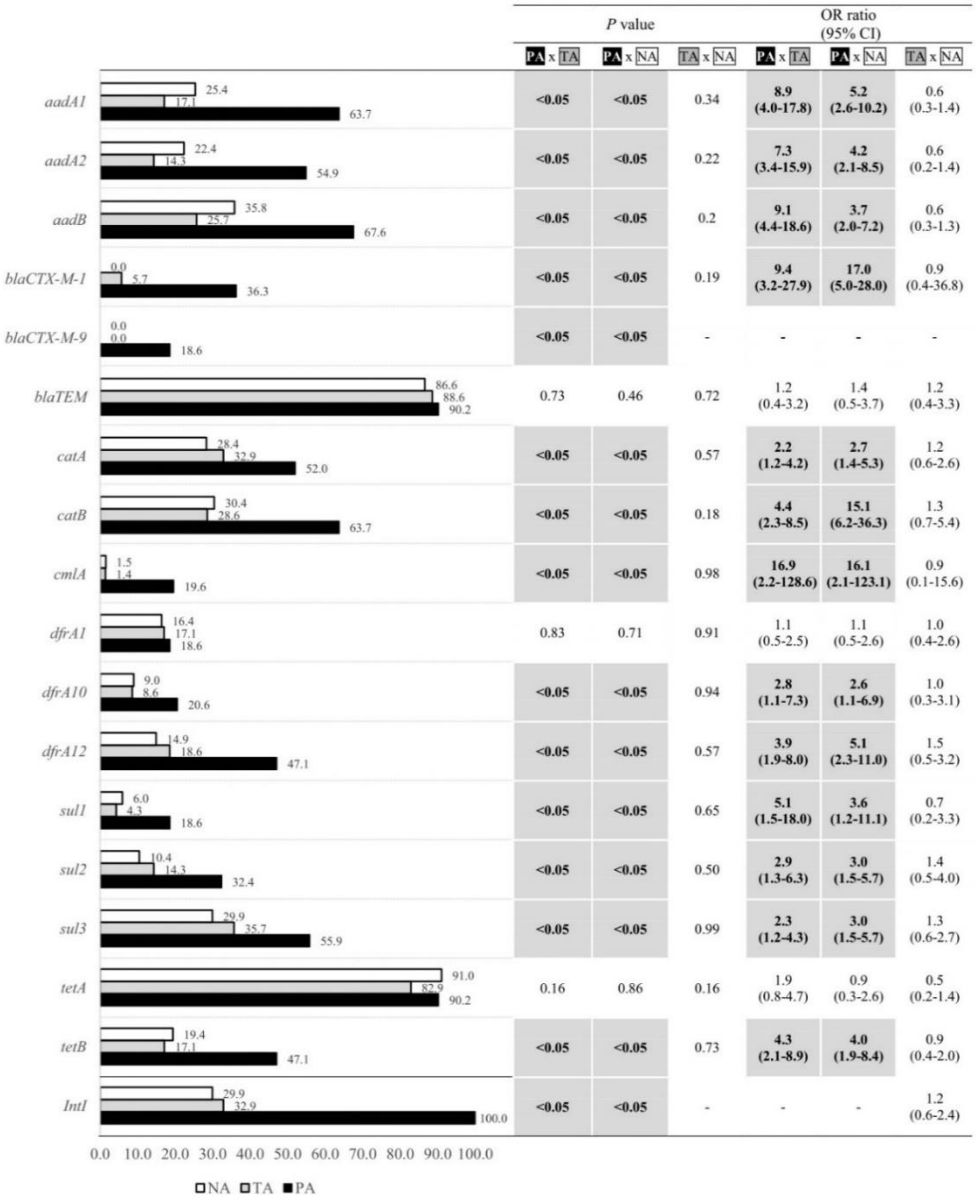


Figure 4 Comparison of rates of antimicrobial resistance in porcine commensal *E. coli* from three categories of farms Legend: Open horizontal bar, no antimicrobials (NA); grey bar, therapeutic antimicrobials (TA); black bars, prophylactic antimicrobials (PA).

1.3 Antimicrobial resistance genotypes

The resistance genes bla_{TEM} and tet*(A)* were the most common type found amongst *E. coli* isolates from pigs on all farms (over 82.9%), with no significant differences in rates between the three farm categories (Figure 5). The gene $bla_{CTX\text{-}M\text{-}9}$ was only found in isolates from PA farms, and at a low rate. The other resistance genes, except for *dfrA*, were significantly more common in isolates from the PA farms than from the other two categories. Rates in TA and NA farms did not differ significantly from each other. The percentage of isolates containing *catA*, *catB*, *dfrA12*, *sul3*, *tet*(B) *aadA1*, *aadA2*, and *aadB* ranged from 47.1 to 67.6% in PA farms, whereas they were only present at 14.3-35.8% in isolates from the other two farm categories (Figure 5).



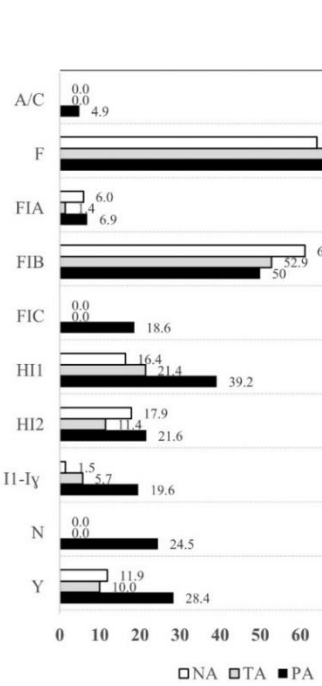
	P value			OR ratio (95% CI)		
	PA x TA	PA x NA	TA x NA	PA x TA	PA x NA	TA x NA
aadA1	**<0.05**	**<0.05**	0.34	**8.9 (4.0-17.8)**	**5.2 (2.6-10.2)**	0.6 (0.3-1.4)
aadA2	**<0.05**	**<0.05**	0.22	**7.3 (3.4-15.9)**	**4.2 (2.1-8.5)**	0.6 (0.2-1.4)
aadB	**<0.05**	**<0.05**	0.2	**9.1 (4.4-18.6)**	**3.7 (2.0-7.2)**	0.6 (0.3-1.3)
blaCTX-M-1	**<0.05**	**<0.05**	0.19	**9.4 (3.2-27.9)**	**17.0 (5.0-28.0)**	0.9 (0.4-36.8)
blaCTX-M-9	**<0.05**	**<0.05**	-	-	-	-
blaTEM	0.73	0.46	0.72	1.2 (0.4-3.2)	1.4 (0.5-3.7)	1.2 (0.4-3.3)
catA	**<0.05**	**<0.05**	0.57	**2.2 (1.2-4.2)**	**2.7 (1.4-5.3)**	1.2 (0.6-2.6)
catB	**<0.05**	**<0.05**	0.18	**4.4 (2.3-8.5)**	**15.1 (6.2-36.3)**	1.3 (0.7-5.4)
cmlA	**<0.05**	**<0.05**	0.98	**16.9 (2.2-128.6)**	**16.1 (2.1-123.1)**	0.9 (0.1-15.6)
dfrA1	0.83	0.71	0.91	1.1 (0.5-2.5)	1.1 (0.5-2.6)	1.0 (0.4-2.6)
dfrA10	**<0.05**	**<0.05**	0.94	**2.8 (1.1-7.3)**	**2.6 (1.1-6.9)**	1.0 (0.3-3.1)
dfrA12	**<0.05**	**<0.05**	0.57	**3.9 (1.9-8.0)**	**5.1 (2.3-11.0)**	1.5 (0.5-3.2)
sul1	**<0.05**	**<0.05**	0.65	**5.1 (1.5-18.0)**	**3.6 (1.2-11.1)**	0.7 (0.2-3.3)
sul2	**<0.05**	**<0.05**	0.50	**2.9 (1.3-6.3)**	**3.0 (1.5-5.7)**	1.4 (0.5-4.0)
sul3	**<0.05**	**<0.05**	0.99	**2.3 (1.2-4.3)**	**3.0 (1.5-5.7)**	1.3 (0.6-2.7)
tetA	0.16	0.86	0.16	1.9 (0.8-4.7)	0.9 (0.3-2.6)	0.5 (0.2-1.4)
tetB	**<0.05**	**<0.05**	0.73	**4.3 (2.1-8.9)**	**4.0 (1.9-8.4)**	0.9 (0.4-2.0)
IntI	**<0.05**	**<0.05**	-	-	-	1.2 (0.6-2.4)

Figure 5 Comparison of antimicrobial resistance genes of porcine commensal *E. coli* from the three categories farms Legend: Open horizontal bar, no antimicrobials (NA); grey bar, therapeutic antimicrobials (TA); black bars, prophylactic antimicrobials (PA).

1.4 Plasmid replicon types and confirmation of bla_{CTX-M} gene conjugation

Plasmid replicons IncX, IncW, IncP, IncT, IncL/M, IncK, IncFIIAs and IncB/O were not detected in this study. The two most common types that were found were IncF and IncFIB,

and there were no significant differences in their rates of occurrence between the three categories of farm (Figure 6). Rates for IncHI2 were low and were not significantly different between the three categories of farms. Replicon types IncFIC, IncN and IncA/C were only found in isolates from PA farms. All the other replicon types, whilst at a low prevalence, were significantly more common in isolates from the PA farms than in the other two categories, which did not differ significantly from each other. The *intI* gene was found at a significantly higher rate in isolates from the PA farms compared to amongst isolates from both the TA and NA farms, while the rates in TA and NA farms were similar (Figure 6). Conjugation assays showed that the IncF and IncFIB replicon plasmids from ESBLP were transferred with a frequency of $1.36x10^{-3}$ and $8.67x10^{-4}$ to transconjugants, respectively. The donor ESBLP strains *E. coli* PCU1 (positive for $bla_{CTX-M-1}$ group and with a single IncF replicon) and *E. coli* PCU2 (positive for $bla_{CTX-M-9}$ and with a single IncFIB replicon) transferred $bla_{CTX-M-1}$ group and $bla_{CTX-M-9}$ group to recipient *E. coli* strain J53, confirming the location of the genes on these conjugative plasmids. Using automated detection with confirmation by the combination disk assay, ESBLP *E. coli* were detected in 54 of the 102 (52.9%) *E. coli* from the PA farms, in 5 of 70 (7.1%) *E. coli* from the TA farms, and in none of the isolates from the NA farms (Table 10). Antibiograms, replicon type and bla_{CTX-M} genes of 59 ESBLP *E. coli* are shown in Table 1. ESBLP strains derived from PA farms either contained $bla_{CTX-M-1}$ group (35, 64.8%), $bla_{CTX-M-9}$ group (17, 31.5%) or both groups of genes (2, 3.7%). The five ESBLP *E. coli* from the TA farms only contained $bla_{CTX-M-1}$. The most common antibiogram for the ESBLP *E. coli* was AMX-AMP-PIP-CEX-CPD-XNL-GM-TM-ENR-MBR-TET-NT-C-SXT in the PA farms (n=28), and AMX-AMP-PIP-CEX-CPD-XNL-NT-SXT in the TA farms (n=2). The genes $bla_{CTX-M-2}$, $bla_{CTX-M-8}$, and $bla_{CTX-M-25/26}$ were not detected in this study. Replicons IncFIB (37.0%), F (22.1%) or IncN (18.5%) were commonly detected in the ESBLP *E. coli* from the PA farms, and FIB was the only replicon detected in ESBLP *E. coli* from the TA farms.



	P value			OR ratio (95% CI)		
	PA x TA	PA x NA	TA x NA	PA x TA	PA x NA	TA x NA
A/C	0.06	0.06	-	-	-	-
F	0.25	0.64	0.14	0.7 (0.3-1.3)	1.2 (0.6-2.2)	1.7 (0.8-3.6)
FIA	0.09	0.82	0.16	5.0 (0.6-42.3)	1.2 (0.3-4.1)	0.2 (0.1-2.1)
FIB	0.71	0.15	0.33	0.9 (0.5-1.6)	0.6 (0.3-1.2)	0.7 (0.4-1.4)
FIC	**<0.05**	**<0.05**	-	-	-	-
HI1	**<0.05**	**<0.05**	0.46	**2.4 (1.2-4.7)**	**3.3 (1.5-7.0)**	1.4 (0.6-3.3)
HI2	0.09	0.56	0.28	2.1 (0.9-5.1)	1.3 (0.3-2.8)	0.6 (0.2-1.6)
I1-Iγ	**<0.05**	**<0.05**	0.19	**4 (1.3-12.4)**	**16.1 (2.1-123.1)**	4 (0.4-36.7)
N	**<0.05**	**<0.05**	-	-	-	-
Y	**<0.05**	**<0.05**	0.72	**3.6 (1.5-8.7)**	**2.9 (1.2-6.9)**	0.8 (0.3-2.4)

Figure 6 Comparison of plasmid replicon types detected in porcine commensal *E. coli* among the three categories of farm. Legend: Open horizontal bar, no antimicrobials (NA); grey bar, therapeutic antimicrobials (TA); black bars, prophylactic antimicrobials (PA).

Table 10 Incidence, phenotypic and genetic characteristics of ESBLP *E. coli* derived from farms routinely used prophylactic antimicrobials (prophylactic antimicrobials: PA), only used antimicrobials therapeutically (therapeutic antimicrobials: TA) and never used antimicrobials (no antimicrobials: NA)

	PA	TA	NA	OR (95% CI) between PA and TA
Incidence rate	52.9%*	7.1%	0.0%	32.1 (12.1 to 85.2)
Common antibiograms	AMX-AMP-PIP-CEX-CPD-XNL-GM-TM-ENR-MBR-TE-NT-C-SXT (28) AMX-AMP-PIP-CEX-CPD-XNL-GM-ENR-MBR-TE-NT-C-SXT (6) AMX-AMP-PIP-CEX-CPD-XNL-GM-TM-ENR-MBR-TE-NT-C (4) AMX-AMP-AMC-PIP-CEX-CPD-XNL-GM-TM-ENR-MBR-TE-NT-C (4) AMX-AMP-PIP-CEX-CPD-XNL-ENR-TE-NT-C-SXT (4)	AMX-AMP-PIP-CEX-CPD-XNL-ENR-NT-C-SXT (2) AMX-AMP-PIP-CEX-CPD-ENR-MBR-TE-NT-C-SXT (1) AMX-AMP-PIP-CEX-CPD-XNL-ENR-MBR-TE-NT (1) AMX-AMP-PIP-CEX-CPD-XNL-ENR-TE-NT-C-SXT (1)	-	
bla_{CTX-M} genes	$bla_{CTX-M-1}$ 64.8% (35/54) $bla_{CTX-M-9}$ 31.4% (17/54) both bla_{CTX-1} and $bla_{CTX-M-9}$ 3.7% (2/54)	$bla_{CTX-M-1}$ 100% (5/5)	-	13.8 (5.2 – 36.8) ($bla_{CTX-M-1}$)
Common replicon types	IncFIB 37.0% (20/54) IncFrep 22.2% (12/54) IncN 18.5% (10/54) IncHI1 5.5% (3/54) IncHI1-Y 3.7% (2/54)	IncFIB 100% (5/5)	-	

* ESBLP incidence rate of PA was significantly higher than TA and NA by Chi-square test at $p<0.05$

Part 2. Longitudinal monitoring of molecular resistance genotypic of *E. coli* monitoring of from pig in the production cycle

2.1 Resistant phenotypic characterizations

A total of 300 *E. coli* isolates were isolated from pigs in this study. Overall, MDR were high in AF (73.3%) and NF (64.7%). The most common antibiogram, AMP-AMX-PIP-TET, was detected in 18.0% and 14.7% from NF and AF, respectively. Overall, antimicrobial resistant phenotypes are described in Fig 7. For β-lactams groups and tetracycline, all isolates were highly resisted in all period of observations in both farm types while *E. coli* resisted to amoxicillin-clavulanate were found only from AF in creeping (6.7%) and finishing (6.7%). In AF farm, the *E. coli* producing ESBL and resisted to all cephalosporin group were 43% in nursery period and ranged from 40.0 to 46.7% in growing period. The rates were reduced to 16.7% in finishing periods, and constantly maintained in pork samples (16.7-20.0%) in AF farms. In NF farms, cephalosporin resistant and ESBLP *E. coli* were consistently detected in the lower rates at all period of observations (creeping period: 10%, nursery: 10.0-16.7%, growing: 16.7-20.0%, finishing: 10.0-16.7%, pork: 16.7%). Regarding aminoglycosides, amikacin resistance was not found in all pigs, while gentamicin and tobramycin resistance were obviously raised in nursery and growing periods in AF farm (GEN and TOB: 56.7-66.7%) and declined in finishing period (GEN and TOB: 30.0%), whereas, the rates were slightly higher in pork. On the other hand, gentamicin and tobramycin steadily detected through growing period, finishing and pork in NF (GEN: 13.3-23.3%, TOB: 13.3-23.3%).

