Appl Microbiol Biotechnol (2015) 99:2533–2548
DOI 10.1007/s00253-015-6439-y

MINI-REVIEW

Acinetobacter species as model microorganisms in environmental microbiology: current state and perspectives

Jaejoon Jung · Woojun Park

Received: 24 October 2014 / Revised: 23 January 2015 / Accepted: 26 January 2015 / Published online: 19 February 2015

Abstract *Acinetobacter* occupies an important position in nature because of its ubiquitous presence in diverse environments such as soils, fresh water, oceans, sediments, and contaminated sites. Versatile metabolic characteristics allow species of this genus to catabolize a wide range of natural compounds, implying active participation in the nutrient cycle in the ecosystem. On the other hand, multi-drug-resistant *Acinetobacter baumannii* causing nosocomial infections with high mortality has been raising serious concerns in medicine. Due to the ecological and clinical importance of the genus, *Acinetobacter* was proposed as a model microorganism for environmental microbiological studies, pathogenicity tests, and industrial production of chemicals. For these reasons, *Acinetobacter* has attracted significant attention in scientific and biotechnological fields, but only limited research areas such as natural transformation and aromatic compound degradation have been intensively investigated, while important physiological characteristics including quorum sensing, motility, and stress response have been neglected. The aim of this review is to summarize the recent achievements in *Acinetobacter* research with a special focus on strain DR1 and to compare the similarities and differences between species or other genera. Research areas that require more attention in future research are also suggested.

Keywords Taxonomy · Genomics · Quorum sensing · Motility · Antibiotic resistance · Hydrocarbon

J. Jung · W. Park (✉)
Laboratory of Molecular Environmental Microbiology, Department of Environmental Science and Ecological Engineering, Korea University, Seoul 136-713, Republic of Korea
e-mail: wpark@korea.ac.kr

Introduction

Acinetobacter, belonging to γ-Proteobacteria and *Pseudomonadales* order, is a genus of gram-negative, oxidase-negative, and strictly aerobic bacteria. The genus includes both nonpathogenic and pathogenic species (de Berardinis et al. 2009). *Acinetobacter* species prevail in natural environments, including soils, fresh water, oceans, sediments, the polar region, and hydrocarbon-contaminated sites (Kostka et al. 2011; Mahjoubi et al. 2013) as deduced by the estimated number of *Acinetobacter* cells: 10^5/mg in soil and 10^5/ml in water (Baumann 1968). *Acinetobacter* species harbor versatile metabolic capabilities such as pathways for degradation of various long-chain dicarboxylic acids and aromatic and hydroxylated aromatic compounds that are associated with plant degradation products (Yoshida et al. 1975). *Acinetobacter* metabolic pathways and regulatory mechanisms have received extensive attention, and most research results have been obtained using *Acinetobacter baylyi* ADP1. Natural transformation of *Acinetobacter* species has also attracted considerable interest, due to their ability to be manipulated by homology-directed recombination with linear DNA fragments (de Vries and Wackernagel 2002; Metzgar et al. 2004; Young et al. 2005). In addition to the ecological importance of *Acinetobacter*, the pathogenic *Acinetobacter baumannii* causes a wide range of infections, especially in hospital intensive care units (Fiester and Actis 2013). Because of the multi-drug resistance trait and rapid resistance development of *A. baumannii*, the advent of a pan-drug-resistant *A. baumannii* strain is anticipated with concern (Abbott et al. 2013). Therefore, remarkable research efforts are being devoted to developing identification of mechanisms of antibiotic resistance, genome plasticity, horizontal gene transfer, inhibitors of certain phenotypes, and diagnostic methods for *A. baumannii*.

With the increasing need for a model microorganism in the environmental microbiology field (other than traditional model organisms such as *Escherichia coli*), *Acinetobacter* species were suggested as a promising candidate because of ecological and clinical importance. Due to enteric characteristics of *E. coli*, the findings of studies on this species often may not be applicable to environmental isolates, and this discrepancy hinders the elucidation of the role of bacteria in a natural environment (Metzgar et al. 2004; Jacobs et al. 2014a). One of the important factors that make *Acinetobacter* sp. a fascinating model microorganism is the simplicity of genetic manipulation because of its natural transformability (Elliott and Neidle 2011). Additionally, these species possess diverse physiological characteristics associated with the important microbiological aspects, such as biofilms, quorum sensing, natural transformation, oxidative stress, antibiotic resistance, motility, genome evolution, and hydrocarbon degradation (Table 1). Traditionally, the major research area regarding *Acinetobacter* includes natural transformation, aromatic compound degradation, and hydrocarbon utilization, as shown in Fig. 1. Emerging topics for this genus are biotechnological applications, genomes analysis and evolution, antibiotic resistance, and diagnosis of pathogenic strains, while many aspects of motility, quorum sensing, diverse stress responses, and the regulatory system remain to be elucidated (Fig. 1). Numerous review articles related to the pathogenicity and antibiotic

