行政院國家科學委員會專題研究計畫
成果報告

臺灣 Ⓡ IRC '},

抗藥性金黃色葡萄球菌抗藥性基因之分型
研究 Ⓡ IRC

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中文摘要

抗藥性金黃色葡萄球菌（MRSA）自1990年代以來，已成為台灣地區的重要致病菌之一。其抗藥性的機轉在於攜帶了一段抗藥性基因──葡萄球菌卡匣染色體（Staphylococcal cassette chromosome，簡稱SCCmec元住）。根據最近的研究，SCCmec元素可分為四種型式：type I、II、III、IV。而截至目前為止，台灣地區並沒有大規模針對本土的MRSA之SCCmec元素進行的分型研究。在我們先前利用脈衝式磁場電泳（pulsed-field gel electrophoresis，PFGE）對全台灣院內感染的MRSA菌株的分子分型研究中，我們發現了全台灣大部分的院內MRSA菌株都是屬於PFGE type C，並且全台灣有七個比較重要的PFGE types。因此，我們由此七個PFGE type中（type B、C、D、G、H、J、L）的每一個亞型（共計31個亞型）挑出一個MRSA菌株，利用聚合酶鍵鎖反應（polymerase chain reaction）的方法來進行SCCmec元素的分型研究。另外，我們也收集2003年1月至2003年12月台大醫院所有符合社區性MRSA感染定義患者的MRSA菌株（CA-MRSA），共計19株，進行其SCCmec元素的分型研究。研究結果顯示，在台灣七種重要的院內MRSA strains中，PFGE type B、C、J、L的菌株均攜帶著第三型的SCCmec元素；而PFGE type D、G、H的菌株則攜帶著第四型的SCCmec元素。至於19株的CA-MRSA中，有5株屬於PFGE type C，有8株屬於type D，有4株屬於type H，而有兩株屬於type S。這19株CA-MRSA中，除了屬於type C的5株攜帶著第三型的SCCmec元素外，其餘均攜帶著第四型的SCCmec元素。根據上述的研究結果，我們歸結了以下幾個重點：全台灣最主要的院內MRSA strain，攜帶著第三型的SCCmec元素，而全台灣絕大部分的院內MRSA菌株不是攜帶著第三型的SCCmec元素就是第四型的SCCmec元素；台灣地區的院內MRSA菌株和CA-MRSA可能已發生交叉傳播的現象；台灣地區的CA-MRSA大部分均攜帶著第四型的SCCmec元素；在所謂的CA-MRSA中，也存在著一個主要的MRSA strain。

關鍵詞：抗藥性金黃色葡萄球菌；抗藥性基因；葡萄球菌卡匣染色體
Methicillin-resistant *Staphylococcus aureus* (MRSA) had caused a great impact on clinical medicine since mid-1990s in Taiwan. The mechanism of resistance to methicillin in MRSA was due to the acquisition of additional genetics called staphylococcal cassette chromosome (SCCmec element). According to the recent studies, the SCCmec elements could be classified into four types: type I, II, III, and IV. Till now, there is still no published report about the detailed genetics of methicillin resistance, the typing of SCCmec element, of MRSA isolates in Taiwan. In our previous study, we found that there was a predominant MRSA strain, pulsed-field gel electrophoresis (PFGE) type C, among all the nosocomial MRSA isolates in Taiwan. And the majority of nosocomial MRSA isolates belonged to seven PFGE types, type B (3 subtypes), C (10 subtypes), D (2 subtypes), G (5 subtypes), H (5 subtypes), J (4 subtypes), and L (2 subtypes). Therefore, we selected one MRSA isolate from each PFGE subtypes, 31 isolates in total, to undergo the typing of their SCCmec elements. In addition, 19 community-acquired MRSA isolates collected from January 2003 to December 2003 at National Taiwan University Hospital were also included in this study. The study result demonstrated that among the nosocomial MRSA isolates, those belonging to PFGE type B, C, J, and L carried type III SCCmec element and those belonging to PFGE type D, G, and H carried type IV SCCmec element. Among the 19 CA-MRSA isolates, all except the 5 isolates belonging to PFGE type C carried type IV SCCmec element. In conclusion, our study found that the predominant nosocomial MRSA strain, PFGE type C, carried type III SCCmec element, the majority of nosocomial MRSA isolates carried either type III or IV SCCmec element, the nosocomial MRSA isolates and CA-MRSA might had been cross-transmitted each other, most CA-MRSA carried type IV SCCmec element, and there was also a predominant strain among the CA-MRSA.