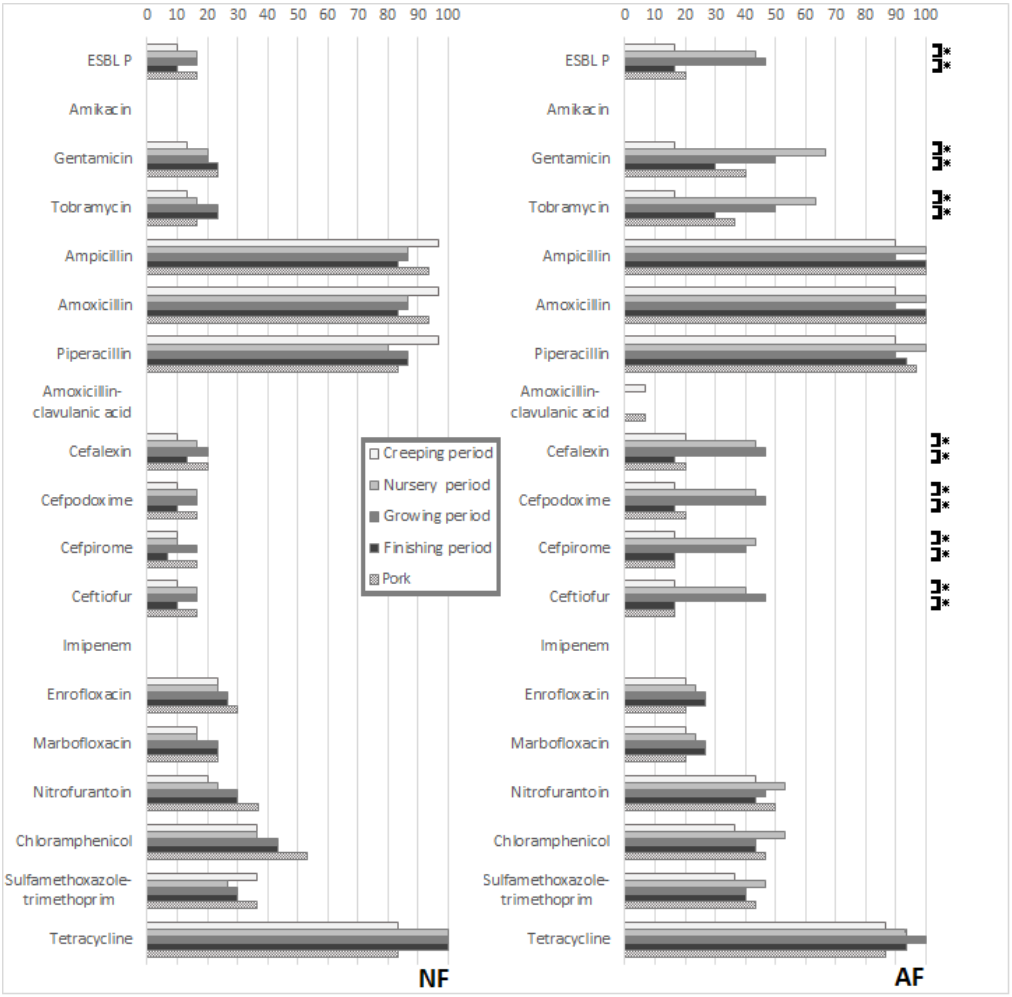


Figure 7 Eighteen antimicrobial resistance rates of and ESBLP *E. coli* which were individually collected from pigs in each growing period and meat period at abattoir in AF farm type and NF farm type. [* significant difference (p<0.05) between the concatenating period performed by Chi-square test]

2.2 Genotypic resistant characterization

The common resistance genes bla_{TEM} and *tet(A)* were found among *E. coli* from both types of farms. The genes resisted to sulfonamide; *sul1, sul2, sul3,* to trimethoprim; *drfA1*, *drfA10*, *drfA12* and to chloramphenicol *catA*, *catB* and *cmlA*, were presented in moderate levels without significant difference between the farm types and the observed periods (6.7-43.3%). Aminoglycoside resistant genes; *aadA1*, *aadA2* and *aadB* were raised to 46.7-66.7% in nursery and growing periods of AF whereas those of NF ranged only 13.3-20.7%. The significant difference of those genes was found between nursery-to-growing

and finishing (p<0.05). The frequency of $bla_{CTX\text{-}M\text{-}1}$ group was highest in AF at growing (36.7%) followed by nursery (33.3%) and pork (23.3%), respectively. While, the detection of $bla_{CTX\text{-}M2}$, $bla_{CTX\text{-}M8}$, $bla_{CTX\text{-}M9}$, $bla_{CTX\text{-}M25/26}$ genes were not different between the both farms. (Fig. 8).

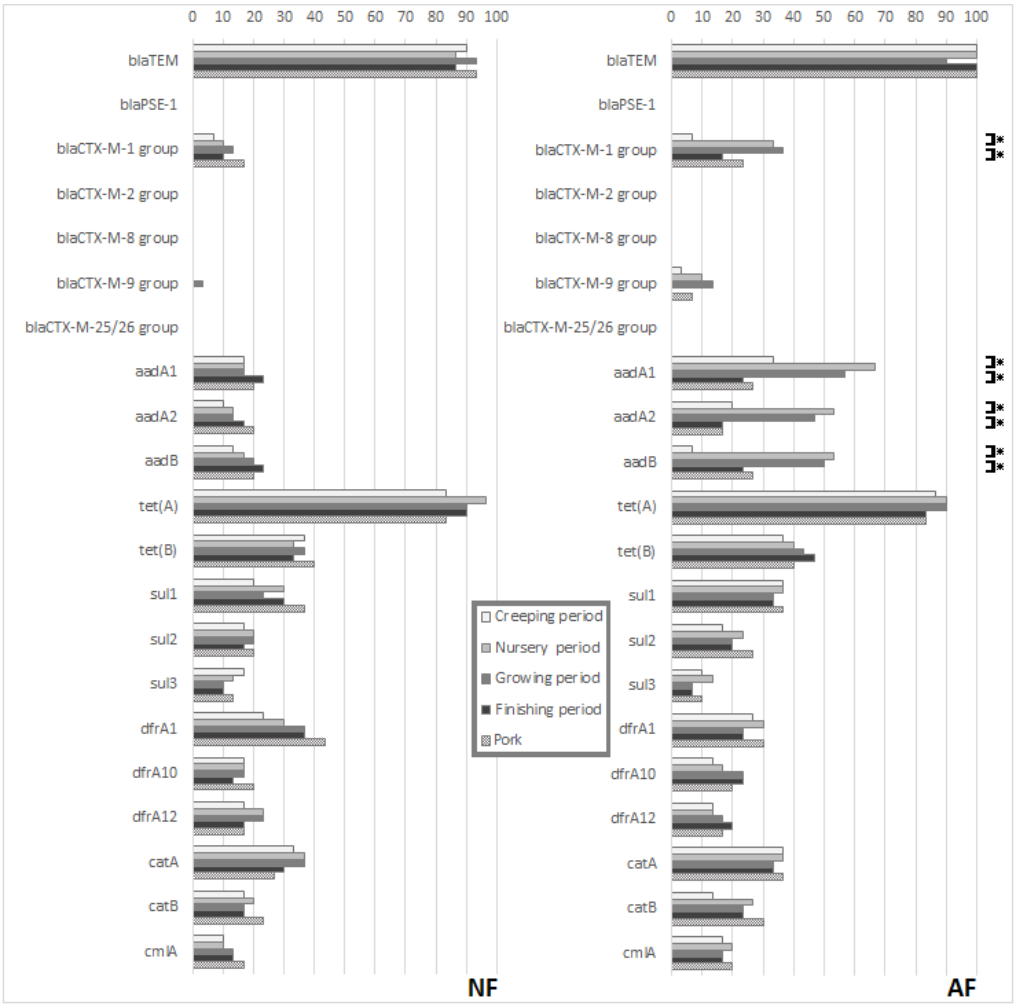


Figure 8 Twenty antimicrobial resistance gene detecting rates which were individually collected from pigs in each growing period and meat period at abattoir in AF farm type and NF farm type. [* significant difference (p<0.05) between the concatenating period performed by Chi-square test]

2.3 Plasmid replicon type detection

The prevalence of 18 replicon types detected from *E. coli* are presenting in Fig 9. Plasmid replicons IncX, IncT, IncP, IncL/M, IncK, IncFIIAs, IncA/C and IncB/O were not

detected in this study. The IncFrep and IncFIB replicons were commonly found with non-significance between any period individually farm type AF (IncFrep; p values were between 0.43 to 1.0, IncFIB; p values were between 0.41 to 0.79) and NF (IncFrep; p values were between 0.59 to 0.79, IncFIB; p values were between 0.6 to 0.79). IncW replicon was only found in the isolates from AF. All the other replicon types were found at a low to moderate prevalence in both farms. Besides, the rates for all replicons from NF data had no a significant difference throughout the periods of observation. While the frequency of IncHI-2 and IncI1-Iγ replicons during nursery to growing were significantly higher than that of AF. For the conjugation assay, *E. coli* PCU12-4 (positive for $bla_{CTX-M-1}$group with single IncI1-Iγ replicon) could transfer $bla_{CTX-M-1}$ group with the frequency of 3.8×10^{-5}, while *E. coli* PCU12-5 (positive for $bla_{CTX-M-1}$group with single IncHI2 replicon) and *E. coli* PCU12-6 (positive for $bla_{CTX-M-9}$ group with single IncHI2 replicon) could transfer bla_{CTX-M} gene with the frequency of 4.1×10^{-6} and 5.6×10^{-6}, respectively. These results confirmed the location of the genes on these conjugative plasmids.

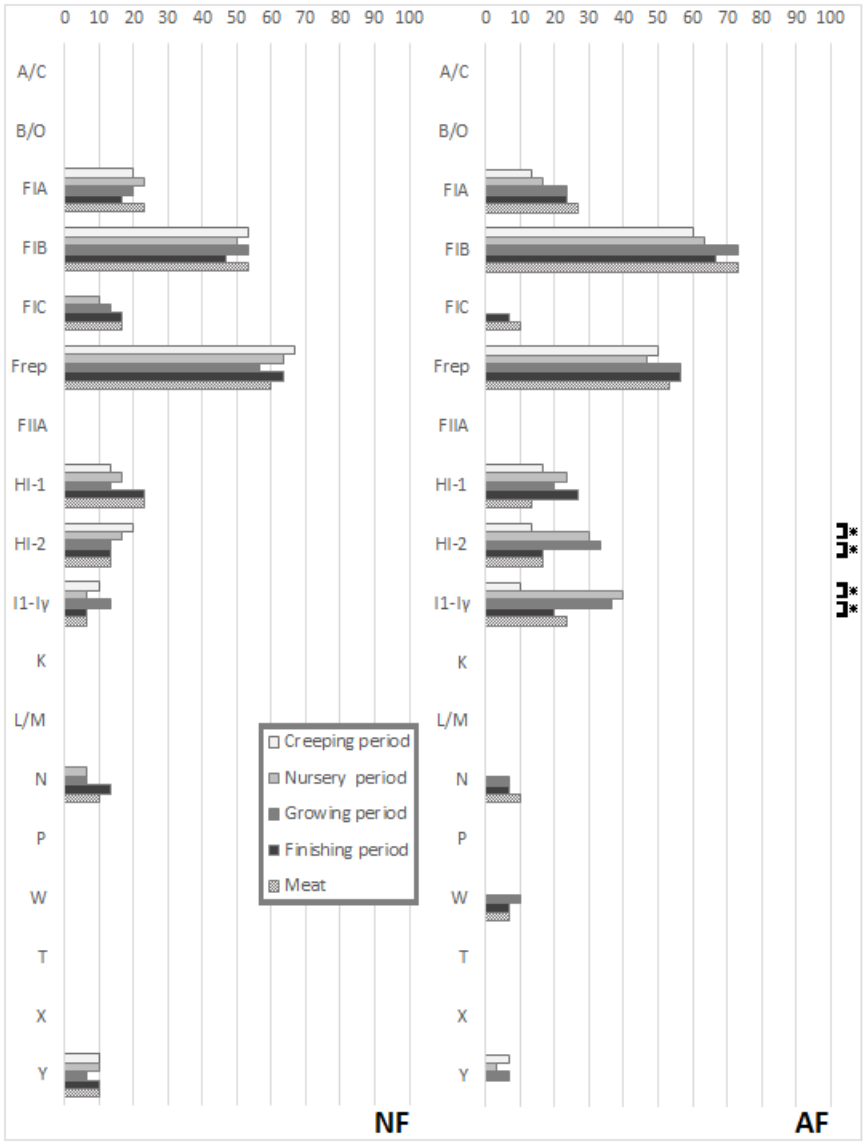


Figure 9 Eighteen plasmid replicon types detecting rates which were individually collected from pigs in each growing period and meat period at abattoir in AF farm type and NF farm type. [* significant difference (p<0.05) between the concatenating period performed by Chi-square test]

2.4 Molecular genotypic characterization

All 300 isolates divided from 240 isolates from pigs and 60 isolates from pork were analyzed their fingerprinting by PFGE and MLST analysis. A diversity of clone types was found comprising 24 sequence types (ST) and 55 pulsotype in NF, whereas the 25 ST and 43 pulsatypes were detected in AF. The MLST results were perfectly relevant to those of

PFGEs (Figure. 10,11). Three datasets obtained from 1 and 2 datasets from NF and AF, respectively, that showed the identical clone types between pigs and pork which could not be observed in the other 17 datasets. The dendrograms incorporated with antibiograms, resistant gene profiles and replicon types are presented in Figure 12,13. Interestingly, these strains did not present ESBLP trait and aminoglycoside resistance by genetic and phenotypic characterizations. On the other hand, the other datasets did not show cross-similarity of strain type between pig and pork samples and all ESBL and aminoglycoside resistant strains restricted only in pig samples. The synopsis of clonal relation of *E. coli* among each period of production and pork in slaughter house is presented in Figure 10,11. ST10 were the most common type to both groups; NF farm types (57/150) and AF farm types (40/150), while it was absent in pork. The ST604, ST877, ST1209 and ST2798 showed ESBLP characters. The clone type from pork samples in NF were found apart from farming periods except only ST638 that found in finishing and pork was non-ESBL and non-aminoglycoside resistance. On one hand, the strains from AF presenting ESBL and/or aminoglycoside resistance were highly distributed from nursery to finishing but there was no homologous clone linked from pigs to pork. However, the strains ST72, ST302 and ST402 performed ESBL and/or aminoglycoside resistance were found only in pork with no linkage between pork and farming period.

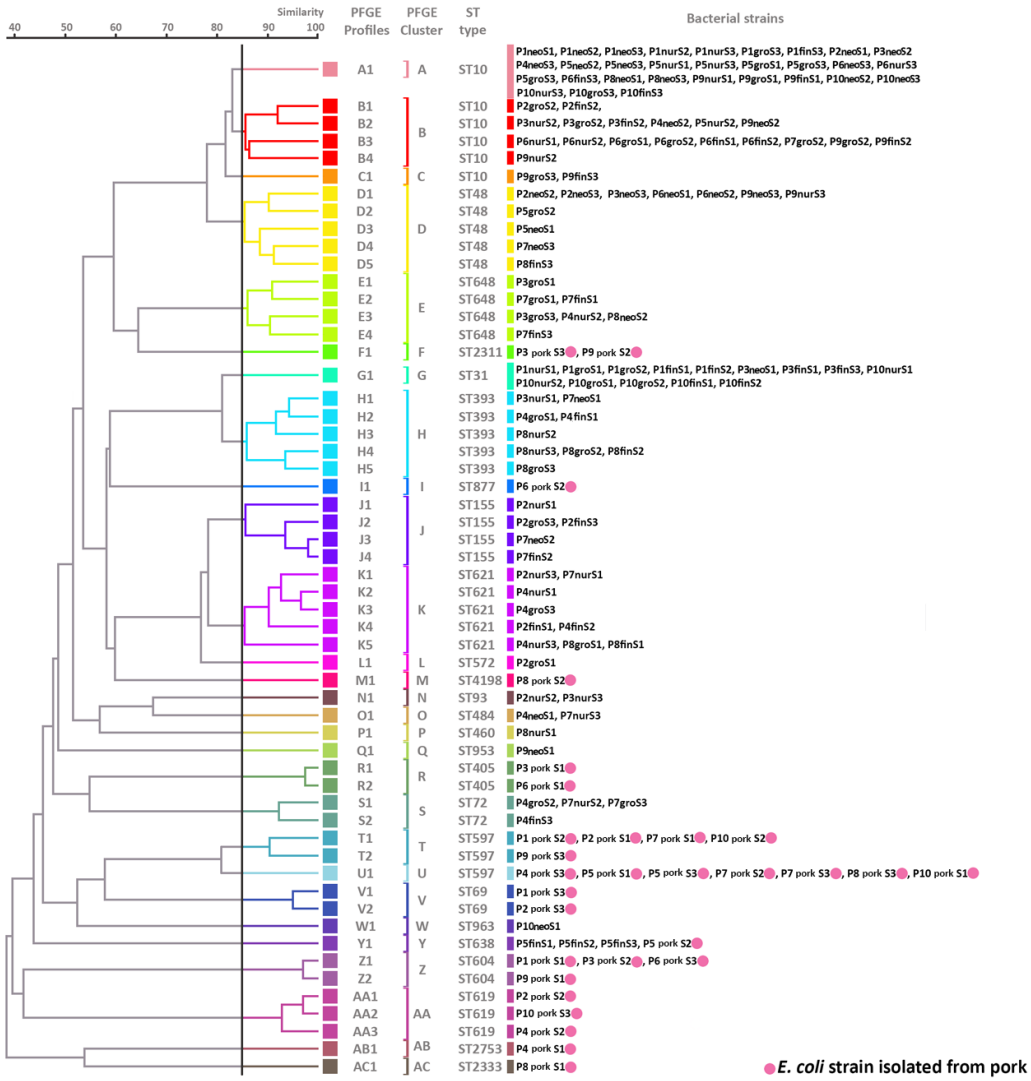


Figure 10 Molecular genetic relatedness of overall *E. coli* strains analyzed by pulsed field gel electrophoresis (PFGE) and multilocus sequence typing (MLST) in NF farm type (A). Pink round spot indicates the clones that were found in pork.

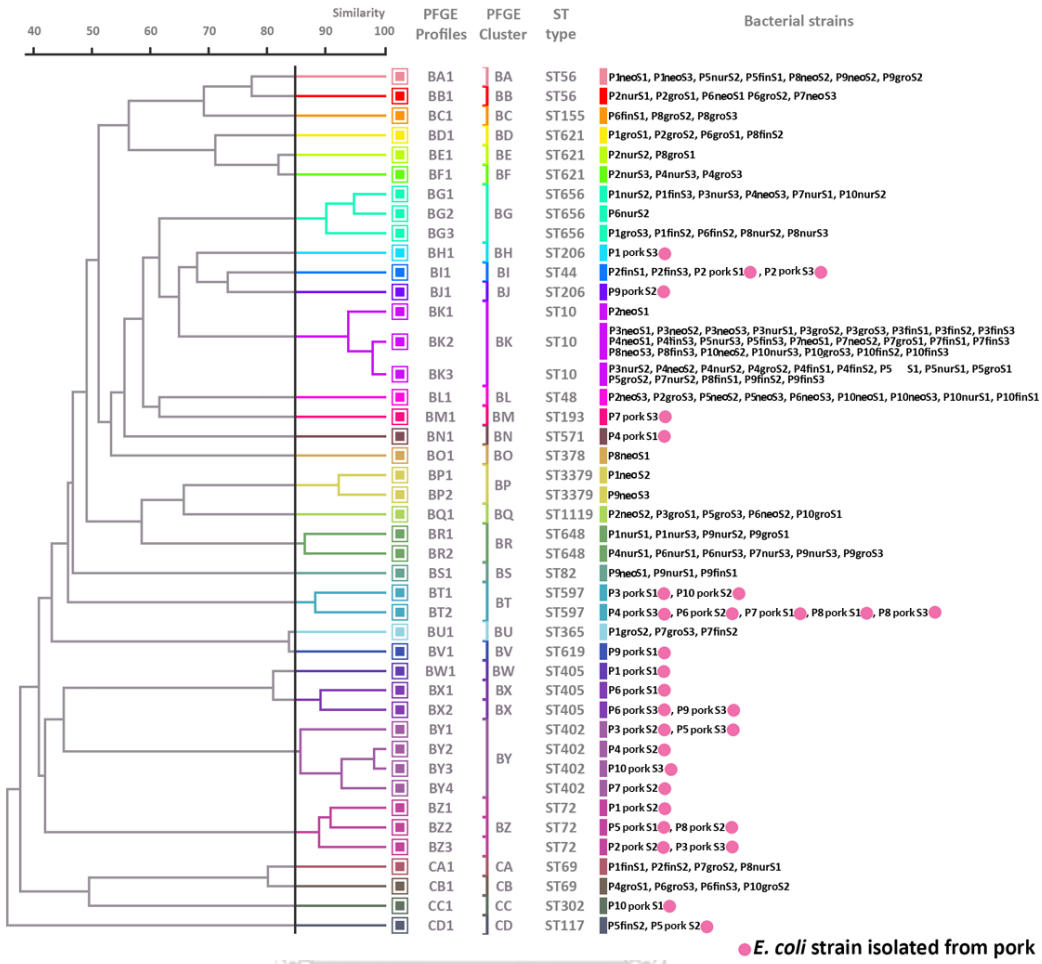


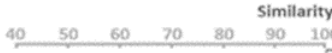
Figure 11 Molecular genetic relatedness of overall *E. coli* strains analyzed by pulsed field gel electrophoresis (PFGE) and multilocus sequence typing (MLST) in AF farm type (B). Pink round spot indicates the clones that were found in pork.