Table 1 Representative research areas on *Acinetobacter*

Species/strain	Isolation source	Major research	References
Acinetobacter oleivorans DR1	Soil	Hydrocarbon degradation, antibiotic resistance, bioreporter, quorum sensing, persister cell formation, cell–cell interactions, comparative genomics, motility, biofilm, natural transformation	Park and Park (2011), Hong and Park (2014), Hong et al. (2014), Kim et al. (2013), Kim and Park (2013a, b), Seo et al. (2012), and Jung et al. (2011a, b)
Acinetobacter baylyi ADP1	Soil	Bioreporter, global transcriptional regulator, ester production, DNA damage response, natural transformation	Song et al. (2014), Withers et al. (2014), Santala et al. (2014), Hare et al. (2014), and Overballe-Petersen et al. (2013)
Acinetobacter baumannii ATCC 17978	Clinic	Biofilm, motility, natural transformation, blue-light regulation, transcriptomic analysis related to imipenem, small RNA, oxidative stress, DNA damage and repair	Mussi et al. (2010), Soares et al. (2010), Chang et al. (2014), Sharma et al. (2014), and Hare et al. (2014)
A. baumannii ATCC 19606	Clinic	Oxidative stress	Zimbler et al. (2012)
A. baumannii sequence type 2	Clinic	Multi-drug resistance	Perilli et al. (2014)
A. baumannii BM4454	Clinic	Efflux pump related to MDR	Sugawara and Nikaido (2014)
A. baumannii M2	Clinic	Quorum sensing	Stacy et al. (2012)
Many strains of *A. baumannii*	Clinic	Classification of β-lactamase, lipopolysaccharide	Hakemi Vala et al. (2014) and Kenyon et al. (2014)
Acinetobacter sp. YC-X 2	Soil	Biosurfactant production	Chen et al. (2012)
Acinetobacter calcoaceticus PHEA-2	Wastewater	Comparative genomics, phenol degradation	Zhan et al. (2012)
A. calcoaceticus YC210	Wastewater	Dye decolorization	Chen et al. (2011)
ACB complex	Clinic	Multi-drug resistance	Figueiredo et al. (2012)
Acinetobacter ursingii	Clinic	DNA repair	Hare et al. (2012)
Acinetobacter gerneri P7	Soil	Polyurethane degradation	Howard et al. (2012)
A. calcoaceticus NCIB 8250	Soil	Bioreporter for phenol	Peng et al. (2010)
A. calcoaceticus 1-7	Soil	Lipase production	Khoramnia et al. (2011)
Acinetobacter johnsonii strains	Soil	Lipase production	Wang et al. (2012)
Acinetobacter sp. D3-2	Soil	Biosurfactant production	Bao et al. (2014)
Acinetobacter venetianus RAG-1	Ocean	Biosurfactant production, genetic regulation of biosurfactants, chemical composition of emulsan	Fondi et al. (2012) and Nakar and Gutnick (2003)
Acinetobacter sp. SY-01	Soil	Characterization of novel enantioselective lipase	Han et al. (2003)
A. venetianus VE-C3	Soil	Comparative genomic study, hydrocarbon degradation	Fondi et al. (2013)
Acinetobacter haemolyticus NCTC 10305	Clinic	Lipopolysaccharide	Zähringer et al. (2013)

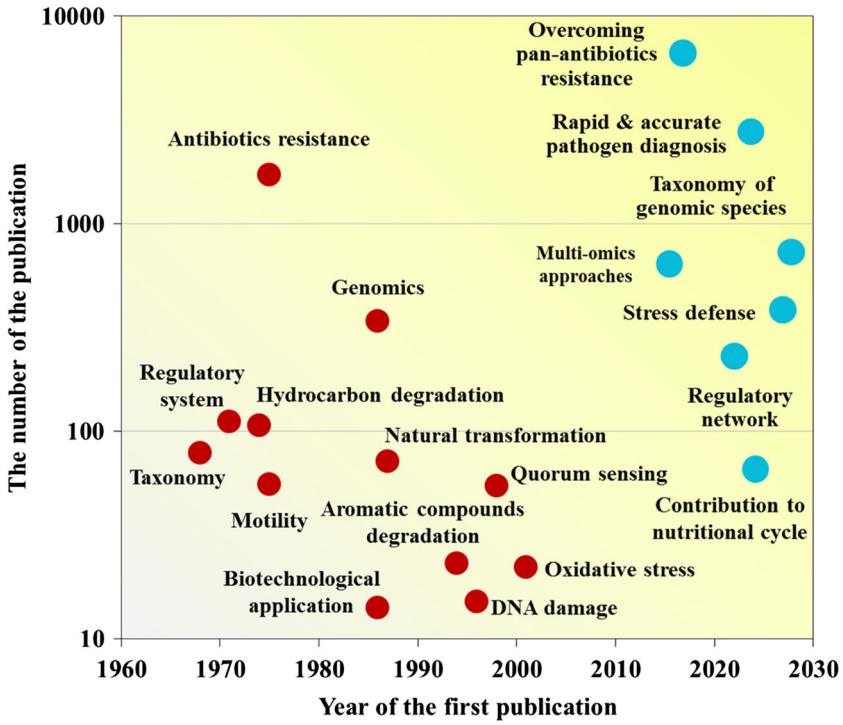

Fig. 1 Schematic presentation of research topics related to the genus *Acinetobacter*. The *x*-axis shows time points of recognition of research fields by the publication year of the first article. The *y*-axis shows the accumulated knowledge on a topic according to the number of articles. *Red squares* indicate a research area that is being currently investigated. *Blue circles* are the suggestions for future studies

resistance of *A. baumannii* are frequently published, whereas in the past 5 years, only two review articles about non-*A. baumannii* species have been published (de Berardinis et al. 2009; Elliott and Neidle 2011). Moreover, no article is covering diverse subjects investigated from *Acinetobacter*. Therefore, we aimed to provide a comprehensive and comparative review of the recent findings about *Acinetobacter*, covering both well-defined and poorly explored topics so that the foundation for future research directions on *Acinetobacter* species can be established.

Complexity of *Acinetobacter* taxonomy from historical and methodological standpoints

An isolate was obtained from a calcium-acetate-minimal medium by Beijerinck in 1911 and named *Micrococcus calcoaceticus*. Characterization of this strain was continued by Brisou and Prévot, and the name *Acinetobacter* was proposed by Baumann in 1968 (Brisou and Prevot 1954; Baumann et al. 1968). The genus *Acinetobacter* entered the official taxonomic system via appearance on the Approved Lists of Bacterial Names and Bergey's Manual of Systematic Bacteriology in 1984 (Skerman et al. 1980; Juni 1984). Since the establishment of this genus, biochemical heterogeneity has been recognized. However, a detailed description of *Acinetobacter* species has not been provided. Until 1986, only two species, *Acinetobacter calcoaceticus* and *Acinetobacter lwoffii*, had been validly classified (Skerman et al. 1980). The first modern taxonomic study of *Acinetobacter* was performed by Bouvet and Grimont (1986). They provided a description of *A. baumannii*, *Acinetobacter haemolyticus*, *Acinetobacter johnsonii*, and *Acinetobacter junii* and amended the description of *A. calcoaceticus* and *A. lwoffii* with evidence of DNA relatedness, biochemical tests, and nutrient requirements to delineate previously identified genospecies. The taxonomy of *Acinetobacter* has been ambiguous due to a lack of biochemical markers proven to distinguish bacterial strains, and they stayed on as genomic species. The name of a genomic species derives from the author's name (e.g., *Acinetobacter* genomic species 13TU proposed by *T*-jernberg and *U*rsing). Attempts to identify genomic species as separate species continued, and the valid names *Acinetobacter bereziniae*, *Acinetobacter guillouiae*, *Acinetobacter pittii*, and *Acinetobacter nosocomialis* were given to genomic species BG10, BG11, BG3, and 13TU, respectively (Nemec et al. 2010, 2011). Since those early members of *Acinetobacter* were reported, the discovery of novel species has continued, and this genus now contains 34 species with valid publications according to the list of prokaryotic names with standing in nomenclature (www.bacterio.net; as of August 2014). It should be noted that the strain that was originally described as BD413 is the same as strain ADP1 of *A. baylyi* (Vaneechoutte et al. 2006). Taxonomic resolution of *Acinetobacter* strains at the species level is still a challenging task, especially in the case of the *A. calcoaceticus*/*baumannii* (ACB) complex containing *A. nosocomialis* and *A. pittii* (Chan et al. 2012) because phenotypic differences within the same genotypes are not easy to detect by means of currently used phenotypic assays (Peleg et al. 2008). Recently, there was an attempt to apply whole-genome data to taxonomic

analysis, and the researchers succeeded in delineating genomic species of *Acinetobacter* (Chan et al. 2012). Nonetheless, the application of genomic data to taxonomic analysis without phenotypic experiments has been neither validated nor accepted.