Table 11 Distribution of ST data of *E. coli* isolated represented in individual pig in NF farm types

	Creeping period	Nursery period	Growing period	Finishing period	Pork
NF1 strain 1	ST10	ST31	ST31	ST31	ST604
NF1 strain 2	ST10	ST10	ST31	ST31	ST597
NF1 strain 3	ST10	ST10	ST10	ST10	ST69
NF2 strain 1	ST10	ST155	ST72	ST621	ST597
NF2 strain 2	ST48	ST93	ST10	ST10	ST619
NF2 strain 3	ST48	ST621	ST155	ST155	ST69
NF3 strain 1	ST31	ST393	ST648	ST31	ST405
NF3 strain 2	ST10	ST10	ST10	ST10	ST604
NF3 strain 3	ST48	ST93	ST648	ST31	ST2311
NF4 strain 1	ST484	ST621	ST393	ST393	ST2753
NF4 strain 2	ST10	ST648	ST72	ST621	ST619
NF4 strain 3	ST10	ST621	ST621	ST72	ST597
NF5 strain 1	ST48	ST10	ST10	ST638	ST597
NF5 strain 2	ST10	ST10	ST48	ST638	ST638
NF5 strain 3	ST10	ST10	ST10	ST638	ST597
NF6 strain 1	ST48	ST10	ST10	ST10	ST405
NF6 strain 2	ST48	ST10	ST10	ST10	ST877
NF6 strain 3	ST10	ST10	ST10	ST10	ST604
NF7 strain 1	ST393	ST621	ST648	ST648	ST597
NF7 strain 2	ST155	ST72	ST72	ST155	ST597
NF7 strain 3	ST48	ST484	ST72	ST648	ST597
NF8 strain 1	ST10	ST460	ST621	ST621	ST2333
NF8 strain 2	ST648	ST393	ST393	ST393	ST4198
NF8 strain 3	ST10	ST393	ST393	ST48	ST597
NF9 strain 1	ST953	ST10	ST10	ST10	ST604
NF9 strain 2	ST10	ST10	ST10	ST10	ST2311
NF9 strain 3	ST48	ST48	ST10	ST10	ST597
NF10 strain 1	ST963	ST31	ST31	ST31	ST597
NF10 strain 2	ST10	ST31	ST31	ST31	ST597
NF10 strain 3	ST10	ST10	ST10	ST10	ST619

Table 12 Distribution of ST data of *E. coli* isolated represented in individual pig in AF farm types

	Creeping period	Nursery period	Growing period	Finishing period	Pork
AF1 strain 1	ST56	ST648	ST621	ST69	ST405
AF1 strain 2	ST3379	ST656	ST365	ST656	ST72
AF1 strain 3	ST56	ST648	ST656	ST656	ST206
AF2 strain 1	ST10	ST56	ST56	ST44	ST44
AF2 strain 2	ST1119	ST621	ST621	ST69	ST72
AF2 strain 3	ST48	ST621	ST48	ST44	ST44
AF3 strain 1	ST10	ST10	ST1119	ST10	ST597
AF3 strain 2	ST10	ST10	ST10	ST10	ST402
AF3 strain 3	ST10	ST656	ST10	ST10	ST72
AF4 strain 1	ST10	ST648	ST69	ST10	ST571
AF4 strain 2	ST10	ST10	ST10	ST10	ST402
AF4 strain 3	ST656	ST621	ST621	ST10	ST597
AF5 strain 1	ST10	ST10	ST10	ST56	ST72
AF5 strain 2	ST48	ST56	ST10	ST117	ST117
AF5 strain 3	ST48	ST10	ST1119	ST10	ST402
AF6 strain 1	ST56	ST648	ST621	ST155	ST405
AF6 strain 2	ST1119	ST656	ST56	ST656	ST597
AF6 strain 3	ST48	ST648	ST69	ST69	ST405
AF7 strain 1	ST10	ST656	ST10	ST10	ST597
AF7 strain 2	ST10	ST10	ST69	ST365	ST402
AF7 strain 3	ST56	ST648	ST365	ST10	ST193
AF8 strain 1	ST378	ST69	ST621	ST10	ST597
AF8 strain 2	ST56	ST656	ST155	ST621	ST72
AF8 strain 3	ST10	ST656	ST155	ST10	ST597
AF9 strain 1	ST82	ST82	ST648	ST82	ST619
AF9 strain 2	ST56	ST648	ST56	ST10	ST206
AF9 strain 3	ST3379	ST648	ST648	ST10	ST405
AF10 strain 1	ST48	ST48	ST1119	ST48	ST302
AF10 strain 2	ST10	ST656	ST69	ST10	ST597
AF10 strain 3	ST48	ST10	ST10	ST10	ST402



Xba I PFGE pattern	Bacterial strains	PFGE Profile	ST	Antibiogram	Resistance genes	Plasmid replicon type
	NF Pig 5 creeping strain 1	D3	ST48	AMP-AMX-PIP-TET-NIT	*bla* TEM-*tet* (A)	FIB-Frep
	NF Pig 5 growing strain 2	D2	ST48	AMP-AMX-PIP-TET-ENR-MBR-NIT	*bla* TEM-*tet* (A)	FIB-Frep
	NF Pig 5 creeping strain 2	A1	ST10	AMP-AMX-PIP-TET	*bla* TEM-*tet* (A)	Frep
	NF Pig 5 creeping strain 3	A1	ST10	AMP-AMX-PIP	*bla* TEM	FIB-HI2
	NF Pig 5 nursery strain 1	A1	ST10	AMP-AMX-PIP-TET	*bla* TEM-*tet* (A)	FIB-Frep
	NF Pig 5 nursery strain 3	A1	ST10	AMP-AMX-PIP-TET	*bla* TEM-*tet* (A)	FIB-Frep
	NF Pig 5 growing strain 1	A1	ST10	AMP-AMX-PIP-TET	*bla* TEM-*tet* (A)	FIB-Frep
	NF Pig 5 growing strain 3	A1	ST10	AMP-AMX-PIP-TET	*bla* TEM-*tet* (A)	FIB-Frep
	NF Pig 5 nursery strain 2	B2	ST10	AMP-AMX-PIP-TET-ENR	*bla* TEM-*tet* (A)	Frep-Y
	NF Pig 5 meat strain 1	U1	ST597	AMP-AMX	*bla* TEM	Frep
	NF Pig 5 meat strain 3	U1	ST597	AMP-AMX	*bla* TEM	Frep
	NF Pig 5 finishing strain 1	Y1	ST638	AMP-AMX-PIP-TET-C	*bla* TEM-*tet* (A)-*catA*	Frep-HI2
	NF Pig 5 finishing strain 2	Y1	ST638	AMP-AMX-PIP-TET-C	*bla* TEM-*tet* (A)-*catA*	Frep-HI2
	NF Pig 5 finishing strain 3	Y1	ST638	AMP-AMX-PIP-TET-C	*bla* TEM-*tet* (A)-*catA*	Frep-HI2
	NF Pig 5 meat strain 1	Y1	ST638	AMP-AMX-PIP-TET-C	*bla* TEM-*tet* (A)-*catA*	Frep-HI2

Figure 12 Datasets of genetic relatedness, antibiogram, resistant gene profiles and replicon profiles among *E. coli* that found clone type similarity between pigs and pork. from individual pig number 5 from NF farm.

Xba I PFGE pattern	Bacterial strains	PFGE Profile	ST	Antibiogram	Resistance genes	Plasmid replicon type
	AF Pig 2 creeping strain 1	BK1	ST10	AMP-AMX-PIP-SXT-C	*bla* TEM-*sul1-sul2-dfrA10-dfrA12-catA-catB*	FIB-Frep
	AF Pig 2 finishing strain 1	BI1	ST44	AMP-AMX-PIP-TET-ENR	*bla* TEM-*tet* (A)-*tet* (B)	FIB-FIC-Frep
	AF Pig 2 finishing strain 3	BI1	ST44	AMP-AMX-PIP-TET-ENR	*bla* TEM-*tet* (A)-*tet* (B)	FIB-FIC-Frep
	AF Pig 2 meat strain 1	BI1	ST44	AMP-AMX-PIP-TET-ENR	*bla* TEM-*tet* (A)-*tet* (B)	FIB-FIC-Frep
	AF Pig 2 meat strain 3	BI1	ST44	AMP-AMX-PIP-TET-ENR	blaTEM-*tet* (A)-*tet* (B)	FIB-FIC-Frep
	AF Pig 2 creeping strain 2	BO1	ST1119	AMP-AMX-PIPTET-SXT	blaTEM-*sul1-sul2-dfrA1-dfrA10*	FIB-HI-2
	AF Pig 2 creeping strain 3	BL1	ST48	AMP-AMX-PIP	*bla* TEM	FIB-Frep
	AF Pig 2 growing strain 3	BL1	ST48	AMP-AMX-PIP-CEX-CPD-CPR-XNL-TET-C	*bla* TEM-*bla* CTX-M-1-*tet* (A)-*tet* (B)-*catA -cmlA*	FIA-HI-1-I1-Iy-W-Y
	AF Pig 2 nursery strain 1	BB1	ST56	AMP-AMX-PIP-TET-SXT-C-NIT	*bla* TEM-*tet* (A)-*tet* (B)-*sul1-sul2-dfrA10-dfrA12-catA-catB*	FIA-FIB
	AF Pig 2 growing strain 1	BB1	ST56	AMP-AMX-PIP-TET-SXT-C-NIT	*bla* TEM-*tet* (A)-*tet* (B)-*sul1-sul2-dfrA10-dfrA12-catA-catB*	FIA-FIB
	AF Pig 2 meat strain 2	BZ3	ST72	AMP-AMX-PIP-GEN-SXT	*bla* TEM-*aadA1-aadA2-aadB-sul1-sul2-sul3-dfrA1-dfrA10*	FIA-FIB
	AF Pig 2 nursery strain 2	BE1	ST621	AMP-AMX-PIP-CEX-CPD-CPR-GEN-TOB-TET-ENR-MAR-SXT-NIT	*bla* TEM-*bla* CTX-M-1-*aadA1-aadA2-aadB-tet(A)-tet(B)-sul1-dfrA1-dfrA12*	FIA-FIC-I1-Iy-N-Y
	AF Pig 2 nursery strain 3	BF1	ST621	AMP-AMX-PIP-CEX-CPD-CPR-XNL-GEN-TOB-TET-C-NIT	*bla* TEM-*bla* CTX-M-1-*aadA1-aadA2-aadB-tet(A)-tet(B)-sul1-dfrA1-catA-catB*	FIA-FIC-HI-1-I1-Iy-N-Y
	AF Pig 2 growing strain 2	BD1	ST621	AMP-AMX-PIP-CEX-CPD-CPR-XNL-TET-ENR-MBR-SXT	*bla* TEM-*bla* CTX-M-1-*aadA1-aadA2-aadB-tet(A)-tet(B)-sul1-dfrA12*	Frep-HI-1-HI-2-I1-Iy
	AF Pig 2 finishing strain 2	CB1	ST69	AMP-AMX-PIP-CEX-CPD-CPR-XNL-TET-ENR-MBR	*bla* TEM-*bla* CTX-M-1-*aadA1-aadA2-aadB-tet(A)-tet(B)*	FIB-HI-2-I1-Iy

Figure 13 Datasets of genetic relatedness, antibiogram, resistant gene profiles and replicon profiles among *E. coli* that found clone type similarity between pigs and pork from individual pig number 2 from AF farm.

Xba I PFGE pattern	Bacterial strains	PFGE Profile	ST	Antibiogram	Resistance genes	Plasmid replicon type
	AF Pig 5 creeping strain 1	BK3	ST10	AMP-AMX-PIP-TET-C-NIT	*bla* TEM-*tet* (A)-*catA-catB*	FIB-Frep-Y
	AF Pig 5 nursery strain 1	BK3	ST10	AMP-AMX-PIP-TET-C-NIT	*bla* TEM-*tet* (A)-*catA-catB*	FIB-Frep-Y
	AF Pig 5 growing strain 1	BK3	ST10	AMP-AMX-PIP-TET-C-NIT	*bla* TEM-*tet* (A)-*catA-catB*	FIB-Frep-Y
	AF Pig 5 growing strain 2	BK3	ST10	AMP-AMX-PIP-TET-C-NIT	*bla* TEM-*tet* (A)-*catA-catB*	FIB-Frep-Y
	AF Pig 5 nursery strain 3	BK2	ST10	AMP-AMX-PIP-TET	*bla* TEM-*tet* (A)	Frep-HI-1
	AF Pig 5 finishing strain 3	BK2	ST10	AMP-AMX-PIP-TET	*bla* TEM-*tet* (A)	Frep-HI-1
	AF Pig 5 growing strain 3	BQ1	ST1119	TET	*tet* (A)	Frep
	AF Pig 5 creeping strain 2	BL1	ST48	AMP-AMX-PIP	*bla* TEM	FIB
	AF Pig 5 creeping strain 3	BL1	ST48	AMP-AMX-PIP	*bla* TEM	FIB
	AF Pig 5 nursery strain 2	BA1	ST56	AMP-AMX-PIP-GEN-TOB-NIT	*bla* TEM-*aadA1 -aadA2 -aadB*	FIA-HI-1-I1-Iy-Y
	AF Pig 5 finishing strain 1	BA1	ST56	AMP-AMX-PIP-GEN-TOB-NIT	*bla* TEM-*aadA1 -aadA2 -aadB*	FIA-HI-1-I1-Iy-Y
	AF Pig 5 meat strain 1	BZ1	ST72	AMP-AMX-PIP-GEN-TOB-TET-ENR-SXT-C-NIT	*bla* TEM-*aadA1 -aadA2 -aadB -tet* (A)-*tet* (B)-*sul1-sul2-dfrA1-dfrA10-catA*	FIA-N
	AF Pig 5 meat strain 3	BZ1	ST402	AMP-AMX-PIP-CEX-CPD-CPR-XNL-TET-C-NIT	*bla* TEM-*bla* CTX-M-1-*aadA1 -aadA2 -aadB -tet* (A)-*tet* (B)-*catA -catB*	FIA-FIC-HI-1-I1-Iy-N-W
	AF Pig 5 finishing strain 2	CD1	ST117	AMP-AMX-PIP-TET-SXT-C-NIT	*bla* TEM-*tet* (A)-*tet* (B)-*sul1-sul2-catA-cmlA*	FIA-FIC-Frep
	AF Pig 5 meat strain 2	CD1	ST117	AMP-AMX-PIP-TET-SXT-C-NIT	*bla* TEM-*tet* (A)-*tet* (B)-*sul1-sul2-catA-cmlA*	FIA-FIC-Frep

Figure 14 Datasets of genetic relatedness, antibiogram, resistant gene profiles and replicon profiles among *E. coli* that found clone type similarity between pigs and pork individual pig number 5 from AF farm.

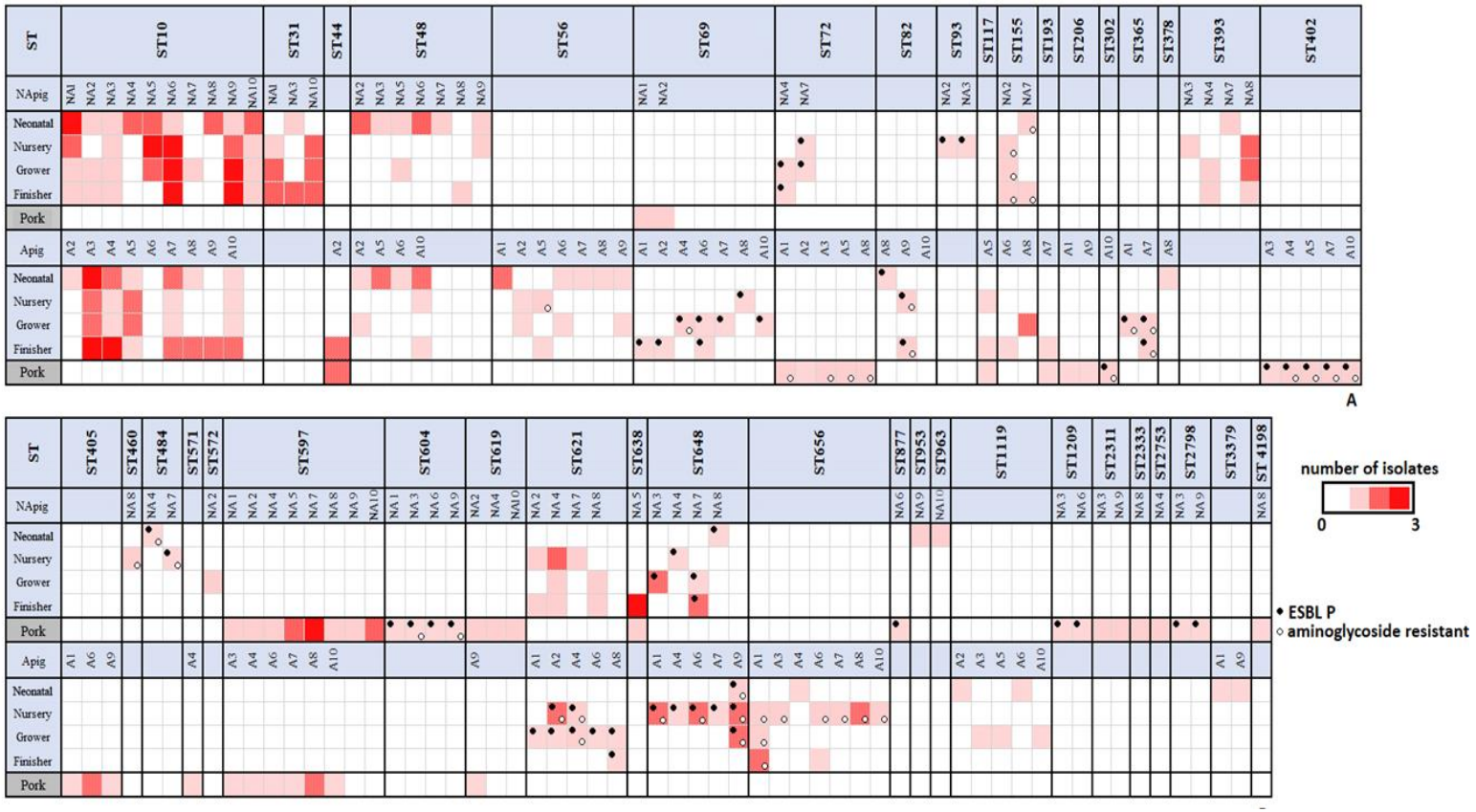


Figure 15 Longitudinal monitoring of the frequency of ST and distribution of ESBLP and aminoglycoside resistance among carriage *E. coli*. Black circle indicates the detection of ESBLP *E. coli*.