Diverse DNA fingerprinting techniques and other biochemical analytical methods have been utilized to differentiate *Acinetobacter* strains because phenotypic characteristics are highly similar across the strains. Phylogenetic analysis based on housekeeping genes other than the 16S ribosomal RNA (rRNA) gene has been considered. Phylogeny based on the RNA polymerase subunit B (*rpoB*) gene sequence correlates with DNA–DNA hybridization (DDH) results and average nucleotide identity (ANI), and it was suggested as a costandard for the conventional 16S rRNA gene-based phylogeny (Adékambi et al. 2008). Phylogenetic analysis based on the DNA gyrase subunit B (*gyrB*) gene also provided an informative glimpse into *Acinetobacter* taxonomy, although it did not offer as good a resolution as did the *rpoB* gene. Because two fundamental and traditional molecular assays that are used in taxonomic analysis, 16S rRNA gene sequence similarity and DDH, have not adequately resolved the complexity of the *Acinetobacter* taxonomy, the importance of phylogenetic analysis involving housekeeping genes increased. As a result, recent projects routinely involved phylogenetic analysis based on housekeeping genes such as *rpoB* and *gyrB*, as well as the 16S rRNA gene (Feng et al. 2014; Li et al. 2014). Other analytical methods such as multi-locus sequence typing (MLST) (Rafei et al. 2014), amplified-fragment length polymorphism (AFLP) (Nemec et al. 2010), pulsed-field gel electrophoresis (Rafei et al. 2014), and matrix-assisted laser desorption ionization-time of flight (MALDI-TOF) mass spectrometry (Lee et al. 2015) are often employed for high-resolution taxonomic studies.

Current status of *Acinetobacter* genomes and comparative genomics

As of September 2014, the Integrated Microbial Genome database (IMG; www.img.jgi.doe.gov) contained 924 *Acinetobacter* genomes. *A. baumannii* genomes account for 81.0 % of the total number of *Acinetobacter* genomes (728 genomes). Among non-*A. baumannii* species, 25 strains have clinical origin. As the number of isolates and the sequenced genome indicates, most research efforts have been focused on clinical isolates, especially *A. baumannii* strains, because they are important pathogens with multi-drug resistance. *A. baumannii* is responsible for many severe nosocomial infections, with the highest mortality reported for ventilator-associated pneumonia and bloodstream infections (Dijkshoorn et al. 2007).

The size of *Acinetobacter* genome ranges from 2.67 Mb for *Acinetobacter nectaris* CIP 110549 (2625 CDS) to 6.05 Mb for *A. baumannii* 1288284 (6274 CDS). Presence of plasmids varies by the strain. GC content is from 37 to 46 %. When the number of genomes being considered increases to 38, the number of core genes decreases dramatically to 911 (Chan et al. 2012). The number of core genes and the differences in the genome size and in the number of genes between the smallest and largest *Acinetobacter* genomes imply that *Acinetobacter* strains may have experienced markedly different evolutionary paths, resulting in different physiological characteristics. For example, IMG shows that *Acinetobacter* sp. CIP 102637 (2.67 Mb genome) is auxotrophic in relation to 19 amino acids and coenzyme A, while *A. bereziniae* LMG 1003 (4.96 Mb genome) is auxotrophic in relation to ten amino acids. Those data are awaiting confirmation, but, at minimum, we can expect physiological differences that result from differences in genome sizes.

Differences in physiological characteristics were highlighted in a comparative genomic study of nonpathogenic *Acinetobacter oleivorans* DR1, *A. baylyi*, and *A. calcoaceticus* and pathogenic *A. baumannii* (Jung et al. 2010, 2011a). Although metabolism of many aromatic compounds and the synteny of their catabolic genes are conserved in nonpathogenic and pathogenic *Acinetobacter* strains, only strain DR1 (isolated from rice paddy soil) possesses the ability to grow on gentisate as the sole carbon source. There is a high chance for a natural environment-inhabiting strain to metabolize a natural compound of plant origin (a possible abundant carbon source in a natural environment); therefore, the broad catabolic ability of the strain DR1 may contribute to improved ecological competitiveness.

Insertion sequence (IS) elements seem to be an important factor of genome diversification (Siguier et al. 2014). At an early step in the genome reduction process, the number of IS elements in the genome increases (IS expansion), and the shift of the lifestyle from free living to symbiosis is responsible for the IS expansion (Plague et al. 2008). IS expansion can produce pseudogenes via IS-mediated intrachromosomal recombination and genome reduction (Lawrence et al. 2001). The number of IS elements and their families are remarkably different among *Acinetobacter* species. For example, *A. baumannii* SDF contains 428 IS elements mainly belonging to the IS*982* and IS*5* families, whereas *A. baumannii* AYE strain contains 33 IS elements belonging to IS*Aba1* (Vallenet et al. 2008). A recent study of *A. baylyi* ADP1 showed that IS*3* family IS*1236* is involved in 86 % of amplification events through homologous recombination between an IS and a duplicated region (Cuff et al. 2012). However, the role of IS elements in *Acinetobacter* genome evolution requires more attention, and it possibly contributes to the genome size reduction via recombination and disruption of genes. Another important characteristic contributing to genome diversification is horizontal gene transfer. Because *Acinetobacter* spp. are naturally transformable, horizontal gene transfer is intuitively

expected to occur more often in this genus. Transformation was one of the recognized characteristics in the early *Acinetobacter* studies and is conserved in environmental and pathogenic *Acinetobacter* strains (Park and Park 2011; Traglia et al. 2014). Genes that are required for competence, such as *com* genes, are conserved across environmental and pathogenic strains (Jung et al. 2011a). Horizontal gene transfer is easy to study in *A. baylyi* ADP1. Natural transformation efficiency of *A. baylyi* BD413 ranges from 0.1 to 0.7 % and is considered remarkably high. *Pasteurellaceae* and *Neisseriaceae* were reported to preferentially take up DNAs containing short conserved sequences that are overrepresented in their genomes (Smith et al. 1999). The uptake signal sequence (USS) and DNA uptake sequence (DUS) might perform an important function in the uptake specificity of *Haemophilus influenzae* and *Neisseria gonorrhoeae*, respectively (Danner et al. 1980; Goodman and Scocca 1988). The uptake bias of a USS- or DUS-containing DNA molecule is >100-fold. In the case of *H. influenzae* and *Neisseria meningitidis*, the uptake sequence is present in every 1 kb of their genome. Selective uptake of DNA may result from specific binding of the ComP minor pilus protein to the uptake sequences (Berry et al. 2013; Cehovin et al. 2013). Nevertheless, *Acinetobacter* spp. have no bias with respect to DNA sources and no species specificity (Lorenz et al. 1992; Palmen et al. 1993; Palmen and Hellingwerf 1997). A shift from low- to high-level nutrients seems to trigger horizontal gene transfer in *Acinetobacter*, in contrast to many other naturally competent gram-negative bacteria whose natural competence is triggered by starvation (Seitz and Blokesch 2013). Therefore, DNA uptake in *Acinetobacter* could be a source of adaption or evolution, thereby generating useful genetic and physiological characteristics in the habitat, in relation to nutritional sources. A complete collection of single-gene deletion mutants of *A. baylyi* ADP1 will help to identify genetic factors related to any research topic on *Acinetobacter* species (de Berardinis et al. 2008).

Antibiotic resistance of *Acinetobacter* species

Antibiotic resistance of *Acinetobacter* was investigated mostly in pathogenic strains of *A. baumannii* because multi-drug resistance is a serious obstacle for the treatment of *A. baumannii* infections. The *A. baumannii* strains known before the 1970s do not seem to have had multi-drug resistance; aminoglycosides, β-lactams, and tetracyclines were routinely used for the treatment of *A. baumannii* infections (Bergogne-Bérézin and Towner 1996). However, a recent study showed that up to 30 % of *A. baumannii* isolates from intensive care units are multi-drug resistant to three classes of antibiotics (Lockhart et al. 2007). Colistin and carbapenem are currently the best option for multi-drug resistant *A. baumannii*; however, resistance to these antibiotics was recently reported, and the advent of pan-drug resistance became a very plausible scenario (Cai et al. 2012). To overcome this multi-drug resistance problem, there have been trials to increase efficacy of antibiotics by combining the use of two or more antibiotics (Dong et al. 2014; Garnacho-Montero et al. 2013; Wojtyczka et al. 2014).

Even though *A. baumannii* isolates were sensitive to many antibiotics in the 1970s, some of the antibiotic resistance determinants seem to have been already present in their genomes. A comparative genomic study showed that *Acinetobacter* strains isolated from a natural environment also possess the *ampC* genes coding for β-lactamase, and one of the *ampC* genes shows high sequence similarity with *ampC* of *A. baumannii* (Jung et al. 2011a). Phylogenetic analysis of β-lactamase from *A. baumannii* strains suggests that β-lactamase of *Acinetobacter* strains is inherited from a common ancestor (Hujer et al. 2005). Intrinsic chromosomal β-lactamase can be categorized into two groups: (1) *ampC*-type cephalosporinase (Mammeri et al. 2003; Segal et al. 2004; Hujer et al. 2005) and (2) OXA-51/OXA-69 β-lactamases (Brown and Amyes 2006; Turton et al. 2006; Vahaboglu et al. 2006). The activity of these enzymes is weak, and hydrolysis of cephalosporins is not observed. However, the expression levels of those genes are greatly increased after insertion of IS*Aba1* upstream of bla_{AmpC} because IS elements provide a strong promoter and result in significant resistance to cefotaxime and ceftazidime (Corvec et al. 2003; Segal et al. 2004; Héritier et al. 2006). Currently, the phylogenetic pattern of intrinsic chromosomal β-lactamase is not well understood.

Whole-genome sequencing and a comparative study of *A. baumannii* strains provided further insight into antibiotic resistance among *Acinetobacter* strains. Genomic data on multi-drug resistance of *A. baumannii* AYE revealed that, putatively, 52 genes are related to antibiotic resistance, whereas in *A. baumannii* SDF (which is sensitive to antibiotics), only seven genes are expected to be resistance determinants (Fournier et al. 2006). A resistance island (RI) conferring antibiotic resistance was identified in many *A. baumannii* strains and was designated as AbaR (it should be noted that the LuxR homolog transcriptional regulator of *A. baumannii* was inopportunely designated *abaR*; it is functions in quorum sensing) (Fournier et al. 2006; Iacono et al. 2008; Vallenet et al. 2008; Post et al. 2010; Liu et al. 2014). Forty-five genes of strain AYE are located closely within an 86-kb AbaR. AbaR has GC content of 52.8 %, which is much higher than the average 38.8 %, implying the possibility of the insertion of RI from an exogenous source. AbaR disrupted the ATPase *comM* gene in *A. baumannii* European clone I and in seven multi-drug-resistant *A. baumannii* strains because the *comM* gene was a target of a transposon (Post et al. 2010; Liu et al. 2014). More detailed analysis showed that the genes within Aba1 originated from *Pseudomonas*, *Salmonella*, and *Escherichia*. Another interesting finding was that putative resistance genes in two strains were not expressed in phenotypes. Therefore,

antibiotic-resistant phenotypes are reflected not only in acquisition of genetic determinants but also in expression or regulation mechanisms and in how environmental cues should be coordinated (Fournier et al. 2006).