Part 3 The evaluation of flavomycin to reduce resistance characteristics and plasmid transfer. *in vitro* and *in vivo*

3.1 Reduction of plasmid transferability in invitro test

For $bla_{CTX\text{-}M\text{-}1}$group, the number of transconjugants colonies in experimental groups I were significantly less than control group and the numbers of transconjugants colonies in experimental group II were also significantly less than control group. Controversially, there was not significant difference between numbers of transconjugants colonies in group I and group II (Figure 16). For $bla_{CTX\text{-}M\text{-}9}$group, the number of transconjugants colonies in experiential groups I were significantly less than control group and the numbers of transconjugants colonies in experimental group II were also significantly less than control group. Controversially, there was not significant difference between numbers of transconjugants colonies in group I and group II (Figure 16).The transconjugation efficiency of $bla_{CTX\text{-}M\text{-}1}$ were $3.8x10^{-4}$, $3.4x10^{-5}$ and $5.6x10^{-5}$ in control group, experimental group I and experimental group II, respectively (Table13,14).

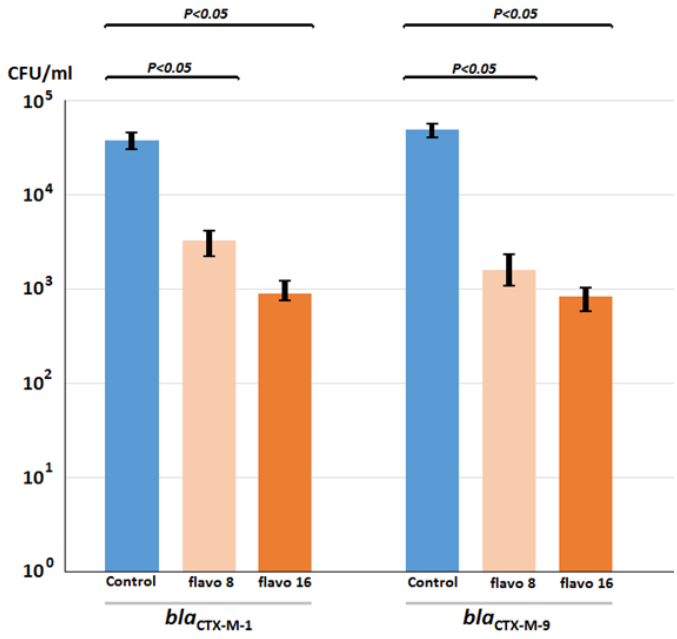


Figure 16 The numbers of transconjugants colonies in control and flavomycin treatment group. (The comparison of numbers of colonies determine by Wilcoxon Sign ranked test)

Table 13 The numbers of transconjugants colonies forming unit/ml in control and flavomycin treatment group

No	Strains Name	Resistance gene	Plasmid replicon types	Repeated time	CFU/ml		
					Control	Experimental group I (Flavomycin 8 μg/ml)	Experimental group II (Flavomycin 16 μg/ml)
1	*E. coli* PCU13	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	1	$6.6x10^4$	$4.5x10^3$	$9.8x10^2$
2	*E. coli* PCU13	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	2	$5.5x10^4$	$4.3x10^3$	$8.0x10^2$
3	*E. coli* PCU13	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	3	$5.7x10^4$	$4.4x10^3$	$7.5x10^2$
4	*E. coli* PCU14	$bla_{CTX\text{-}M\text{-}1}$group	single Inc HI2	1	$5.5x10^4$	$4.5x10^3$	$7.2x10^2$
5	*E. coli* PCU14	$bla_{CTX\text{-}M\text{-}1}$group	single Inc HI2	2	$6.6x10^4$	$4.3x10^3$	$7.8x10^2$
6	*E. coli* PCU14	$bla_{CTX\text{-}M\text{-}1}$group	single Inc HI2	3	$5.5x10^4$	$4.4x10^3$	$7.6x10^2$
7	*E. coli* PCU15	$bla_{CTX\text{-}M\text{-}1}$group	single Inc HI2	1	$5.7x10^4$	$3.9x10^3$	$8.0x10^2$
8	*E. coli* PCU15	$bla_{CTX\text{-}M\text{-}1}$group	single Inc HI2	2	$5.5x10^4$	$5.0x10^3$	$7.5x10^2$
9	*E. coli* PCU15	$bla_{CTX\text{-}M\text{-}1}$group	single Inc HI2	3	$5.3x10^4$	$5.2x10^3$	$7.2x10^2$
10	*E. coli* PCU16	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	1	$5.4x10^4$	$3.9x10^3$	$7.8x10^2$
11	*E. coli* PCU16	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	2	$6.6x10^4$	$5.0x10^3$	$8.9x10^2$
12	*E. coli* PCU16	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	3	$5.5x10^4$	$5.2x10^3$	$8.0x10^2$
13	*E. coli* PCU17	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	1	$5.7x10^4$	$5.7x10^3$	$9.0x10^2$
14	*E. coli* PCU17	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	2	$5.5x10^4$	$5.8x10^3$	$8.0x10^2$
15	*E. coli* PCU17	$bla_{CTX\text{-}M\text{-}1}$group	single IncHI2	3	$6.6x10^4$	$5.9x10^3$	$9.8x10^2$

16	*E. coli* PCU18	*bla* $_{\text{CTX-M-}1}$group	single IncHI2	1	5.5×10^{4}	5.0×10^{4}	9.6×10^{2}
17	*E. coli* PCU18	*bla* $_{\text{CTX-M-}1}$group	single IncHI2	2	5.7×10^{4}	5.3×10^{3}	9.8×10^{2}
18	*E. coli* PCU18	*bla* $_{\text{CTX-M-}1}$group	single IncHI2	3	5.5×10^{4}	5.7×10^{3}	9.4×10^{2}
19	*E. coli* PCU19	*bla*$_{\text{CTX-M-}1}$group	single Inc HI2	1	5.3×10^{4}	5.8×10^{3}	9.2×10^{2}
20	*E. coli* PCU19	*bla*$_{\text{CTX-M-}1}$group	single Inc HI2	2	5.4×10^{4}	5.2×10^{3}	8.0×10^{2}
21	*E. coli* PCU19	*bla*$_{\text{CTX-M-}1}$group	single Inc HI2	3	5.7×10^{4}	3.9×10^{3}	7.8×10^{2}
22	*E. coli* PCU20	*bla*$_{\text{CTX-M-}1}$group	single Inc HI2	1	5.5×10^{4}	5.0×10^{3}	6.4×10^{2}
23	*E. coli* PCU20	*bla*$_{\text{CTX-M-}1}$group	single Inc HI2	2	5.3×10^{4}	5.2×10^{3}	9.8×10^{2}
24	*E. coli* PCU20	*bla*$_{\text{CTX-M-}1}$group	single Inc HI2	3	5.4×10^{4}	5.7×10^{3}	9.6×10^{2}
25	*E. coli* PCU21	*bla*$_{\text{CTX-M-}1}$group	single IncHI2	1	6.6×10^{4}	5.8×10^{3}	9.4×10^{2}
26	*E. coli* PCU21	*bla*$_{\text{CTX-M-}1}$group	single IncHI2	2	5.5×10^{4}	5.9×10^{3}	9.2×10^{2}
27	*E. coli* PCU21	*bla*$_{\text{CTX-M-}1}$group	single IncHI2	3	5.3×10^{4}	6.3×10^{3}	8.0×10^{2}
28	*E. coli* PCU22	*bla*$_{\text{CTX-M-}1}$group	single IncHI2	1	5.4×10^{4}	6.0×10^{3}	9.0×10^{2}
29	*E. coli* PCU22	*bla*$_{\text{CTX-M-}1}$group	single IncHI2	2	6.6×10^{4}	5.7×10^{3}	8.0×10^{2}
30	*E. coli* PCU22	*bla*$_{\text{CTX-M-}1}$group	single IncHI2	3	5.4×10^{4}	5.4×10^{3}	6.0×10^{2}
Means of cfu/ml					5.7×10^{4}	5.1×10^{3}	8.4×10^{2}
31	*E. coli* PCU23	*bla*$_{\text{CTX-M-}9}$group	single IncHI2	1	7.5×10^{4}	3.5×10^{4}	6.0×10^{2}
32	*E. coli* PCU23	*bla*$_{\text{CTX-M-}9}$group	single IncHI2	2	7.7×10^{4}	4.0×10^{4}	8.0×10^{2}
33	*E. coli* PCU23	*bla*$_{\text{CTX-M-}9}$group	single IncHI2	3	7.8×10^{4}	2.8×10^{3}	9.8×10^{2}
34	*E. coli* PCU24	*bla*$_{\text{CTX-M-}9}$group	single Inc HI2	1	$8.\times10^{4}0$	2.7×10^{3}	9.6×10^{2}

35	*E. coli* PCU24	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	2	$6.5x10^4$	$3.0x10^4$	$9.4x10^2$
36	*E. coli* PCU24	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	3	$6.6x10^4$	$4.0x10^4$	$8.0x10^2$
37	*E. coli* PCU25	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	1	$7.7x10^4$	$2.0x10^4$	$9.8x10^2$
38	*E. coli* PCU25	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	2	$7.8x10^4$	$2.8x10^3$	$9.6x10^2$
39	*E. coli* PCU25	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	3	$8.0x10^4$	$6.5x10^3$	$9.4x10^2$
40	*E. coli* PCU26	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	1	$6.5x10^4$	$6.0x10^3$	$8.0x10^2$
41	*E. coli* PCU26	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	2	$6.8x10^4$	$3.6x10^3$	$8.6x10^2$
42	*E. coli* PCU26	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	3	$8.0x10^40$	$4.0x10^3$	$9.6x10^2$
43	*E. coli* PCU27	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	1	$6.5x10^4$	$3.0x10^3$	$9.4x10^2$
44	*E. coli* PCU27	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	2	$6.6x10^4$	$4.0x10^3$	$8.0x10^2$
45	*E. coli* PCU27	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	3	$7.7x10^4$	$2.6x10^3$	$8.6x10^2$
46	*E. coli* PCU28	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	1	$7.8x10^4$	$2.8x10^3$	$9.8x10^2$
47	*E. coli* PCU28	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	2	$8.0x10^40$	$3.0x10^3$	$9.6x10^2$
48	*E. coli* PCU28	$bla_{CTX\text{-}M\text{-}9}$group	single IncHI2	3	$6.5x10^4$	$2.8x10^3$	$9.4x10^2$
49	*E. coli* PCU29	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	1	$5.8x10^4$	$3.0x10^3$	$9.2x10^2$
50	*E. coli* PCU29	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	2	$5.9x10^4$	$2.0x10^3$	$8.0x10^2$
51	*E. coli* PCU29	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	3	$6.5x10^4$	$2.8x10^3$	$7.8x10^2$
52	*E. coli* PCU30	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	1	$8.7x10^4$	$2.6x10^3$	$8.0x10^3$
53	*E. coli* PCU30	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	2	$8.8x10^4$	$2.8x10^3$	$8.0x10^2$
54	*E. coli* PCU30	$bla_{CTX\text{-}M\text{-}9}$group	single Inc HI2	3	$8.6x10^4$	$3.6x10^3$	$7.5x10^2$

55	*E. coli* PCU31	$bla_{CTX-M-9}$group	single IncHI2	1	$8.4x10^4$	$4.0x10^3$	$7.2x10^2$
56	*E. coli* PCU31	$bla_{CTX-M-9}$group	single IncHI2	2	$8.6x10^4$	$3.0x10^3$	$7.8x10^2$
57	*E. coli* PCU31	$bla_{CTX-M-9}$group	single IncHI2	3	$8.4x10^4$	$4.0x10^3$	$9.6x10^2$
58	*E. coli* PCU32	$bla_{CTX-M-9}$group	single IncHI2	1	$4.3x10^4$	$2.0x10^3$	$9.4x10^2$
59	*E. coli* PCU32	$bla_{CTX-M-9}$group	single IncHI2	2	$5.4x10^4$	$2.8x10^3$	$9.2x10^2$
60	*E. coli* PCU32	$bla_{CTX-M-9}$group	single IncHI2	3	$6.8x10^4$	$2.6x10^3$	$8.0x10^2$
Means of cfu/ml					$7.2x10^4$	$3.3x10^3$	$8.7x10^2$

Table 14 Transconjugants efficiency of $bla_{CTX-M-1}$ group and $bla_{CTX-M-9}$ group in control group, experimental group I and experimental group II and control group.

resistance genes	Transconjugation efficiency		
	Control group	experimental group I (Flavomycin 8 μg/ml)	experimental group II (Flavomycin 16 μg/ml)
$bla_{CTX-M-1}$group	$3.8x10^4$±0.09	$3.4x10^{-5}$±0.12	$5.6x10^{-5}$±0.08
$bla_{CTX-M-9}$group	$4.8x10^4$±0.08	$2.2x10^{-5}$±0.21	$1.2x10^{-5}$±0.15

3.2 in vivo reduction of plasmid transferability test

3.2.1 Resistant phenotypic characterizations

In FF, MDR rate were higher (63.3%) and NF (64.7%). The most common antibiogram, AMP-AMX-PIP-TET, was detected in 12.0% . Overall, antimicrobial resistant phenotypes are described in Fig 15. For β-lactams groups and tetracycline, all isolates were highly resisted in all period of observations in FF types while *E. coli* resisted to amoxicillin-clavulanate were found only from AF in creeping (3.3%). The *E. coli* producing ESBL and resisted to all cephalosporin group were 20.0 to 26.7% in nursery period which were significant less than AF in the same period. In the growing period, rate of cephalosporin resistance ranged from 23.3 to 30.0% in growing period which were significant less than AF in the same period. The rates were reduced to 16.7-23.3% in finishing periods. Regarding aminoglycosides, amikacin resistance was not found in all

pigs, while gentamicin and tobramycin resistance were obviously raised in nursery and growing periods in the same rate as in AF (GEN and TOB: 60.0-66.7%) and declined in finishing period (GEN and TOB: 33.3%). The other resistance rates were not difference to AF (Figure 17).

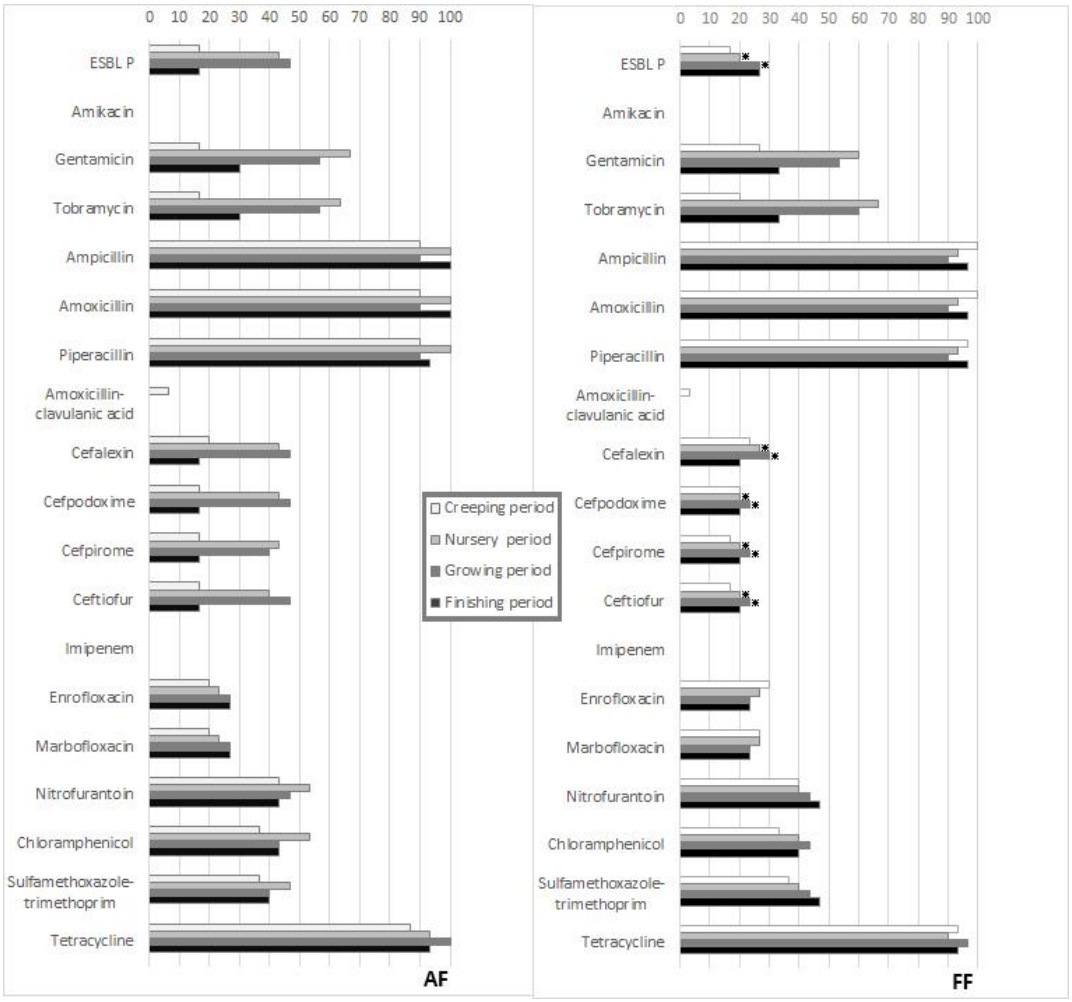


Figure 17 Eighteen antimicrobials resistance rates of and ESBLP *E. coli* which were individually collected from pigs in each growing period and meat period at abattoir in AF farm type and FF farm type. [* significant difference (p<0.05) in each growing period between farm performed by Chi-square test]

3.2.2 Genotypic resistant characterization

The common resistance genes were also bla_{TEM} and *tet*(A) genes. The genes resisted to sulfonamide; *sul1*, *sul2*, *sul3*, to trimethoprim; *drfA1*, *drfA10*, *drfA12* and to chloramphenicol *catA*, *catB* and *cmlA,* were presented in moderate levels without significant difference with AF and the observed periods (6.7-40.3%). Aminoglycoside resistant genes; *aadA1, aadA2* and *aadB* were raised to 30.0-60.0% in nursery and growing periods and decrease in finishing period (6.7-20.0%) and no significant difference with AF were observed. The significant difference of $bla_{CTX\text{-}M\text{-}1}$ group genes was found between FF and AF in nursery period (16.7%) and growing period (20.0%). While, the detection of $bla_{CTX\text{-}M2}$group, $bla_{CTX\text{-}M8}$group, $bla_{CTX\text{-}M9}$group, $bla_{CTX\text{-}M25/26}$group genes were not different between the both farms (Figure 18).