If antibiotic resistance is an inherited characteristic, we can ask whether there should be a resistance mechanism for environmental-origin *Acinetobacter* strains, and we could expect the answer to help overcome the problem of multi-drug-resistant strains. In case of soil-borne *A. oleivorans* DR1 exposed to norfloxacin, a nonheritable resistance mechanism (persister formation) was suspected of a major contribution to antibiotic resistance (Kim et al. 2013) because of a 10-fold lower mutation frequency of a target gene (the *gyrA* gene encoding DNA gyrase) and unchanged minimum inhibitory concentration in subculture of the surviving colonies. Transcriptomic analysis also identified upregulation of genes related to the SOS response, phage-related sequences, DNA repair, and iron homeostasis; those might be important for persister formation and antibiotic resistance (Herold et al. 2005; Liu and Imlay 2013). Simultaneously acquired antibiotic resistance may be a trade-off between resistance and biological fitness (Kang and Park 2010a). Strain DR1 acquired heritable antibiotic resistance to rifampicin, and then could not produce quorum-sensing signals, motility, and many other phenotypes. Screening of the transposon mutant library of *A. baylyi* ADP1 revealed that a mutation in genes related to efflux pumps (*acrB* and *oprM*) and peptidoglycan synthesis and modification (*ampD*, *mpl*, and *pbpG*) results in hypersensitivity to β-lactam antibiotics. Mutations in genes responsible for the functions unrelated to the antibiotic mechanisms such as glutathione biosynthesis (*gshA*) can also result in hypersensitivity to metronidazole (Gomez and Neyfakh 2006). A loss of phenotypes and changes in cellular characteristics are considered a cost to survive antibiotic treatment. Even though it is difficult to determine which cells are more competitive in a natural environment, the above result provides a new perspective on the role of antibiotics in microbial ecological characteristics.

Quorum sensing as an important but neglected physiological trait in *Acinetobacter*

Quorum sensing (QS) is a bacterial communication method for recognizing cell population density with signal molecules (Miller and Bassler 2001). The concentration of a signal molecule correlates with the density of signal-producing cells, and when the concentration of a quorum signal reaches the threshold, physiological properties and expression of diverse genes are altered, often resulting in a multi-cellular phenotype of unicellular species (Garg et al. 2014). Acyl homoserine lactone (AHL) is the most well-known signal molecule, and the AHL-mediated QS system typically consists of a LuxI homolog AHL synthase and a LuxR homolog transcriptional regulator. AHL interacts with LuxR, and the AHL-LuxR complex binds to promoter sequences, thereby regulating expression of QS target genes (Egland and Greenberg 2001). This mechanism has been extensively studied in diverse bacterial species including plant-pathogenic species and gram-negative and gram-positive bacteria (Whitehead et al. 2001; Von Bodman et al. 2003; Shank and Kolter 2011), mainly because QS regulates many aspects of gene expression and cellular physiology such as virulence (Hentzer et al. 2003), motility (Weiss et al. 2008), antibiotic production (Duerkop et al. 2009), and biofilm formation (Sarkar and Chakraborty 2008; Waters et al. 2008). *Vibrio fischeri* and *Pseudomonas aeruginosa* have been studied as QS model organisms (Schuster et al. 2013). *V. fischeri* leads a symbiotic lifestyle and maintains a mutualistic relationship with marine animals. Colonization of the light organ at high cell density allows the AHL signal to accumulate; hence, the QS regulon (in this case, luminescence genes) is induced. Therefore, *V. fischeri* is thought to lead two possible lifestyles: the free-living, low-density, sea water-living type or the high-cell-density lifestyle associated with a light organ via the QS system (Engebrecht et al. 1983). In the case of *P. aeruginosa*, more than 300 genes are under QS control (Wagner et al. 2003), and the proteins encoded by the QS regulon are related to the virulence factor. The essential role of QS in infection has been demonstrated in mammalian model organisms (Wu et al. 2001).

We believe that QS is not a field that is aggressively explored in the area of *Acinetobacter* research. This is because a search for literature containing the terms "quorum sensing" and "*Acinetobacter*" in the title and abstract yields only 54 hits in PubMed, whereas as many as 1310 papers have "quorum sensing" and "*Pseudomonas*" in their title and abstract. Nonetheless, QS of *Acinetobacter* is gaining attention at present because the number of publications within the last 5 years is 41, whereas there are only 13 papers since the first article in 2001 (González et al. 2001) until 2009. Unlike *P. aeruginosa* that possesses two paired QS systems (LasR-LasI and RhlR-RhlI) and one orphan QS regulator QscR (Chugani and Greenberg 2014), *A. baumannii* was reported to have one paired QS system: AbaR-AbaI (Niu et al. 2008). Biofilm formation by *A. baumannii* is a QS-regulated physiological feature. The biofilm-forming ability of the *abaI* mutant is reduced by 30–40 %, and exogenous AHL from the parental strain restores this biofilm-forming ability (Irie and Parsek 2008). Because the ability of *A. baumannii* to adhere to and to form a biofilm on biotic and abiotic surfaces supports survival in nosocomial environments and infections, QS of *A. baumannii* urgently needs further investigation.

Genome sequencing data on *Acinetobacter* species indicates the presence of a LuxI-homolog acyl-homoserine lactone (AHL) synthase protein and a LuxR-homolog transcriptional regulator (Vallenet et al. 2008; Kang and Park 2010a; Kim and Park 2013a). Operonic structure of Lux homologs

and genes related to fatty acid biosynthesis indicate that AHL synthesis in *Acinetobacter* strains utilizes fatty acid derivatives to produce an AHL ring and an acyl group (Black and DiRusso 1994; Val and Cronan 1998). AHL molecules synthesized by LuxI-homolog of *Acinetobacter* (AbaI in *A. baumannii* strains) are mainly C6-HSL, C8-HSL, and 3OH-C12-HSL (González et al. 2001; Niu et al. 2008; Kim and Park 2013a; Chan et al. 2014). Interaction between LuxR and AHL is more than that of a receptor protein and a signal molecule. LuxR homologs require their cognate signal molecule for appropriate protein folding and protease resistance (Zhu and Winans 2001; Vannini et al. 2002; Zhang et al. 2002; Costa et al. 2012). The LuxR–LuxI interaction can be disrupted by a small molecule such as indole (Kim and Park 2013b). When the strain DR1 is incubated with indole, the stability and folding of the LuxR homolog, the AqsR protein, decreases, whereas messenger RNA (mRNA) expression of *aqsR* is not changed. Inhibition of the QS system and related phenotypes by indole may be widespread in gram-negative bacteria as shown in *Chromobacterium violaceum*, *Pseudomonas chlororaphis*, and *Serratia marcescens* (Hidalgo-Romano et al. 2014). Their QS-dependent pigmentation does not appear when they are incubated with *E. coli* producing indole. Therefore, indole might be a general inhibitor of QS involving AHL signaling in gram-negative bacteria.