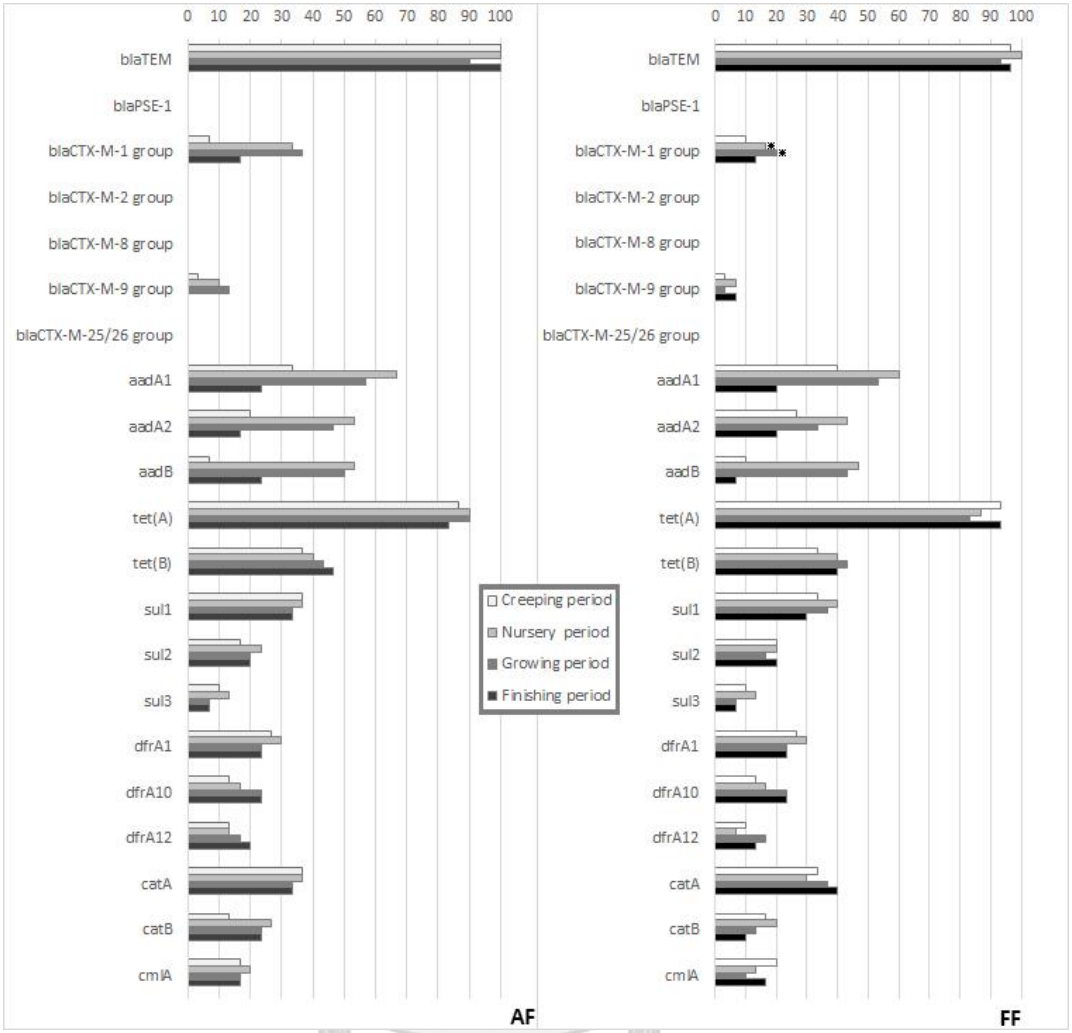


Figure 18 Twenty antimicrobial resistance gene detecting rates which were individually collected from pigs in each growing period and meat period at abattoir in AF farm type and FF farm type. [* significant difference (p<0.05) in each growing period between farm performed by Chi-square test]

3.2.3 Plasmid replicon type detection

The prevalence of 18 replicon types detected from *E. coli* are presenting in Figure 17. Plasmid replicons IncX, IncT, IncP, IncL/M, IncK, IncFIIAs, IncA/C and IncB/O were not detected in FF as in AF. The IncFrep and IncFIB replicons were commonly found FF (53.3-60.0%) While, the frequency of IncHI-2 (10.0-16.7%) and IncI1-Iγ replicons (16.7%) during nursery to growing were significantly lower in FF when compared with AF(Figure 19).

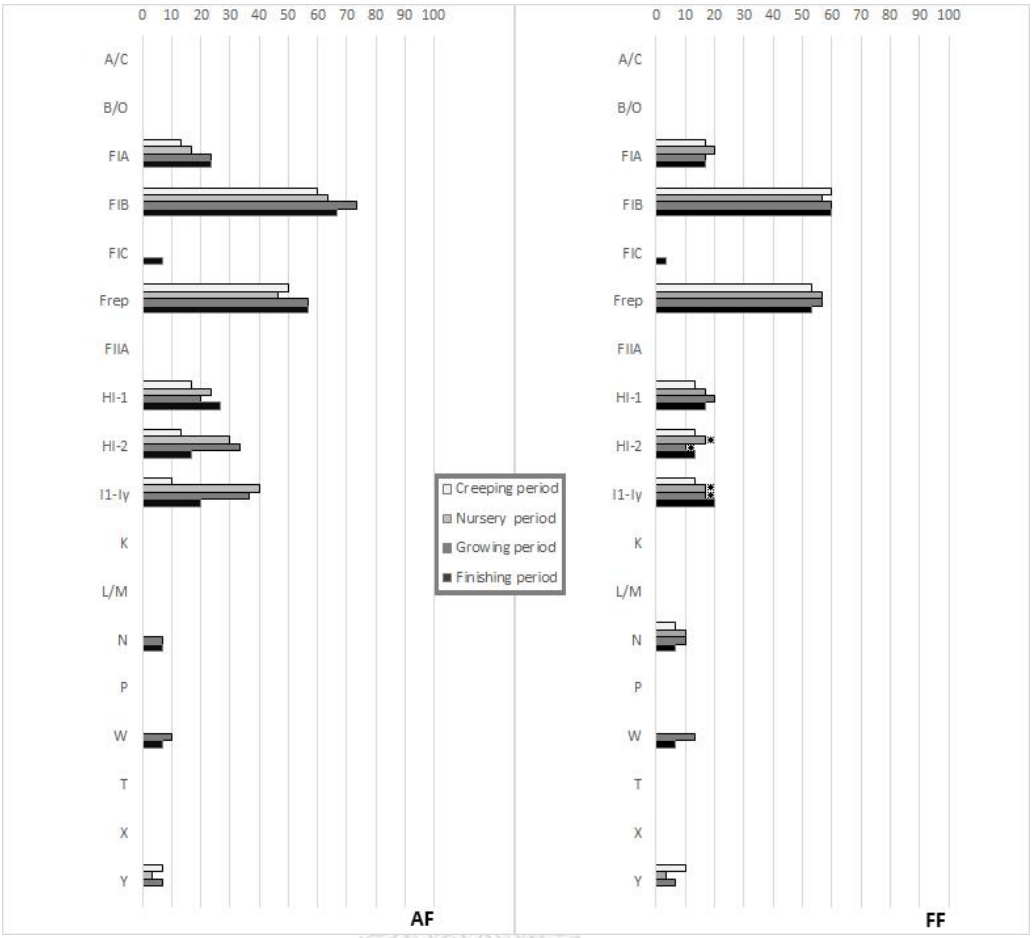


Figure 19 Eighteen plasmid replicon types detecting rates which were individually collected from pigs in each growing period and meat period at abattoir in AF farm type and FF farm type. [* significant difference (p<0.05) in each growing period between farm performed by Chi-square test.

CHAPTER VI: Discussions

Part 1. Distribution of resistance characteristics of *E. coli* isolated from pigs in different antibiotic using status in Thailand

1.1 Background

Antimicrobial are commonly used in the Thai pig industry to control endemic bacterial diseases in intensive farms. Many large intensive farms in Thailand use antimicrobials prophylactically in specific age groups of pigs, in anticipation of regular bacterial disease outbreaks, whilst some only use targeted antimicrobials for treatment of individual sick pigs. This study aimed to investigate and compare antimicrobial resistance rates in commensal *E. coli* from fattening pigs on farms that had different patterns of antimicrobial usage: farms routinely using prophylactic antimicrobials; farms only using antimicrobials for specific therapy; and farms not using antimicrobials at all. Similarly, sized and managed farms in the first two categories were included in this study. The two farms that only administered therapeutic antimicrobials to sick pigs were somewhat unusual in that they only used injectable enrofloxacin or gentamicin, and this choice might limit development of resistance compared to the more typical use of a broader range of targeted antimicrobials on many farms. Unfortunately, it was not possible to match the husbandry and conditions in the third category of farm with those in the first two categories, as the only farms that could be identified as not using antimicrobials at all were small village-based piggeries where they had neither access to nor the financial capacity to purchase antimicrobials. It is possible that other issues associated with factors such as stocking rates, nutrition or genetics may have had an influence on the findings on these farms.

1.2 Rates of phenotypic resistance

The rates of phenotypic resistance amongst the commensal *E. coli* from all three categories of farms for piperacillin, amoxicillin, ampicillin and tetracycline were similar and very high. This indicates that there is a widespread and almost universal set of resistances

to these drugs in *E. coli* from fattening pigs in Thailand, whether or not they have been exposed to antimicrobials in the past. This is disturbing since it implies that this resistance is not reversible, even in the absence of antimicrobial selection pressure. The resistance phenotypes found were associated with the common occurrence of *tet*(A) and bla_{TEM}, genes that frequently have been reported in porcine commensal *E. coli* in Southeast Asia (Changkaew et al., 2015; Khamsarn et al., 2016; Lay et al., 2012; Nhung et al., 2016; Trongjit et al., 2016). Our study suggests the existence of resistant clones that are widespread and persist in the environment. According to the fitness-cost model *tet*(A) and bla_{TEM} might be maintained in *E. coli* in animals and the environment without needing selective pressure from the use of tetracyclines and β-lactams, respectively (Enne et al., 2005; Kirchner et al., 2014). As a consequence, resistance to β-lactams and tetracycline are not a useful proxy to reflect the overall trends in antimicrobial resistance in surveillance programs. In relation to the other antimicrobials tested, no resistance was found to amikacin and imipenem, and resistance to amoxicillin/clavulanic and cefpirome was found only in the PA farms, and only in a few isolates. On the other hand, high rates of resistance to all the other antimicrobials tested were commonly found in isolates from PA farms, but were less commonly found in isolates from the other farm categories; furthermore, the resistance rates for enrofloxacin, chloramphenicol, sulfamethozaxole-trimethroprim and nitrofurantoin were substantially higher than previously reported in the pig industry in Thailand (Lay et al., 2012; Prapasarakul et al., 2010; Trongjit et al., 2016). Importantly, apart from tiamulin-fumarate and amoxicillin the other antimicrobials had not been used in the pigs in the PA farms, including chloramphenicol and nitrofurantoin that have been banded from use in Thai livestock since 1999 (MoAC, 1999). From this it seems that the regular prophylactic use of tiamulin-fumarate and/or amoxicillin has selected for commensal *E. coli* that are resistance to these two antimicrobials in the PA farms, but also for co-resistance in these isolates to the other antimicrobials that were tested. For example, co-selection for resistance to these antimicrobials may have occurred with resistance to amoxicillin on the same mobile genetic elements or adjacent genes (Johnson et al., 2016). Resistance to tiamulin mainly derives from chromosomal mutations

in the 23S rRNA and *rplC* genes that result in reduced drug binding, and consequently may lead to cross-resistance to other antimicrobials that target the bacterial ribosome (van Duijkeren et al., 2014). In addition, genes such as *cfr* that encode rRNA methyltransferase which methylates position A2503 on the 23S rRNA and confers resistance to chloramphenicol, florfenicol, clindamycin, pleuromutilins (tiamulin), oxazolidinones, and streptogramin can be located on plasmids or transposons which are transferable between bacterial species (Long et al., 2006). Their transfer potentially also could co-select for resistance genes located on these elements. An investigation into the genetic basis of resistance to tiamulin in these isolates, and potential cross-resistance to other drugs, will be the subject of a future study.

1.3 Genotypic resistance

The genes *aadA1*, *aadA2, aadB, sul1, sul2, sul3, dfrA12, catA, catB* and *cmlA,* as well as the integron gene *intI* were detected in isolates from the PA group at a significantly higher rate than in isolates from the TA and NA groups. This group of resistance genes usually is located on class I integrons (Kadlec and Schwarz, 2008; Povilonis et al., 2010; Szmolka and Nagy, 2013), and especially *dfrA1*, *dfrA12, aadA1* and *aadA2* are commonly found on class I integron gene cassettes in isolates from Southeast Asia (Lay et al., 2012; Trongjit et al., 2016). This group of resistance genes might be class 1 integron-borne resistance determinants that were co-selected with integron I and have become widely spread amongst isolates from PA farms under ongoing antimicrobial selection pressure.

1.4 ESBLP *E. coli*

Generally, the occurrence of ESBL-producing bacteria in pigs varies depending on micro-environmental factors and the growth phase of the pigs (Schmithausen et al., 2015). Commensal strains of *E. coli* are considered to represent an important reservoir of ESBLs (Zheng et al., 2012).This study found a high rate of commensal ESBLP *E. coli* in pigs close to slaughter age on PA farms, and this rate was greater than in younger pigs that previously have been tested in Thailand [48.4% (Boonyasiri et al., 2014);

44.3%(Changkaew et al., 2015)]. The high frequency of ESBLP *E. coli* supports the suggestion that amoxicillin may select for these strains in pig farms and is consistent with the results of a previous study (Cavaco et al., 2008). Whilst no ESBLP *E. coli* were found in the farms that did not use antimicrobials (NA), it was unclear what resulted in ESBLP also being present in the two farms that only used therapeutic enrofloxacin or gentamicin by injection. Infrequent use of these antimicrobials for sick pigs would not be predicted to select for ESBLP *E. coli.* The strains are unlikely to have been acquired from other PA farms, as they would have been expected to be resistant to a greater range of antimicrobials than was found. Over 50% of the *E. coli* from the PA farms showed ESBL properties, commonly possessing $bla_{\text{CTX-M-1}}$ group or $bla_{\text{CTX-M-9}}$ group genes that have a worldwide distribution in both healthy and ill humans in Southeast and East Asia.[38-40] By contrast, only a few ESBLP *E. coli* were detected in the TA farms, and none in the farms that did not use antimicrobials. Previously, ESBL phenotypes were reported in 2.4%-75% of porcine *E. coli*, but in those studies, there was lack of historical data, especially relating to antimicrobial use (Kiratisin et al., 2008; Nhung et al., 2016; Trongjit et al., 2016). This study demonstrated a relationship between the use of prophylactic in-feed antimicrobials in pig farms and a high risk of finding ESBLP *E. coli*. Moreover, all ESBLP *E. coli* in this study were resistant to enrofloxacin, which has been on the list of WHO concerns for monitoring programs (WHO, 2011). However, as indicated previously, the prevalence of ESBL related genes like $bla_{\text{TEM-1}}$ variants ($bla_{\text{TEM-25}}$), $bla_{\text{SHV-1}}$ variants ($bla_{\text{SHV-12}}$) and $bla_{\text{VEB-1}}$ should be confirmed by PCR and sequencing. Resistant strains from pigs could represent a potential risk to human health, and this requires more detailed investigation. The genes $bla_{\text{CTX-M-1}}$ and $bla_{\text{CTX-M-9}}$ that were detected with single FIB and F replicons, and that could be transfer in a conjugation assay, had the potential to undergo horizontal transfer to other strains and the associated environment (Moodley and Guardabassi, 2009; Zurfluh et al., 2014).

1.5 Plasmid replicon type detection

IncFIB and IncF plasmids are narrow host range genetic elements which have been reported worldwide in human and avian *Enterobacteriaceae,* associated with tet(A),

bla$_{TEM}$ group and *bla*$_{CTX-M}$ group genes (Johnson et al., 2007; Mshana, 2009). The IncFIB and IncF plasmid encoded plasmid addiction systems are self-beneficial for their maintenance and spread, both with and without antibiotic selection pressure. On the other hand, in our study broad-host range plasmids N and HI1 contained *bla*$_{CTX-M-1}$group and *bla*$_{CTX-M-9}$ group and these are known to have epidemiological significance in humans and livestock in Europe, Asia and the USA (Carattoli et al., 2005; Cottell et al., 2013; Jakobsen et al., 2015). Plasmid IncN was only detected in isolates from the PA group: this distribution might have from a high fitness cost of the plasmid such that it is less well maintained in an environment lacking antibiotic selective pressure (Humphrey et al., 2012) Some other potential plasmid-mediated resistance genes were not investigated in the current study, and these included *floR* and *ampC* that previously have only been detected at very low rates in studies in Southeast Asia and East Asia (Huang et al., 2012; Humphrey et al., 2012; Lee et al., 2014). Other resistance genes including plasmid-mediated quinolones resistance (PMQR) genes that have been associated with resistant strains from human patients in this area also need to be studied to see if they may have an origin in isolates from pigs.

Part 2. Longitudinal monitoring of molecular resistance genotypic of *E. coli* monitoring of from pig in the production cycle

2.1 Backgrounds

Eventhough, a number of AMR researches in pigs and pork products have been attempting to reveal on prevalence, characterization and clonal analysis using the bacterial carrier *E. coli,* but there was still no evidence verified the linkage between belief of AMR bacteria from farm to meat product (Jakobsen et al., 2011; Kaesbohrer et al., 2012; Thorsteinsdottir et al., 2010). By longitudinal monitoring, the AMR in production cycle to slaughtering could illustrate the possible clonal linkage between intestinal *E. coli* in production cycle and in pork. This study could provide the complete datasets of AMR distribution using clone typing, antimicrobial characterizations especially ESBLP and aminoglycoside resistance in carriage *E. coli* in along the process. The farm

managements contained with and without antibiotic feed additive yielded the variety rate of AMR and antibiogram but both still showed the non-relation of clone types between farms and slaughter samples. In general, the management and geographical differences closely effected to the genetic diversity and existence of antimicrobial resistance genes of porcine isolated *E. coli* (Leistner et al., 2013; Richards et al., 2006). Use of antibiotic in feed additive was also included the major impact to clone selection directly based on selective pressure (Lugsomya et al., 2017; Makita et al., 2016). The high clonal diversity of *E. coli* was undoubtedly found in individual pig to pen levels (Herrero-Fresno et al., 2017) but outstanding strains ST10 became a dominant clone and acted as a persister along pig growing periods (Ahmed et al., 2017). However, the 3 selected colonies per sample from high dilution (10^{-4}) , this criteria could enhance the recruitment of variety of *E. coli* strains in intestine and ST10 was still the major population at over 50% of observation in our study (Lautenbach et al., 2008; Lugsomya et al., 2017).