QS regulates expression of many other genes and phenotypes. Proteomic and transcriptional analysis that was conducted in *A. oleivorans* DR1 uncovered changes in mRNA and protein expression related to the type IV pilus system, oxidative stress defense, AHL lactonase, ppGpp synthase, a histidine kinase sensor, *S*-adenosyl methionine (SAM) methyltransferase, and multi-drug resistance efflux (MDF)-, ABC-, and RND-type pumps (Kang and Park 2010a; Kim and Park 2013a). Although the QS regulon in *Acinetobacter* is not well understood, our research provided a consensus sequence of AqsR binding sites (5′-TRTNRRANYTRNYADKW-3′); direct binding to the promoter region of a putative surface adhesion protein and L-asparaginase was confirmed using an electrophoretic mobility shift assay (Kim and Park 2013a). An autoinducer is recognized not only by the producer cell but also by the co-occurring cell without an ability to produce an autoinducer molecule; therefore, a QS signal plays an important role in the interaction between two or more bacterial species. The presence of many microorganisms that degrade autoinducers such as AHL (quorum quenching) makes it difficult to understand the ecology of a microbial community (Ochiai et al. 2013). For example, interaction between two pathogenic bacteria, *P. aeruginosa* and *A. baumannii*, via QS and biofilm formation was found to be possibly affecting the severity of coinfection (Bhargava et al. 2012). Coexistence of the strain DR1 with quorum-quenching *Pseudomonas* sp. AS1 is even more complex due to metabolic commensalism and biofilm formation of the 2 strains (Seo et al. 2012).

The motile phenotype of "a non-motile rod"

The name "*Acinetobacter*" means "a non-motile rod" and was coined because an early taxonomic study suggested that a non-motile phenotype was a common characteristic in this genus. In that tradition, many papers start with a description of *Acinetobacter* as a non-motile cell. However, twitching motility was already reported in 1975 by Henrichsen and Blom (1975). Motility of *A. calcoaceticus* strains according to their work is very conditional and is observed in a small portion of tested strains (Henrichsen 1975a; Henrichsen and Blom 1975). A common physiological characteristic of twitching strains is relatively thick (50 Å) polar fimbriae, whereas non-motile strains possess relatively thin (30 Å) peritrichous fimbriae (Henrichsen 1975b). The fimbriae phenotype could be growth-phase dependent because polar fimbriae are only observed during the exponential growth phase, and peritrichously arranged fimbriae are seen in the late growth phases (Henrichsen and Blom 1975). Surface swarming motility was also reported by Barker and Maxted (Barker and Maxted 1975). Some of the strains produce channels (ditches) in the agar plate. Later studies reported gliding motility of *A. anitratus* and twitching motility of *A. calcoaceticus* without gliding (Mukerji and Bhopale 1983; Henrichsen 1984). Several recent studies have begun to describe motile phenotypes in *Acinetobacter*. Kang and Park (2010b) surveyed swimming and swarming motility of 17 *Acinetobacter* species and found branched-type propagates from strain DR1, *A. baylyi* and *A. gerneri* on semi-solid agar. Twitching motility of *A. baumannii* was reported in many other studies (Bitrian et al. 2013; Harding et al. 2013; Wilharm et al. 2013; Heindorf et al. 2014; Hidalgo-Romano et al. 2014; Jacobs et al. 2014b; Nait Chabane et al. 2014; Withers et al. 2014). We found that articles about the motility of *Acinetobacter* had not been published between the 1970 and the 2010 (see the publication years of the citations in this section). The number of such publications is increasing, but the understanding of this phenomenon is still nascent. Therefore, we concluded that *Acinetobacter* is not in fact "a non-motile rod" and the motile phenotype is also a relatively unexplored research topic in *Acinetobacter*. We can speculate that the discrepancies regarding motile phenotypes among various studies are due to the differences in strains, nonstandardized assay methods, poor reproducibility of motility assays, and the types of motility each research project focused on.

As the number of available *Acinetobacter* genomes grew, the genetic basis for the motile phenotype became evident because of the presence of motility-related genes. The structural basis of *Acinetobacter* motility is related to extracellular appendages such as pili. The *pilA*, *pilD*, and *pilT* mutant strains of *A. baumannii* do not show twitching motility (Harding et al. 2013). The *acu* gene cluster of *A. baylyi* shows that thin pili are assembled via the chaperone/usher pathway (Gohl

et al. 2006). Motility of *Acinetobacter* is a complex phenotype interwoven with other important physiological characteristics, cellular stressors such as oxidative stress, or the type of pili (Gohl et al. 2006; Heindorf et al. 2014). Association of motile phenotype with other physiology was also reported from *P. aeruginosa*. Swarming cells of *P. aeruginosa* show stronger resistance to multiple antibiotics (Overhage et al. 2008), and the expression of genes related to the virulence factor is up-regulated (Lai et al. 2009). Although the relationship between motility and pathogenicity in *Acinetobacter baumannii* is unclear, Mattick brought up the possibility of the contribution of type IV pilus-twitching motility to virulence because type IV pili affect bacterial virulence (Mattick 2002).

Natural transformation is closely related to the motile phenotype (Harding et al. 2013; Wilharm et al. 2013). For co-occurrence of motility and natural transformation, both structure and function of pili seem to be essential because the *pilT* mutant strain of *A. baumannii* M2 shows an increase in the number and length of pili while the strain lacks motility and the DNA uptake ability. In terms of regulation of motility, the transcription factor AtfA, a ribonuclease T2 family protein, and the sensor kinase GacS are known to regulate gene expression related to other diverse phenotypes such as biofilm formation, sensitivity to antibiotics, sensitivity to ethanol, and motility of *A. baumannii* ATCC 17978 (Cerqueira et al. 2014; Withers et al. 2014). It is also worth noting that blue light can affect the motile phenotype, and activity of BLUF domain-containing proteins was suggested in *A. baylyi* ADP1 (Mussi et al. 2010; Bitrian et al. 2013). Regulation of motility in *Acinetobacter* is poorly characterized, and only a few studies have been published (Cerqueira et al. 2014; Withers et al. 2014), suggesting that *Acinetobacter* motility has not received much attention; therefore, many aspects of motility are waiting to be characterized.