2.2 Resistant phenotypic and genotypic characterizations

The amino penicillin resistance (ampicillin and amoxicillin) and urevido-penicillin (piperacillin) conferring bla_{TEM} gene and tetracycline resistance conferring *tet*(A) gene in *E. coli* were the most common AMR in both farms and all observation periods was consistent to all cross-sectional studies in Southeast Asian (Lugsomya et al., 2017; Nhung et al., 2016). The persistent bla_{TEM} and *tet*(A) genes in *E. coli* may not cause by the selective pressure but it may imply abundance of bla_{TEM} and *tet*(A) in *Enterobacteriaceae* bacteria in the area (Assawatheptawee et al., 2017; Honda et al., 2016). Despite cephalosporins and aminoglycosides were not involved in farm management but tiamulin and ampicillin, the frequency of ESBLP *E. coli* contained $bla_{CTX\text{-}M\text{-}1\ group}$ and aminoglycosides resistant *E. coli* contained *aadA1*, *aadA2* and *aadB* were high in nursery period in only AF farm. This evidence confirmed the finding of the previous study (Lugsomya et al., 2017) that might be explained by coharboring the multi-resistance genes including *cfr* and $bla_{CTX\text{-}M}$ genes on the conjugative plasmid (Zhang et al., 2015). Thus, the significant frequency of ESBLP and aminoglycoside resistance were used as the monitoring marker in this study. From this study, tiamulin and amoxicillin in feed might

associating with multidrug resistance selection. From previous study, tiamulin resistant genes were located on the same third generation cephalosporin resistant genes which were co-resistance between tiamulin and third generation cephalosporin. Moreover, *cfr* genes which were resist to pleuromutilin also resist to chloramphenicol.

2.3 Plasmid replicon type detection

The replicons IncFrep and IncFIB were the most common spread in human and animal sources including both groups in this study. These plasmids encoding factors e.g. iron uptake, toxin enzyme and variety of resistance genes e.g. bla_{CTX-M} were mostly spread in *Enterobacteriaceae* (Kim et al., 2011; Lugsomya et al., 2017; Naseer and Sundsfjord, 2011; Rozwandowicz et al., 2018). In AF farm, the high frequency of IncI1-Iγ, IncHI2 in nursery periods were strongly relevent to detection of ESBLP and $bla_{CTX-M-1}$group gene. Previously, IncHI2 carried not only *esbl* gene but also included a variety of genes encoding sulfonamides, aminoglycosides, tetracycline and streptomycin resistance (Dierikx et al., 2010; Doublet et al., 2014). IncI1-Iγ was also the common plasmids in *E. coli* from livestock carrying $bla_{CTX-M-1}$ (Borjesson et al., 2013; Shaheen et al., 2011). Thus, the nursery pigs in from antibiotic used farm could act as the important reservoir of AMR source possibly impact to public health.

2.4 Molecular genotypic characterization

The fingerprinting analysis by MLST and PFGE were performed as tracking marker presenting the diversity of clone type making the linkage throughout the observations and they showed the consensal outcomes (Bae et al., 2014; El Garch et al., 2017; Nemoy et al., 2005). Most of clone types in pork were not detected in pigs except STs 44, 117, 155, 638 that were non-ESBPL *E. coli* and negative for bla_{CTX-M} genes. The most common ST10 carriage *E. coli* in pig was also reported in human, chicken and other animals, (Herrero-Fresno et al., 2017; Herrero-Fresno et al., 2015; Leverstein-van Hall et al., 2011b; Toval et al., 2014) whereas pork was not a source of origin in this study. Interestingly, the ST597 became the predominate clone in pork derived from both farms which previously reported as enteric pathogen in human patients (Hasman et al., 2014; Khong et al., 2016). However,

neither this clone could be observed in pig samples from any farming periods nor presence of ESBLP and aminoglycoside resistance. Thus, the source of food-poisoning might be from non-farming process. Our study found no relation of the molecular clone contained genes encoding ESBLP and aminoglycoside resistance from farm toward slaughter house as well as the evidences from cross section surviellances (Boonyasiri et al., 2014; Sunde et al., 2015; Thorsteinsdottir et al., 2010). The high distribution of multidrug resistant *E. coli* was found in all tested farms and periods of collection. The aminoglycoside resistant and ESBLP *E. coli* were predominant at nursery period in antibiotic used farm. The complete molecular characteristics and clone typing indicates that the pig production should not be accused as the origin of AMR bacterial distribution to consumers. The standard slaughtering process, meat trimming and packaging should be more investigated using harzard analysis critical control point in the further study.

Part 3 The evaluation of Flavomycin to reduce resistance characteristics and plasmid transfer. *in vitro* and *in vivo*

3.1 Reduction of plasmid transferability in vitro test

flavomycin at 8 μg/ml and 16 μg/ml could decreased transconjugation efficiency and number of transconjugants of $bla_{CTX\text{-}M\text{-}1}$ with IncHI2 positive and $bla_{CTX\text{-}M\text{-}9}$ with IncHI2 positive *E. coli* were conformed to the previous study which reported that Flavomycin at 8 μg/ml could reduce transconjugation efficiency *in vitro* horizontal gene transfer in *E. coli*. (Poole et al., 2006). The *vivo* test, the results from previous were variable (Riedl et al., 2000; van den Bogaard et al., 2002) might depend on flavomycin resistance mechanism of targeting bacteria and others in surrounding context. Species of animals which influenced to their intestinal conditions. Normally, the transconjugation efficiency and number of transconjugants depended on many factors like mating pair formation (MPF) factor especially, *traY* (Cottell et al., 2014); the one type of pilus encoding genes and mobilization families (MOB) (Smillie et al., 2010). Flavomycin inhibit the bacterial transglycosylases reaction which catalyze the bond between disaccharide pentapeptide subunits into nascent peptidoglycan during cell wall synthesis (Riedl et al., 2000).

Flavomycin also interacted with lytic transglycosylases encoded on conjugative plasmids (Hoskins et al., 1999; Mani et al., 1998) which was found in both Gram-positive and Gram-negative bacteria. These enzymes were capable of locally enlarging gaps in the peptidoglycan meshwork to allow the efficient assembly and anchoring of supramolecular transport complexes in the cell envelope. These was the positive effect to facilitate the passage of plasmid DNA through the peptidoglycan layer during conjugation (Bayer et al., 1995; Koonin and Rudd, 1994). The hindering effect of Flavomycin against plasmid transferring also conformed to the results of previous studies(Poole et al., 2006; Riedl et al., 2000; van den Bogaard et al., 2002), flavomycin usage in pig farms can reduce bacterial conjugation rate at 20-23.3% in vivo study, however the antimicrobial usage in field should concern. The flavomycin susceptibility against *E. coli* and other *Enterobacteriaceae* should be performed and co-resistance against flavomycin with other antimicrobials should be investigated.

3.2 in vivo reduction of plasmid transferability test

The cephalosporins resistance phenotypes and ESBL P *E. coli* phenotypes detected in lower rates in nursery period and growing periods when comparing with AF. However, the ESBL P *E. coli* and cephalosporins resistance were also encoded in chromosomal DNA, they were commonly found in conjugative plasmid and mobilizable plasmid in *Enterobacteriaceae* (Brolund and Sandegren, 2016; Carattoli, 2009; Day et al., 2016; Freitag et al., 2017; Schaufler et al., 2016). These data might represent potential of flavomycin which can reduce the resistance phenotypes in *in vivo* study in pigs like Corpet and colleagues which found that chlortetracycline resistant rates reduced in *Enterobacteriaceae* after using Flavomycin 5 μg/ml in water (Corpet,1984). For the resistant genes, $bla_{\text{CTX-M-1}}$ group detecting rates in FF were lower in nursery and growing period than in the same period in AF. Even though there were many scientific articles show that flavomycin could reduce the appearance of resistance genes in both Gram-positive and Gram-negative bacteria like *vanA* and bla_{TEM} (Pfaller, 2006; Poole et al., 2006; Riedl et al., 2000), no one reported the effect of Flavomycin against ESBL genes like $bla_{\text{CTX-M-1}}$ group before. The reducing rates *in vivo* might depend on the summation of reducing rate

of R plasmid conjugation (Poole et al., 2006). For replicon types of plasmid data, we found that IncHI2 and IncI1-Iγ detecting rates were lower in nursey and growing period in FF than in AF. There was first report which described antimicrobial alternative substance against plasmid replicon resistance profiles before. Overalls, the evidence of ESBLs phenotypes, third generation cephalosporins resistant rates, $bla_{CTX\text{-}M\text{-}1}$ group detecting rates and IncHI2 and IncI1-Iγ detecting rates in nursery and growing period in FF were lower than AF were represent the effect of flavomycin when use 10 ppm in feed in nursery and growing period could reduce important resistance genes like $bla_{CTX\text{-}M\text{-}1}$ group and IncHI2 and IncI1- Iγ plasmid replicon types.

CHAPTER VII: Conclusions, future recommendation and executive summary

Conclusions from Part 1: Distribution of resistance characteristics of *E. coli* isolated from pigs in different antibiotic using status in Thailand

The data from this study proved the hypothesis that there was high rate of AMR in porcine fecal *E. coli* are commonly found in Thai pig production cycle. The percentage of resistance and types of resistance were quite depended on antimicrobial usage form in farm. The AMR rate, resistant genotypic detecting rates in farm which used routine antimicrobial for prophylactic purpose were higher than farm which use antimicrobial for therapeutic propose and non-antimicrobial usage farms. The antimicrobial resistance rates were quite high even the critically important drugs in human like ^{3rd}generation cephalosporin in *E. coli* isolated common pig farms which were usage antimicrobials for prophylactic purpose in feeds. Some of resistance characteristic and genotypes were quite common in pig production cycle, it became as a common MDR *E. coli* in the study area showing β-lactam resistance and tetracycline resistance even in the farms did not use antimicrobials. The antimicrobial usage in therapeutic propose (especially by individual injected pigs) did not affect or had no association to antimicrobial resistance rates by phenotypes, genotypes and the mobile genetic elements. However, we still confirmed that use of antimicrobials mixed in feed generated the high resistance rate to aminoglycosides and third generation of cephalosporins represented ESBLP *E. coli* in fattening pigs. This is a crucial caution of antibiotic use in prophylactic purpose. A more holistic understanding should result from longitudinal surveillance at different points through the production cycle through to meat at slaughter, especially if exposure or lack of exposure to antimicrobials can be recorded.

Future recommendation from part 1: Distribution of resistance characteristics of *E. coli* isolated from pigs in different antibiotic using status in Thailand

The antimicrobial use in therapeutic propose had no association to antimicrobial resistance rate and could be still approved to use in swine practice but that in feed

additive must keep in consideration of emerging crucial MDR especially ESBL and aminoglycoside resistance in pig farms.

Conclusions from Part 2: Longitudinal monitoring of molecular resistance genotypic of *E. coli* monitoring of from pig in the production cycle

From the second hypothesis, the phenotype and genotype associated AMR of fecal *E. coli* in pigs were different in each period of observation and could be altered by routine antibiotic used (amoxycillin and tiamulin as feed additive). Our study confirmed that the antimicrobial usage in feed for prophylactic purpose was the crucial factor to rise the AMR rates in both phenotypes, genotypes and the mobile genetic elements when observed individually and longitudinally in this part. By cohort of pigs on farms, the commensal *E. coli* with AMR traits (especially resistance to β-lactam group antibiotics and to tetracycline) were still common in both farms and at all collection periods. The antimicrobial resistance rate especially the third generation cephalosporins and aminoglycosides, again increased in nursery periods and growing periods beneath antimicrobials selective pressure by feed additive. Interestingly, the resistance rates were temporal changes and decreasingly reverse in slaughtering periods. *E. coli* that were aminoglycoside resistant and ESBLP were predominant found in the nursery and grower periods and were significantly more common in antibiotic used farm than in feed additive free farm. Tiamulin and amoxicillin usage in animal feed selected the multidrug resistant *E. coli* especially in nursery and growing period even though bacterial clonal transfer between pig and pork were detected in very low rate, the antimicrobial usage should be awareness under veterinarian supervision. Pig in nursery and growing period were the risk growing period for AMR bacteria detecting especially third generation cephalosporin and aminoglycosides resistance. The farmers, veterinarians and pig relating people should be good sanitize after pig handling and bacterial contaminating from nursery pigs and growing pigs to environment should be concerned. The common molecular clonal type of *E. coli* in live pigs was ST10 that were non-ESBLP strain and could not found in meat. In contrast, ST597 was the most common clonal types of meat

in both farms. Molecular typing failed to identify a direct relationship between strains in meat and those found in the corresponding live animals during their production cycle. An investigation study in slaughtering process is helpful for AMR risk assessment in slaughterhouse.

Future recommendation from part 2: Longitudinal monitoring of molecular resistance genotypic of *E. coli* monitoring of from pig in the production cycle

Even though the high antimicrobial resistance rates in pig farms in Thailand was confirmed by both cross-sectional and longitudinal study. In nursery and growing periods were obviously demonstrated as the risk part for ESBLP *E. coli* existence. Thus, the farmers and veterinarians should pay attention to crucial AMR bacteria transmission and must deal this risky by the proper hygienic strategy. Clone ST597 could have been a cross-contaminant from the environment of the abattoir, from equipment, or from other carcasses, although the pigs from the two farms were killed in different abattoirs and so no single external source existed. The route for its appearance in pig meat in abattoirs is important. Further studies are required using hazard analysis critical control point analysis during the standard slaughtering process, meat trimming and packaging to identify the source of *E. coli* with AMR characteristics. The dominant types found in meat may have other attributes that could help to explain why they are present than other more common types found in the live animals.

Conclusions from Part 3 The evaluation of Flavomycin to reduce resistance characteristics and plasmid transfer. in vitro and in vivo

The flavomycin at 8 μg/ml concentration and 16 μg/ml concentration could confirm reduction of the conjugative rates of $bla_{CTX\text{-}M\text{-}1}$ and $bla_{CTX\text{-}M\text{-}9}$, *in vitro,* and might have a potential to control the resistant *E. coli* spreading. *In vivo* study, use of flavomycin at 10 ppm in feed could reduce antimicrobial resistance rates in both phenotypes and genotypes especially the third generation cephalosporins resistance in nursery and growing periods. The *in vivo* outcome was strongly consistent to those of *in vitro* study.

Future recommendation from part 3: The evaluation of flavomycin efficacy to reduce the resistance rates and plasmid transfer via conjugative mechanism.

This study suggested that use of flavomycin as feed additive in pig production cycle could reduce the rate of AMR *E. coli* in term of genotype and phenotypes especially the third generation cephalosporins and aminoglycosides resistance in both *in vitro* and *in vivo* studies, however flavomycin as a feed additive is still an alternative indication but is in curiosity for in other resistances via cross resistance selection. This hypothesis should be proved in detail by whole genome sequencing.

Executive summary

To date most studies have only involved cross-sectional observations taken at specific periods of production, with the studied farms having a variety of management procedures, geographical locations and a lack of availability of historical data about antimicrobial use. Our results were able to confirm by the high AMR rate in fecal carriage *E. coli* in Thai pig production cycle especially in the farm where routinely used antimicrobials (amoxycillin and tiamulin) for prophylactic indication. *E. coli* resisted to β-lactam antibiotic and tetracycline in pig farms was very common trait and ubiquitous in the study area, no matter whether antimicrobial was used or not. This finding supported our research question that the high rate of AMR in porcine fecal *E. coli* are commonly found in Thai pig production cycle at the time of investigation. The antimicrobial resistance rates were composed of that to the third generation cephalosporins and aminoglycosides which are in the priority pathogen lists of world health organization and caution in human hospitals. Thus, at nursery and grower periods in pig production became the critical periods which could distribute the MDR bacteria to environment and farmers accelerating by antimicrobial in feed additive. This phenomenal was confirmed by cross-sectional and cohort pigs in farms. All observations consisting antibiogram, resistant gene profiles and plasmid profiles were proved their association to the high rate in fecal carriage *E. coli* in pigs which were dominant in that particular period of observation and altered by routine antibiotic used. Moreover, the complete clone type characterization in fecal carriage *E. coli* from live pigs to meat was the first comprehensive investigation. The unlink clones between live pigs and meat implied only farm management but the slaughtering standard should have also pay more attention to reduce the distribution of AMR bacteria from farms to consumers. This finding can be a caution for persons associated to pig farms and preventing of AMR bacterial cross contamination from pigs should pay attention on these stages. To reduce the AMR rate in practice, flavomycin is an alternative tool on the basis of their mechanism and scientific evidence supports. Use of flavomycin at 10 ppm in feed could support that final hypothesis described its possible reduction of AMR rate in live pigs especially aminoglycoside resistant and ESBLP *E. coli* at nursery and growing

periods. This finding was consistent to the *in vitro* outcome by plasmid conjugative assay. Altogether, these studies demonstrated the high incidence of AMR by using fecal carriage *E. coli* the proxy that revealed which part of production and type of antimicrobials should be ultimately crucial issue for an efficient monitoring and hygienic strategy in relation to swine management and veterinary public health.