Aromatic compounds and hydrocarbon catabolism

Since the early days of taxonomic studies, utilization of aromatic compounds has been a common characteristic of *Acinetobacter*. Growth of 106 strains on 15 aromatic compounds as the sole carbon source was confirmed (Baumann et al. 1968). Strains grown on phenyl acetate use the gentisate pathway, but there has not been a follow-up study explaining this pathway before our group's report (Jung et al. 2011a). The ability to degrade aromatic compounds is also active in a natural environment where diverse microorganisms interact (Simarro et al. 2013). Well-known aromatic-compound degraders include *Pseudomonas*, *Sphingomonas*, *Ralstonia*, and many other genera (Lee and Lee 2001; Coronado et al. 2012; Arora et al. 2014). Their strains are often capable of degrading recently synthesized anthropogenic compounds; however, the list of aromatic compounds that are utilized by *Acinetobacter* strains was found to contain only natural products originating from plants (Parke and Ornston 2004; Young et al. 2005). Enrichment culture with an exotic compound rarely shows the capacity of *Acinetobacter* for degradation of those compounds. This lifestyle may be indicative of the important role of *Acinetobacter* in nutrient cycling in natural environments.

Whole-genome sequencing of *A. baylyi* revealed that the catabolic genes for aromatic compounds are concentrated in five genomic loci (Barbe et al. 2004), whereas other aromatic-compound degraders such as *Sphingomonas* strains harbor the genes necessary for degradation pathways in a scattered arrangement within their genomes (Pinyakong et al. 2003). Syntenic localization of genes associated with a metabolic pathway might relieve the energy burden in terms of the transcriptional and translational machinery, although it has never been clearly elucidated. Catabolism of many aromatic compounds yields the intermediate metabolites catechol and protocatechuate, feeding the β-ketoadipate pathway. Genome sequencing data and comparative genomic studies showed that both environmental-origin strains (*A. calcoaceticus*, *A. baylyi*, and *A. oleivorans* DR1) and pathogenic strains (*A. baumannii*) have catabolic pathways for diverse aromatic compounds, along with the β-ketoadipate pathway, with almost identical syntenic arrangement (Jung et al. 2011a). Therefore, utilization of aromatic compounds is believed to be conserved among *Acinetobacter* strains.

Most gene clusters for aromatic-compound catabolism contain a transcriptional regulator (usually LysR type) such as *catM* for the catechol branch of the β-ketoadipate pathway (Romero-Arroyo et al. 1995) and *benM* for benzoate degradation (Collier et al. 1998). Two branches of the β-ketoadipate pathway are under cross-regulation, resulting in dominance of the catechol branch over the protocatechuate branch (Brzostowicz et al. 2003; Siehler et al. 2007). However, more globally, aromatic-compound degradation is under carbon catabolite repression (Dal et al. 2002; Fischer et al. 2008). Although many enterobacteria and gram-positive bacteria such as *Bacillus* spp. prefer a sugar molecule in their carbon catabolite repression mechanism, organic acids are more important carbon sources for carbon catabolite repression in *Acinetobacter* strains (Fujita 2009). This phenomenon may be related to the fact that few *Acinetobacter* strains are capable of utilizing simple sugars such as glucose. In the strain ADP1, succinate and acetate repress gene expression of the β-ketoadipate pathway and 8 different gene clusters for aromatic compound catabolism (e.g., *catBCIJFD*, *van*, *sal*, *are*, *ant*, *ben*, *hca*, and *dca* for *cis*, *cis*-muconate, vanillate, salicylate, benzyl esters, anthranilate, benzoate, hydroxycinnamate, and dicarboxylate metabolism, respectively). Because succinate and acetate are the products of the β-ketoadipate pathway, carbon catabolite repression is considered a negative feedback mechanism of the metabolic pathway. Pyruvate and lactate do not cause carbon catabolite repression. It was speculated that

pyruvate and lactate might not have been an abundant carbon source where *A. baylyi* ADP1 evolved; therefore, the corresponding mechanism had not developed during evolution. Carbon catabolite repression in ADP1 is controlled by Crc via a posttranscriptional mechanism (Zimmermann et al. 2009). In case of *P.aeruginosa*, the Crc protein does not exhibit the nuclease or DNA-binding activity (MacGregor et al. 1991, 1996). Crc of *Pseudomonas putida* is an RNA-binding protein, and its interaction with transcriptional regulators AlkS and BenR has been reported (Moreno et al. 2007; Moreno and Rojo 2008), which underlies translation interruption. In contrast to *Pseudomonas* species, *A. baylyi* Crc controls aromatic-compound catabolism by changing RNA stability of *pca-qui* mRNA, as shown by the increased mRNA half-life in a *crc*-mutant strain (Zimmermann et al. 2009).

Acinetobacter is also a famous hydrocarbon degrader, especially with respect to alkanes of diverse chain lengths. *Acinetobacter* is frequently found in diverse hydrocarbon-contaminated sites, including soils, mangrove sediments, Antarctic marine sediments, and pristine environments, showing the potential for alkane biodegradation (Kuhn et al. 2009; Kang et al. 2011; Rocha et al. 2013). Regulation of the alkane degradation pathway has not been elucidated, and we could not find good literature on this topic. The general lack of interest in the regulation of alkane metabolism may be due to the simplicity of the mechanism of alkane degradation where hydroxylation is followed by β-oxidation. However, a detailed description of alkane metabolism is far from clear. For example, one of the mysterious steps in alkane degradation is transportation of alkane to the cytoplasm for subsequent catabolism. An important physiological characteristic for alkane degradation seems to be the ability to adhere to an oil droplet and to form a biofilm (Kang et al. 2008a, b; Jung et al. 2011b). When *A. oleivorans* DR1 is cultured in biofilm-unfavorable conditions, hexadecane degradation is also hindered. Genome sequencing data on the hydrocarbonoclastic marine bacterium *Alcanivorax borkumensis* indicate that biofilm formation at the oil–water interface is an important physiological parameter (Schneiker et al. 2006). Other phenotypes related to hydrocarbon degradation were explored in *A. borkumensis*. A transposon mutagenesis study of this microorganism showed that UV exposure, temperature changes, and high salinity are the environmental factors affecting hydrocarbon degradation and oxidation, and cyclic-di-GMP performs a function in signal transduction in response to environmental stressors (Sabirova et al. 2008). Environmental factors, substrate sensing, signaling, and many other genetic factors in *Acinetobacter* spp. need further research for a clearer understanding of hydrocarbon degradation.