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APPENDIX

Preparation of media, solution and buffers

1. **Media for biochemical test**

1.1 Cary-Blair Transport medium

Disodium hydrogen phosphate	1.1 g	
Sodium thioglycolate		1.5 g
Sodium chloride	5.0 g	
Calcium chloride	0.09 g	
Agar		5.6 g
Add water to		1000 ml

Adjusted pH 8.4 ± 0.2 and sterilized at 121 °C 15 minutes

1.2 0.85 % NaCl

Sodium chloride	8.5 g	
Add water to		1000 ml

Adjusted pH 8.4 ± 0.2 and sterilized at 121 °C 15 minutes

1.3 Eosine Methylene Blue agar medium

1.4 Nutrient agar medium

Peptone	5.0 g	
Sodium chloride	5.0 g	
Peptone	1.5 g	
Yeast extract 1.500		
Agar 15.000		
Final pH (at 25°C) 7.4±0.2		
Pancreatic Digest of Gelatin	10.0 g	
Lactose	5.0 g	
Sucrose	5.0 g	
Dipotassium Phosphate	2.0 g	
Agar		13.5 g
Eosin Y		0.4 g
Methylene Blue	0.065 g	
Add water to		1000 ml

Adjusted pH 7.4 ± 0.2 and sterilized at 121 °C 15 minutes

1.5 Methyl Red- Voges Proskauer (MR-VP) broth medium

Buffered peptone	7.0 g
Dextrose	5.0 g
Dipotassium phosphate	5.0 g

	Add water to	1000 ml
	Adjusted pH 6.9 ± 0.2 and sterilized at 121 °C 15 minutes	

1.6 MR reagents

Methyl Red	0.02 g
Ethyl Alcohol	60.0 ml
Add water to	100 ml

1.7 VP reagent A

Alpha-Naphthol 5%	50.0 g
Absolute Ethanol	1000 ml

1.8 VP reagent B

Potassium Hydroxide	400.0 g
Water	1000 ml

1.9 Indole broth medium

Casein Peptone	20.0 g
Potassium Hydroxide	400.0 g
Water	1000 ml
Adjusted pH 7.0 ± 0.2 and sterilized at 121 °C 15 minutes	

1.10 Indole Kovacs reagents

p-Dimethylaminobenzaldehyde	50.0 g
Hydrochloric Acid, 37%	250 ml
Amyl Alcohol	750 ml

1.11 Simmon Citrate agar medium

Magnesium sulphate	0.2 g
Ammonium dihydrogen phosphate	1.0 g
Dipotassium phosphate	1.0 g
Sodium citrate	2.0 g
Sodium chloride	5.0 g
Bromothymol blue	0.08 g
Agar	15.0 g
Adjusted pH 6.8 ± 0.2 and sterilized at 121 °C 15 minutes	

2. Buffer for PFGE

2.1 1M Tris-HCl, pH 8.0

Tris base	121.0 g
Ultrapure water	700.0 ml
Adjust pH to 8.0 by 6 N HCl	
Add ultrapure water to be 1000 ml and sterilize by autoclaving	

2.2 10 N NaoH

NaOH 400.0 g

Add sterile ultrapure water 800.0 ml

Cool solution to room temperature

Add sterile ultrapure water to be 1000 ml

2.3 0.5 M EDTA, pH 8.0

$Na_2EDTA.2H_2O$ 186.1 g

Ultrapure water 800.0 ml

Adjust pH to 8.0 by 10 N NaoH

Add ultrapure water to be 1000 ml and sterilize by autoclaving

2.4 20% SDS

SDS 20.0 g

Sterile ultrapure water 80.0 ml

2.5 20 mg/ml Proteinase K stock solution

Proteinase K 100.0 mg

Sterile ultrapure water 5.0 ml

Mix and dispense in 500 µl in microcentrifuge tubesand store at -20°C

2.6 10% N-lauryl sarcosine, Sodium salt (Sarcosyl)

Sodium lauryl sarcosine 10.0 g

Sterile ultrapure water 90.0 ml

2.7 10X Tris-Borate EDTA (TBE) buffer, pH 8.3

Tris base 108.0 g

Boric acid 55.0 g

0.5 M EDTA, pH 8.0 40.0 ml

Dilute to 1000 ml with sterile ultrapure water and sterilize by autoclaving

2.8 Ethidium Bromide stock solution

Ethidium bromide 10.0 mg

Sterile ultrapure water 1.0 ml

For staining, dilute 10 µl in 100 ml distilled water

2.9 Tris-EDTA (TE) buffer, pH 8.0

1M Tris-HCl, pH 8.0 10.0 ml

0.5 M EDTA, pH 8.0 2.0 ml

Dilute to 1000 ml with sterile ultrapure water

2.10 Cell suspension buffer, pH 8.0

1M Tris-HCl, pH 8.0 10.0 ml

0.5 M EDTA, pH 8.0 20.0 ml

Dilute to 100 ml with sterile ultrapure water

2.11 Cell lysis buffer

1M Tris-HCl, pH 8.0 25.0 ml

0.5 M EDTA. pH 8.0	50.0 ml
10% Sodium lauryl sarcosine	50.0 ml
Dilute to 500 ml with sterile ultrapure water	

Script for MLST data analysis

Adenylate kinase (ADK)

(1)ATTCTGCTTGGCGCTCCGGGCGCGGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCA
AATCTCCACTGGCGATATGCTGCGTGCTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGG
ATGCTGGCAAACTGGTCACCGACGAACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAA
TGGTTTCCTGTTGGACGGCTTCCCGCGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGAT
TACGTTCTGGAATTCGACGTACCGGACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTG
GTCGTGTTTATCACGTTAAATTCAATCCGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACC
CGTAAAGATGATCAGGAAGAGACCGTACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCT
ACTACGCCAAAGAAGCAGAAGCGGGTAATACCAAATACGCGAAAGTTGACGG(584)

Fumarate hydratase (Fumc)

(1)GCGGCAAAAGTTAATGAAGATTTAGGCTTGTTGTCTGAAGAGAAAGCGAGCGCCATTCGTCAGGCGGCGGATGA
AGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGCAGACCGGCTCCGGCACGCAAAGTAACAT
GAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCGGTGTGCGCGGGATGGAACGTAAAGTTCA
CCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCCGACGGCGATGCACGTTGCGGCGCTGCTG
GCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGACACTGAATGAGAAATCCCGTGCTTTTGCCGA
TATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAACGCTGGGGCAGGAGATTTCCGGCTGGGTA
GCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCACGTAGCGGAACTGGCTCTTGGCGGTACAG
CGGTGGGTACTGGACTAAATACCCATCCGGA(560)

(gyrB) DNA gyrase B

(1)AGTGATCATGACCGTTCTGCACGCAGGCGGTAAATTTGACGATAACTCCTATAAAGTGTCCGGCGGTCTGCACGG
CGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTAAAATTCACCGTC
AGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCGGCACCATGGTGC
GTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAACGTCTGCGTGAG
TTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGATAAGCGCGACGGCAAAGAAGACCACTTCCACTATGA
AGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATCTTCTACTTCTCCA
CTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACATCTACTGCTTTACC
AACAACATTCCG(547)

ICD (isocitrate dehydrogenase)

(1)GCAAAACGGCAAACTCAACGTTCCTGAAAATCCGATTATCCCTTACATTG(51)AAGGTGATGGAATCGGTGTAGAT
GTAACTCCGACCATGCTGAAAGTGGTCGATGCTGCCGTCGAGAAAGCCTATAAAGGTGAGCGTAAAATCTCCTGGAT
GGAAATTTACACCGGTGAAAAATCCACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACCCTTGATCTGA
TTCGTGAATATCGCGTTGCTATCAAAGGCCCTCTGACCACTCCGGTTGGCGGCGGTATTCGTTCTCTTAACGTTGCCC

TGCGCCAGGAGCTGGATCTTTACATCTGCCTACGTCCGGTACGTTACTACCAGGGCACTCCAAGCCCGGTTAAACA
CCCTGAACTGACCGATATGGTTATCTTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACT
CTGCCGACGCAGAGAAAGTGATTAAATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGTTTCCCGGAACATTGT
GGTATCGGCATTAAGCCGTGTTCTGAAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAA
TGATCGAGACTCTGTGACTCTGGTGCACAAAGGCAACATCATGAA(657)ATTCACCGAAGGTGCGTTTAAAGACTGG
GGCTACCAGTTAGCGCGTGAAGAGTTTGGCGGAGAATTGATCGACGGCGGCCCGTGGCTGAAAGTCAAAAACCCG
AACACCGGCAAAGAGATCGTCATTAAAGACGTGATCGCTGATGCGT

MDH (malate dehydrogenase)

(1)GCGCAGATGTCGTTCTTATCTCTGCAGGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAA
CGCCGGCATCGTGAAAAACCTGGTACAGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAAC
CCGGTTAACACCACAGTTGCAATTGCTGCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGG
CGTTACCACGCTGGATATCATTCGTTCCAACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAG
TGCCGGTTATTGGCGGTCACTCTGGTGTTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGC
AGGAAGTGGCTGATCTGACCAAACGCATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGT
CTGCAACCCTGTCTATGGGCCAGGCAGCTGCACGTTTTGGTCTGTCT(506)

RECA (recombinase)

(1)GTTTATCGATGCTGAACACGCGCTGGACCCAATCTACGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTG
CTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAATCTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTAT
CGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGCGGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCT
TGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCTGGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTC
ATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCGGTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATT
CTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGCGCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAAC
CCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCCGTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGT
ATCAACTTCTACGGCGAACTGGTTGACCTGGGCGTAAAAGAGAAG(574)

purA (adenylate synthase)

(1)ATGTCCGCTGATCCTTGATTATCACGTTGCGCTGGATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCG
GCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAGATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTT
CGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTGATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGC
TGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACGATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGT
TTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCGATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGAT
ATCGACCACGGTACTTATCCGTACGTAACTTCTTCCAACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGG
GCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAAAGCTTACTCAACTCGTGTAGGTGCAGGTCCTTTCCCGACCG
AACTGTTTGATGAAACTGGCGAGTTCCTCTGCAAGCAGGGTAACGAATT(585)

Sequence data from HiMLST

ST10

Clonal complex (Cplx) = ST10 Cplx

Allelic profiles

adk 11

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTTCAAAGAAGCGGAAGCGGGT
A

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 8

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG

TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST31

Clonal complex (Cplx) = ST31 Cplx

Allelic profiles

adk 18

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGATCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCTAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCGT
GCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGGT
A

fumC 22

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTTGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACG
CTGAGTGAAAAATCCCGTGCATTTGCCGATATCGTAAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTAA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATTGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 17

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCTAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAAGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCAAATATC
TTCTACTTCTCCACCGAAAAAGATGGTATTGGCGTTGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 6

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTACGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGCATCGGTATTAAGCCGTGTTCTGA
AGAAGGTACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 5

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 4

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST44

Clonal complex (Cplx) = ST10 Cplx

Allelic profiles

adk 10

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCTACTACTCCAAAGAAGCAGAAGCGGG
TA

fumC 11

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCATAATGGTTTCCTGTTGGACGGCTTCCCGC
GTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGGA
CGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 8

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG
TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 7

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCACC
GTTTAAACAGGCTGAATTTCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST48

Clonal complex (Cplx) = ST10 Cplx

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC

ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 8

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG
TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG

GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST56

Clonal complex (Cplx) = ST155 Cplx

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 4

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 18

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAAGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA

ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATCAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTCCGTGCCGCGATTGAATACGCAATTGCCAACGACC

mdh 20

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACTTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 14

CGCACGTAAACTGGGCGTCGATATTGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCACC
GTTTAAACAGGCTGAATTTCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST69

Clonal complex (Cplx) = ST69 Cplx

Allelic profiles

adk 21

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTTACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGATCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCTAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCGT
GCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGGT
A

fumC 35

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCATTGGCTATCTGGC
AGACTGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACG
CTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTAAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTAA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATTGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 27

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCAAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGTAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGATGGTATCGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 6

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTACGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGCATCGGTATTAAGCCGTGTTCTGA
AGAAGGTACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 5

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 4

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST72

Clonal complex (Cplx) = ST405 Cplx

Allelic profiles

adk 35

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACTGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 37

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAATATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACA
CTGAATGAGAAATCGCGCGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGATGCCACGCCGCTAA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCGCA
TGTAGCGGAACTGGC

gyrB 29

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGTAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGATGGTATCGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 25

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA

GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACATCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCGGA
AGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 4

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGTGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 18

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGTGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACTACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCTCCG
TTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST82

Clonal complex (Cplx) = none

Allelic profiles

adk 38

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCAGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGCAACGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGACCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG

TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 39

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGTG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCACTACTGGCGCTGCGCAAGCAACTCATTCCACAACTTAAAACCCTGACCCAGACG
CTGAGTGAAAAATCCCGCGCATTTGCTGATATCGTCAAAATCGGTCGTACCCACTTGCAGGACGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTGGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 30

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACAG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 13

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGTTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 29

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACTACAGTTGCGATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACCCACTGTTCGGCGTTACCACGCTGGATATCATTCGTTC
CAACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGTGGTCACTCTGGT
GTTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACG
TATCCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 25

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTTGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCGA
TTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACATATCCGTACGTAACTTCTTCCAA

CACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAAA
GCTTACTCCACTCGTGT

recA 28

CGCACGTAAACTGGGCGTCGATATTGACAACCTGCTGTGCTCCCAGCCGGATACCGGCGAGCAGGCACTGGAAAT
CTGTGATGCCCTGGCACGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGCG
GAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCTG
GCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCGG
TAACCCGGAAACCACTACTGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGATATCCGTCGTATCGGCG
CGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCCGT
TTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST93

Clonal complex (Cplx) = ST168 Cplx

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 10

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 7

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 6

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST117

Clonal complex (Cplx) = none

Allelic profiles

adk 20

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA

ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGCAACGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGATCGTATCGTCGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCTAAAGAAGCGGAAGCGGGT
A

fumC 45

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGTG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCACTACTGGCGCTGCGCAAGCAACTCATTCCACAACTTAAAACCCTGACCCAGACG
CTGAGTGAAAAATCCCGCGTATTTGCTGATATCGTCAAAATCGGTCGTACCCACTTGCAGGACGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTGAAACATATCGAATACAGCCTGCCGCAT
GTAGCGGAACTGGC

gyrB 41

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCAAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAGAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 43

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATCAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTGCGTGCAGCGATCGAATACGCAATTGCGAACGATC

mdh 5

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 32

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGAGGTATCGGGCCTGCTTATGAA
GATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGT
GATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATAC
GATGGCTGTTGCCGACATCCTGACTTCTATGGTTGTTGACGTTTCTGACCTGCTTGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACATATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTAGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST155

Clonal complex (Cplx) = ST155 Cplx

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 4

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 14

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG

GCACCATGGTGCGTTTCTGGCCTAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 16

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAAGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 24

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACTGTTGCAATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 14

CGCACGTAAACTGGGCGTCGATATTGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCACC
GTTTAAACAGGCTGAATTTCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST193

Clonal complex (Cplx) = ST10 Cplx

Allelic profiles

adk 10

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCTACTACTCCAAAGAAGCAGAAGCGGG
TA

fumC 7

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGTATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 12

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG

TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST206

Clonal complex (Cplx) = ST206 Cplx

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 7

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGTATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 5

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 1

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG
TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 18

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCAACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST302

Clonal complex (Cplx) = none

Allelic profiles

adk 79

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTTACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 84

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCACTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCATTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGCTTA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 71

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATTCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 78

CGACGCAGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATATATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCCAACGATC

mdh 52

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACTGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATCCGTTC
CAACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGT
GTTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACG
TATCCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 57

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCTGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCGA
TTTCGTCATGTTCGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCAA
CACCACTGCTGGTGGTGTGGCGACCGGTTCCGGCTTGGGCCCGCGTTATGTTGATTACGTTCTCGGTATCCTCAAAG
CTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST365

Clonal complex (Cplx) = ST101 Cplx

Allelic profiles

adk 43

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGTTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 41

CGAGCGCCATTCGGCAGGCAGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC

GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCCCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 15

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCATCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTAGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 18

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAAGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATCAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTCCGTGCCGCGATTGAATACGCAATTGCCAACGACC

mdh 11

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTTCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCAGGTACTGAAGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 11

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTTGTTGACGTTTCTGATCTGCTCGACCAGGCGCGTCAGCGTGGCGA
TTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGCACTTATCCGTACGTAACTTCTTCCAA
CACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAAA
GCTTACTCCACTCGTGT

recA 6

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT

GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST378

Clonal complex (Cplx) = ST10 Cplx

Allelic profiles

adk 10

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCTACTACTCCAAAGAAGCAGAAGCGGG
TA

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 1

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA

ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG
TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 66

ATAACGCGCGTGAGAAAGCGCGCGGCGCGAAAGCGATCGGCACCACGGGGCGTGGTATCGGGCCTGCTTATGAA
GATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGT
GATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGTTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATGT
GATGGCCGTTGCCGACATCCTGACTTCCATGGTGGTTGACGTTTCTGACCTGCTTGACCAGGCGCGTCAGCGTGGC
GATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTTCTGGATATCGACCACGGTACTTATCCGTACGTAACCTCTTCC
AACACCACTGCGGGCGGCGTGGCGACCGGTTCCGGCCTGGGCCCACGTTATGTTGATTACGTTCTGGGTATCCTCA
AAGCTTACTCTACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST393

Clonal complex (Cplx) = ST31 Cplx

Allelic profiles

adk 18

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGATCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCTAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCGT
GCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGGT
A

fumC 106

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTTGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACG
CTGAGTGAAAAATCCCGTGCATTTGCCGATATCGTAAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTAA
CACTGGGACAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATTGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 17

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCTAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAAGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCAAATATC
TTCTACTTCTCCACCGAAAAAGATGGTATTGGCGTTGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 6

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTACGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGCATCGGTATTAAGCCGTGTTCTGA
AGAAGGTACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 5

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 4

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST402

Clonal complex (Cplx) = ST405 Cplx

Allelic profiles

adk 35

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACTGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 35

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCATTGGCTATCTGGC
AGACTGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACG
CTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTAAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTAA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATTGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 29

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGTAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGATGGTATCGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 25

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA

GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACATCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCGGA
AGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 4

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGTGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 73

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGTGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCTCCG
TTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST405

Clonal complex (Cplx) = ST405 Cplx

Allelic profiles

adk 35

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACTGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG

TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 37

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAATATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACA
CTGAATGAGAAATCGCGCGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGATGCCACGCCGCTAA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCGCA
TGTAGCGGAACTGGC

gyrB 29

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGTAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGATGGTATCGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 25

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACATCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCGGA
AGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 4

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGTGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA

ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 73

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGTGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCTCCG
TTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST460

Clonal complex (Cplx) = ST168 Cplx

Allelic profiles

adk 56

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAACGGG
TA

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 10

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 7

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST484

Clonal complex (Cplx) = ST168 Cplx

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG

ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 10

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 7

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 84

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG

ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGATCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTATGTTCTGGGTATCCTCAA
AGCTTACTCAACTCGTGT

recA 6

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST571

Clonal complex (Cplx) = ST10 Cplx

Allelic profiles

adk 112

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTTACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGTAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAAACCGT
ACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGGT
A

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC

TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 1

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG
TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST597

Clonal complex (Cplx) = ST69 Cplx

Allelic profiles

adk 21

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTTACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGATCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCTAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCGT
GCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGGT
A

fumC 35

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCATTGGCTATCTGGC
AGACTGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACG
CTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTAAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTAA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATTGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 115

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCAAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGTAAAGAAAACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGATGGTATCGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 6

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTACGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGCATCGGTATTAAGCCGTGTTCTGA
AGAAGGTACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 5

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT

ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 4

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST604

Clonal complex (Cplx) = ST101 Cplx

Allelic profiles

adk 43

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGTTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 41

CGAGCGCCATTCGGCAGGCAGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCCCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 15

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCATCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTAGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 18

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAAGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATCAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTCCGTGCCGCGATTGAATACGCAATTGCCAACGACC

mdh 59

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCTGGCATCGTAAAAAACCTGGTACA
GCAAGTTGCGAAAACTTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACTACAGTGGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 7

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTATGTTCTGGGTATCCTCAA
AGCTTACTCAACTCGTGT

recA 6

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST619

Clonal complex (Cplx) = ST101 Cplx

Allelic profiles

adk 43

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGTTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 41

CGAGCGCCATTCGGCAGGCAGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCCCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 15

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCATCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTAGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 18

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAAGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATCAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTCCGTGCCGCGATTGAATACGCAATTGCCAACGACC

mdh 59

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCTGGCATCGTAAAAAACCTGGTACA
GCAAGTTGCGAAAACTTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACTACAGTGGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 7

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTATGTTCTGGGTATCCTCAA
AGCTTACTCAACTCGTGT

recA 6

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST621

Clonal complex (Cplx) = ST155 Cplx

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 4

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC

GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 12

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCTAGCCTCGAAACTTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 16

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAAGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 36

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGTATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACTACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGTGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 14

CGCACGTAAACTGGGCGTCGATATTGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT

GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCACC
GTTTAAACAGGCTGAATTTCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST638

Clonal complex (Cplx) = ST73 Cplx

Allelic profiles

adk 76

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGCAACGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGACCGTATCGTAGGCCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 24

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGTTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCACAACTTAAAACCCTGACCCAGAC
GCTGAGTGAAAAATCCCGCGCATTTGCCGATATCGTCAAAATCGGTCGTACCCACTTGCAGGACGCCACGCCGTTA
ACGCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTGGAGCATAATCTCAAACATATCGAATACAGCCTGCCTC
ACGTAGCGGAACTGGC

gyrB 9

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACTATGGTGCGTTTCTGGCCAAGCCTTGAAACCTTCACCAATGTGACCGAGTTCGAATATGACATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGATGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCATTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 13

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGTTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAA

ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 17

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACTACAGTTGCGATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGTGGTCACTCTGGTGT
TACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTA
TCCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 11

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTTGTTGACGTTTCTGATCTGCTCGACCAGGCGCGTCAGCGTGGCGA
TTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGCACTTATCCGTACGTAACTTCTTCCAA
CACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAAA
GCTTACTCCACTCGTGT

recA 25

ST648

Clonal complex (Cplx) = ST468 Cplx

Allelic profiles

adk 92

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTCGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCTAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCGT
GCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCAGGT
A

fumC 4

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 87

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGATAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 96

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTACCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATCAAA
GGCCCGCTGACCACTCCGGTTGGTGGTGGTATTCGCTCTCTCAACGTTGCTCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGCATCGGTATTAAGCCGTGTTCTGA
AGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 70

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTGGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 58

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTTGTTGACGTTTCTGATCTGCTCGACCAGGCGCGTCAGCGTGGCGA
TTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCAA
CACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCACGTTATGTTGATTACGTTCTGGGTATCCTCAAA
GCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST656

Clonal complex (Cplx) = ST10 Cplx

Allelic profiles

adk 10

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCTACTACTCCAAAGAAGCAGAAGCGGG
TA

fumC 7

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGTATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 4

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 8

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG
TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST877

Clonal complex (Cplx) = none

Allelic profiles

adk 9

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCATAATGGTTTCCTGTTGGACGGCTTCCCGC
GTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGGA
CGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 175

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGATGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC

GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 33

GGTCTGCACGGCGTTGGTGTTTCGGTTGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 131

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGGGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGTGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAATGATC

mdh 24

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACTGTTGCAATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 7

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT

GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCACC
GTTTAAACAGGCTGAATTTCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST953

Clonal complex (Cplx) = none

Allelic profiles

adk 8

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTTGATGTACCGGA
CGAACTGATCGTTGACCGTATCGTGGGTCGCCGTGTTCACGCGCCGTCTGGCCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGTAAAGACGACGTTACCGGTGAAGAGCTGACCACCCGTAAAGATGATCAGGAAGAAACCG
TACGTAAACGTCTGGTTGAATATCATCAGATGACTGCGCCGCTGATTGGCTACTATTCAAAAGAAGCTGAAGCGGGT
A

fumC 7

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGTATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 1

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGTGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCATCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 8

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCGGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACA
TCTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATC
TTCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA

ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 125

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCTGTTATTGGCGGTCACTCTGGTGT
TACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGCA
TCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 6

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST963

Clonal complex (Cplx) = none

Allelic profiles

adk 4

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTTACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATTGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAACC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 26

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCATTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGTGGGATGGAGCGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCTAACGATGTCTTTCCA
ACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGACG
CTGAGTGAAAAATCCCGTGCATTTGCCGATATCGTAAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTAA
CACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATTGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 151

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
ATATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCGG
CACCATGGTGCGTTTCTGGCCAAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAAC
GTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGAGACAAGCGCGACGGTAAAGAAGACCA
CTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATCT
TCTACTTCTCCACCGAGAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 25

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGCCCTCTGACCACTCCGGTTGGTGGCGGTATTCGTTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACCCCAAGCCCGGTTAAACATCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCTGACTCTGCCGACGCCGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATTAAGCCGTGTTCGGA
AGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 5

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGCTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 5

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTCGAAGGTGCTCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACATCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 19

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGTGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST1119

Clonal complex (Cplx) = ST468 Cplx

Allelic profiles

adk 92

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTCGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCTAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCGT
GCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCAGGT
A

fumC 4

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 87

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGATAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 96

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTACCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATCAAA

GGCCCGCTGACCACTCCGGTTGGTGGTGGTATTCGCTCTCTCAACGTTGCTCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGCATCGGTATTAAGCCGTGTTCTGA
AGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 70

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTGGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
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ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 144

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTTGTTGACGTTTCTGATCTGCTCGACCAGGCGCGCCAGCGTGGCGA
TTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCAA
CACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCACGTTATGTTGATTACGTTCTGGGTATCCTCAAA
GCTTACTCCACTCGTGT

recA 2

CGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST2311

Clonal complex (Cplx) = ST468 Cplx

Allelic profiles

adk 92

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTCGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCTAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCGT

GCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCAGGT
A

fumC 4

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 87

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGATAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 96

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTACCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATCAAA
GGCCCGCTGACCACTCCGGTTGGTGGTGGTATTCGCTCTCTCAACGTTGCTCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCTGAGAAAGTGATTAAA
TTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGCATCGGTATTAAGCCGTGTTCTGA
AGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 70

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTGGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 58

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTGGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTTGTTGACGTTTCTGATCTGCTCGACCAGGCGCGTCAGCGTGGCGA
TTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCAA

CACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCACGTTATGTTGATTACGTTCTGGGTATCCTCAAA
GCTTACTCCACTCGTGT

recA 18

TGCACGTAAACTGGGCGTCGATATCGACAACCTGCTGTGCTCCCAGCCGGACACTGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGTGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACTACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGTGTGAAAGTGGTGAAGAACAAAATCGCTGCTCCG
TTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTATGGCGA

ST2753

Clonal complex (Cplx) = none

Allelic profiles

adk 10

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGATGATCAGGAAGAGACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACAGCACCGCTGATCGGCTACTACTCCAAAGAAGCAGAAGCGGG
TA

fumC 11

CGAGCGCCATTCGTCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GTGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCTCAGCTTAAAACCCTGACACAGAC
ACTGAATGAGAAATCCCGTGCTTTTGCCGATATCGTCAAAATTGGTCGTACTCACTTGCAGGATGCCACGCCGTTAAC
GCTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCAC
GTAGCGGAACTGGC

gyrB 5

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 10

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGACGTCTGGCTGCCTGCTGAAACTCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 8

GGCGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTAC
AGCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCAATTGCT
GCTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCC
AACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTG
TTACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGC
ATCCAGAACGCGGGTACTGAAGTGGTTGAAGCGAAGGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 8

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTACGTTCTGGGTATCCTCAA
AGCTTACTCCACTCGTGT

recA 6

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCGCC
GTTTAAACAGGCTGAATTCCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST3379

Clonal complex (Cplx) = none

Allelic profiles

adk 6

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTCACCGACGA

ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATTGTTGATCGTATCGTAGGCCGCCGCGTTCATGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TGCGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATCGGCTACTACTCCAAAGAAGCGGAAGCGGG
TA

fumC 95

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTCAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
GCTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTAAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTGGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTC
ACGTAGCGGAACTGGC

gyrB 3

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCCCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG
GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGTGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACCGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 18

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAAGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGCGTGAAGAAAATTCGCTTCCCGGAACATTGCGGTATCGGTATCAAGCCGTGTTCTG
AAGAAGGCACCAAACGTCTGGTCCGTGCCGCGATTGAATACGCAATTGCCAACGACC

mdh 24

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTGCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACTGTTGCAATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCGGGTACTGAGGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 7

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTATGTTCTGGGTATCCTCAA
AGCTTACTCAACTCGTGT

recA 14

CGCACGTAAACTGGGCGTCGATATTGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCACC
GTTTAAACAGGCTGAATTTCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

ST4198

Clonal complex (Cplx) = none

Allelic profiles

adk 137

GGGGAAAGGGACTCAGGCTCAGTTCATCATGGAGAAATATGGTATTCCGCAAATCTCCACTGGCGATATGCTGCGTG
CTGCGGTCAAATCTGGCTCCGAGCTGGGTAAACAAGCAAAAGACATTATGGATGCTGGCAAACTGGTTACCGACGA
ACTGGTGATCGCGCTGGTTAAAGAGCGCATTGCTCAGGAAGACTGCCGTAATGGTTTCCTGTTGGACGGCTTCCCG
CGTACCATTCCGCAGGCAGACGCGATGAAAGAAGCGGGCATCAATGTTGATTACGTTCTGGAATTCGACGTACCGG
ACGAACTGATCGTTGACCGTATCGTCGGTCGCCGCGTTCACGCGCCGTCTGGTCGTGTTTATCACGTTAAATTCAATC
CGCCGAAAGTAGAAGGCAAAGACGACGTTACCGGTGAAGAACTGACTACCCGTAAAGACGATCAGGAAGAAACCG
TACGTAAACGTCTGGTTGAATACCATCAGATGACTGCACCGCTGATTGGCTACTACTCCAAAGAAGCGGAAGCGGGT
A

fumC 4

CGAGCGCCATTCGGCAGGCGGCGGATGAAGTACTGGCAGGACAGCATGACGACGAATTCCCGCTGGCTATCTGGC
AGACCGGCTCCGGCACGCAAAGTAACATGAACATGAACGAAGTGCTGGCTAACCGGGCCAGTGAATTACTCGGCG
GCGTGCGCGGGATGGAACGTAAAGTTCACCCTAACGACGACGTGAACAAAAGCCAAAGTTCCAACGATGTCTTTCC
GACGGCGATGCACGTTGCGGCGCTGCTGGCGCTGCGCAAGCAACTCATTCCGCAGCTTAAAACCCTGACACAGAC
ACTGAGTGAAAAATCGCGTGCATTTGCCGATATCGTCAAAATCGGTCGAACCCACTTGCAGGACGCCACGCCGCTA
ACACTAGGGCAGGAGATTTCCGGCTGGGTAGCGATGCTCGAGCATAATCTCAAACATATCGAATACAGCCTGCCTCA
CGTAGCGGAACTGGC

gyrB 5

GGTCTGCACGGCGTTGGTGTTTCGGTAGTAAACGCCCTGTCGCAAAAACTGGAGCTGGTTATCCAGCGCGAGGGTA
AAATTCACCGTCAGATCTACGAACACGGTGTACCGCAGGCTCCGCTGGCGGTTACCGGCGAGACTGAAAAAACCG

GCACCATGGTGCGTTTCTGGCCCAGCCTCGAAACCTTCACCAATGTGACCGAGTTCGAATATGAAATTCTGGCGAAA
CGTCTGCGTGAGTTGTCGTTCCTCAACTCCGGCGTTTCCATTCGTCTGCGCGACAAGCGCGACGGCAAAGAAGACC
ACTTCCACTATGAAGGCGGCATCAAGGCGTTCGTTGAATATCTGAACAAGAACAAAACGCCGATCCACCCGAATATC
TTCTACTTCTCCACTGAAAAAGACGGTATTGGCGTCGAAGTGGCGTTGCAGTGGAACGATGGCTTCCAGGAAAACAT
CT

icd 1

CGACGCTGCAGTCGAGAAAGCCTATAAAGGCGAGCGTAAAATCTCCTGGATGGAAATTTACACCGGTGAAAAATCC
ACACAGGTTTATGGTCAGGATGTCTGGCTGCCTGCTGAAACCCTTGATCTGATTCGTGAATATCGCGTTGCCATTAAA
GGTCCGCTGACCACTCCTGTTGGTGGCGGTATTCGCTCTCTGAACGTTGCCCTGCGCCAGGAACTGGATCTCTACAT
CTGCCTGCGTCCGGTACGTTACTATCAGGGCACTCCAAGCCCGGTTAAACACCCTGAACTGACCGATATGGTTATCT
TCCGTGAAAACTCGGAAGACATTTATGCGGGTATCGAATGGAAAGCAGACTCTGCCGACGCCGAGAAAGTGATTAA
ATTCCTGCGTGAAGAGATGGGGGTGAAGAAAATTCGCTTCCCGGAACATTGTGGTATCGGTATTAAGCCGTGTTCGG
AAGAAGGCACCAAACGTCTGGTTCGTGCAGCGATCGAATACGCAATTGCTAACGATC

mdh 11

GGTGTAGCGCGTAAACCGGGTATGGATCGTTCCGACCTGTTTAACGTTAACGCCGGCATCGTGAAAAACCTGGTACA
GCAAGTTTCGAAAACCTGCCCGAAAGCGTGCATTGGTATTATCACTAACCCGGTTAACACCACAGTTGCGATTGCTG
CTGAAGTGCTGAAAAAAGCCGGTGTTTATGACAAAAACAAACTGTTCGGCGTTACCACGCTGGATATCATTCGTTCCA
ACACCTTTGTTGCGGAACTGAAAGGCAAACAGCCAGGCGAAGTTGAAGTGCCGGTTATTGGCGGTCACTCTGGTGTT
ACCATTCTGCCGCTGCTGTCACAGGTTCCTGGCGTTAGTTTTACCGAGCAGGAAGTGGCTGATCTGACCAAACGTAT
CCAGAACGCAGGTACTGAAGTGGTTGAAGCGAAAGCCGGTGGCGGGTCTGCAACCCTGTCTATGGG

purA 7

ATAACGCGCGTGAGAAAGCGCGTGGCGCGAAAGCGATCGGCACCACCGGTCGTGGTATCGGGCCTGCTTATGAAG
ATAAAGTAGCACGTCGCGGTCTGCGTGTTGGCGACCTTTTCGACAAAGAAACCTTCGCTGAAAAACTGAAAGAAGTG
ATGGAATATCACAACTTCCAGTTGGTTAACTACTACAAAGCTGAAGCGGTTGATTACCAGAAAGTTCTGGATGATACG
ATGGCTGTTGCCGACATCCTGACTTCTATGGTGGTTGACGTTTCTGACCTGCTCGACCAGGCGCGTCAGCGTGGCG
ATTTCGTCATGTTTGAAGGTGCGCAGGGTACGCTGCTGGATATCGACCACGGTACTTATCCGTACGTAACTTCTTCCA
ACACCACTGCTGGTGGCGTGGCGACCGGTTCCGGCCTGGGCCCGCGTTATGTTGATTATGTTCTGGGTATCCTCAA
AGCTTACTCAACTCGTGT

recA 7

CGCACGTAAACTGGGCGTCGATATCGATAACCTGCTGTGCTCCCAGCCGGACACCGGCGAGCAGGCACTGGAAAT
CTGTGACGCCCTGGCGCGTTCTGGCGCAGTAGACGTTATCGTCGTTGACTCCGTGGCGGCACTGACGCCGAAAGC
GGAAATCGAAGGCGAAATCGGCGACTCTCACATGGGCCTTGCGGCACGTATGATGAGCCAGGCGATGCGTAAGCT
GGCGGGTAACCTGAAGCAGTCCAACACGCTGCTGATCTTCATCAACCAGATCCGTATGAAAATTGGTGTGATGTTCG
GTAACCCGGAAACCACTACCGGTGGTAACGCGCTGAAATTCTACGCCTCTGTTCGTCTCGACATCCGTCGTATCGGC
GCGGTGAAAGAGGGCGAAAACGTGGTGGGTAGCGAAACCCGCGTGAAAGTGGTGAAGAACAAAATCGCTGCACC
GTTTAAACAGGCTGAATTTCAGATCCTCTACGGCGAAGGTATCAACTTCTACGGCGA

VITA

NAME: Kitttitat Lugsomya

DATE OF BIRTH: 7 September 1984

EDUCATION: Doctor of Philosophy Program in Veterinary Pathobiology (2011 – present)

: Master of Science Program in Veterinary Pathobiology (2009 – 2011)

: Doctor of Veterinary Medicine (D.V.M.) (2002 - 2008)

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PUBLICATIONS:

1.Routine prophylactic antimicrobial use is associated with increased phenotypic and genotypic resistance in commensal Escherichia coli isolates recovered from healthy fattening pigs on farms in Thailand Lugsomya K, Chatsuwan T, Niyomtham W, Tummaruk P, Hampson DJ and Prapasarakul N Microb Drug Resist. (Page Proofs) 23 MDR-2017-0042.

2.Molecular detection and isolation of pathogenic Leptospira from asymptomatic humans, domestic animals and water sources in Nan Province, a rural area of Thailand Kurilung A. Chanchaithong P Lugsomya K, Niyomtham W, Wuthiekanun V, Prapasarakul. Res Vet Sci. 2017 Mar:115:146-154.

3.Prevalence of Plasmid-mediated colistin resistance genes (mcr-1) in commensal Escherichia coli from fattening pigs in Thailand. Lugsomya K, Chanchaithong P, Tribuddharat C, Thanawanh N, Niyomtham W and Prapasarakul N Thai J Vet supple 2016 46:327-8.

4.Salmonella serovar distribution in cobras (Naja kaouthia), snake-food species, and farm workers at Queen Saovabha Snake Park, Thailand.Prapasarakul N, Pulsrikarn C, Vasaruchapong T, Lekcharoen P, Chanchaithong P, Lugsomya K, Keschumras N, Thanomsuksinchai N, Tanchiangsai K, Tummaruk P.J Vet Diagn Invest. 2012 Mar;24(2):288-94.

5.Development of a modified selective medium to enhance the recovery rate of Brachyspira hyodysenteriae and other porcine intestinal spirochaetes from faeces. Lugsomya K, Tummaruk P, Hampson DJ, Prapasarakul N. Lett Appl Microbiol. 2012 Apr;54(4):330-5.

6.Faecal excretion of intestinal spirochaetes by urban dogs, and their pathogenicity in a chick model of intestinal spirochaetosis.Prapasarakul N, Lugsomya K, Disatian S, Lekdumrongsak T, Banlunara W, Chetanachan P, Hampson DJ.Res Vet Sci. 2011 Dec;91(3):e38-43.