Alkane catabolism in *A. baylyi* ADP1 consists of three components: alkane monooxygenase, rubredoxin, and rubredoxin reductase encoded by *alkM*, *rubA*, and *rubB*, respectively; each component is essential to alkane metabolism (Ratajczak et al. 1998). Alkane degradation is expected to cause stress because it is a series of oxidation procedures such alkane oxidation followed by β-oxidation; a contact with a hydrocarbon will possibly disrupt cell membrane structure. Proteomic research performed on alkane-utilizing cells of *A. oleivorans* DR1 showed significant upregulation of oxidative stress defense proteins along with a glyoxylate bypass, fatty acid metabolism, and gluconeogenesis (Jung et al. 2011b). In this context, it is worth mentioning that *oxyR* (a LysR-type global transcriptional regulator that is related to oxidative stress) is adjacent to *rubB*. The *rubA-rubB-estB-oxyR* operon shows polycistronic expression driven by the σ^{70} promoter (Geissdörfer et al. 1999).

Aside from alkane monooxygenase, cytochrome P450 also participates in alkane metabolism (Funhoff et al. 2006). *Acinetobacter* sp. strain DSM 17874 can utilize alkanes with chain length from C10 to C40 and has two copies of alkane monooxygenase. However, in the catabolism of long-chain alkanes, a putative flavin-binding monooxygenase, *almA*, is required (Throne-Holst et al. 2007). After the first description of *almA* in *Acinetobacter* spp., the presence of an *almA* homolog was identified in *Alcanivorax dieselolei* B-5, *Alcanivorax hongdengensis* A-11-3, and many other genera such as *Salinisphaera*, *Parvibaculum*, and *Marinobacter* (Wang and Shao 2012).

The biotechnological perspective

A variety of catabolic genes and their regulatory systems could have been used for constructing a bioreporter. For example, the phenol-inducible *mphK* promoter from *A. calcoaceticus* PHEA-2 was fused to the β-galactosidase gene (*lacZ*) and the transcriptional regulator *mopR* of *A. calcoaceticus* NCIB 8250 to produce an *E. coli*-based bioreporter (Peng et al. 2010; Qu et al. 2010; Zhang et al. 2012; Hošková et al. 2013). However, the more promising use of *Acinetobacter* spp. as bioreporters includes the whole-cell use, mainly because these species have physiological characteristics that are different from those of a traditional bioreporter host such as *E. coli*, with respect to growth and survival. These properties allow for the use of ADP1 to search for and detect oil spills in water and soil environments (Zhang et al. 2012). Aside from contaminants, one of our studies showed that TetR repressor-based bioreporters (*E. coli* and *A. oleivorans* DR1) detect doxycycline (Hong and Park 2014). Tetracycline and doxycycline are difficult to detect in terms of sensitivity and methodological feasibility (Yang et al. 2004), in spite of their extensive use and prevalence in natural environments (Miao et al. 2004); therefore, the use of a whole-cell bioreporter for tetracycline and doxycycline seems to be a simple way to quantitatively assess their presence in a natural environment.

Acinetobacter spp. are good sources of lipase. Many *Acinetobacter* strains isolated from diverse sources were

found to be lipolytic strains (Blaise and Armstrong 1973; Kaplan and Rosenberg 1982; Snellman and Colwell 2004). Biochemical properties of *Acinetobacter* strains are well characterized. Their usual optimal culture conditions are pH 7–9 and 30–55 °C (Kok et al. 1995; Han et al. 2003). To utilize a lipase in a broader range of pH and temperatures, screening efforts for isolation have been continuing in diverse environments; alkaline lipase from *A. calcoaceticus* 1-7, a thermotolerant lipase from an unnamed *Acinetobacter* strain, and a cold-adapted lipase were successfully characterized (Khoramnia et al. 2011; Zheng et al. 2011; Wang et al. 2012). Wang et al. (2012) showed the possibility of enzymatic engineering using genome shuffling to improve production of a low-temperature alkalophilic lipase in *A. johnsonii*. The *Acinetobacter* lipase activity can be stabilized or increased by the presence of Ca^{2+} due to the presence of a Ca^{2+}-binding pocket, leading to correct active-site configuration (Lang et al. 1996). Lipases are categorized based on sequence similarity, and subfamilies I.1 and I.2 are encoded in an operon with their cognate Lif chaperone (Arpigny and Jaeger 1999).

In addition to the use of *Acinetobacter* spp. as bioreporters and lipase producers, *Acinetobacter*-derived biosurfactant production (e.g., emulsan) has important practical applications such as production of biopolymers (Gross et al. 2001), biodiesel (Noureddini et al. 2005), therapeutics (Ono et al. 2001), and of cosmetics (Kiyota et al. 2001; Satpute et al. 2010). Production of a biosurfactant has been reported for many *Acinetobacter* strains such as *Acinetobacter* sp. D3-2 (Bao et al. 2014). Characterization and production of a biosurfactant (emulsan) was best studied in *Acinetobacter venetianus*. The genome of *A. venetianus* RAG-1 was sequenced (Fondi et al. 2012), and bioinformatics analysis of the *A. venetianus* VE-C3 genome was also conducted (Fondi et al. 2013). Because emulsan contains hydrophobic side chains consisting of fatty acids, its production is often enhanced when a fatty acid or alkane is provided as a carbon source. The *wee*, *wzc*, and *wzy* gene clusters are responsible for the emulsan production (Dams-Kozlowska et al. 2008). The *wee* gene cluster is separated by two σ^{70} promoters. Interference with this cluster by a transposon results in translucent colony morphology and a decrease in emulsan production (Nakar and Gutnick 2003). Detailed metabolic pathways and genetic mechanisms underlying emulsan production were reviewed by Dams-Kozlowska et al. (2008).

Concluding remarks

Acinetobacter strains have been proposed as model organisms for clinical, environmental, and industrial studies (de Berardinis et al. 2009; Elliott and Neidle 2011; Jacobs et al. 2014a). As described in this review, important achievements of studies on *Acinetobacter* spp. include the following: aromatic compound degradation, natural transformation, production of chemicals, and antibiotic resistance and pathogenicity of *A. baumannii*. Other research topics such as quorum sensing, motility, biofilms, and stress response and resistance are expected to bloom soon. Moreover, comparative genomic studies are not covering the increasing number of available genomes; consequently, the genomic data have not been systematically organized and analyzed for subsequent research. Although considerable further research is needed to understand physiology, genetics, and ecological functions of *Acinetobacter* spp., the efforts currently under way and a steady stream of fascinating findings are expected to produce a clearer picture of *Acinetobacter* biology.

Acknowledgments This work was supported by the Mid-career Researcher Program through an NRF grant (2014R1A2A2A05007010) funded by the Ministry of Science, ICT, and Future Planning (MSIP).

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