Key words: methicillin-resistant *Staphylococcus aureus*, MRSA, resistant gene, staphylococcal cassette chromosome
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III
The first report of MRSA in the world is by Dr. Jevons in 1961 (1). And the first isolate of MRSA in Taiwan is found in 1981 (2). Thereafter, the rates of nosocomial MRSA infections increased rapidly in Taiwan and most hospitals in Taiwan now have a high incidence of nosocomial MRSA infections (3, 4). The rates of MRSA over all nosocomial isolates of S. aureus in some Taiwan hospitals has already exceeded 80% in 1998 (5), which was much higher than that reported by the National Nosocomial Infection Surveillance System (NNIS) (6). Our previous studies have proved that the reasons leading to rapid increased of MRSA infection in Taiwan include overuse of antibiotics, poor adherence to isolation precaution of health care worker, and introduction of endemic strain (7-10). MRSA thus has become a major pathogen in Taiwan. In addition, because of its resistance, limited choice of drug to treat MRSA infection is another important clinical problem. However, there is still no detailed study on the genetic mechanism of methicillin resistance of MRSA in Taiwan. Understanding the detailed genetics of methicillin resistance of MRSA may be helpful to overcome this resistance in the future.

The genetic coding for methicillin resistance in S. aureus has been proven to be mecA gene (11). The expression of mecA gene results in a specific penicillin-binding protein, PBP2’, that has a decreased binding affinity to β-lactam antibiotics and thus leading to methicillin resistance. The expression of mecA gene is regulated by two adjacent regulatory gene, mecl and mecR1. The mecA, mecl, and mecR1 genes consist of the mecA gene complex (12). The mecA gene complex is widely distributed among S. aureus species as well as among other staphylococcal species collectively called coagulase-negative staphylococci (13-15). Therefore, it has been speculated that mec may be freely transmissible among staphylococcal species. In the 1980s, direct chromosome analysis of MRSA strains revealed that a substantial length of the chromosomal DNA segment (greater than 30 kb) carrying mec has no allelic equivalence in meticillin-susceptible S. aureus (MSSA) strains; the segment was called “additional DNA” or “mec DNA” (16, 17). The size, structure, and biological properties of mec DNA had long remained unclear (18).

The most striking findings in recent studies on MRSA are the existence of SCCmec gene element (19). According to the studies of Hiramatsu et al, the “mec DNA” is now formally renamed as “SCCmec (Staphylococcal cassette chromosome mec) element” and the SCCmec element is almost universally found in all MRSA isolates (20). The most important components in SCCmec element are the ccr
(cassette chromosome recombinase), \textit{ccrA} and \textit{ccrB}, genes and \textit{mecA} gene complex (19, 20). The \textit{mecA} gene complex can be classified into four types, type A, B, C, D. The \textit{ccr} genes can be classified into three types. The function of \textit{ccr} genes is to precisely excise and insert the SCC\textit{mec} element in way of both site and orientation specifically (19). Based on the structures and combinations of \textit{ccr} genes and \textit{mecA} gene complex, the SCC\textit{mec} gene element in MRSA can be classified into four types (20, 21). Type III SCC\textit{mec} gene carries more drug-resistant determinants than any other types. According to the study, conducted by Ito et al, on the analysis of SCC\textit{mec} element of 38 major hospital-acquired MRSA strains isolated worldwide, SCC\textit{mec} elements of MRSA isolates from Europe belong to type I and III, those from northern America belong to type II, most of those from Japan belong to type II, those from Australia and southeastern Asia belong to type III, and those from south Africa belong to type I (20). Type IV SCC\textit{mec} element is so far only found in community-acquired MRSA isolates (21).

As mentioned above, there is still no published report about the detailed genetics of methicillin resistance of MRSA isolates in Taiwan. The type of SCC\textit{mec} elements of MRSA isolated in Taiwan is also obscure. Our study is designed to illuminate the \textit{mecA} complex (in the first year), and \textit{ccr} genes (in the second year) of MRSA isolates in Taiwan as well as to determine the types of SCC\textit{mec} elements in Taiwan and compare these results with MRSA isolates in other country. In addition, there are some sporadic cases of community-acquired MRSA infections. By typing the SCC\textit{mec} gene element of MRSA isolates isolated from patients with nosocomial MRSA infections and those with community-acquired MRSA infections, whether the methicillin resistance comes from the same source between these isolates or not can also be determined.
 Definitions and MRSA isolates:

Patients with community-acquired MRSA (CA-MRSA) septicemia is defined as that patients had no history of hospitalization within prior 30 days develop signs and symptoms of sepsis before admission and their blood cultures taken within 48 hours after hospitalization yielded MRSA. Patients with nosocomial MRSA septicemia is defined as that patients developed signs and symptoms of sepsis three more days after admission and their blood cultures yielded MRSA. Based on our previous study, there were seven major types of nosocomial MRSA isolates determined by pulsed-field gel electrophoresis (PFGE) in Taiwan, including type B (3 subtypes), C (10 subtypes), D (2 subtypes), G (5 subtypes), H (5 subtypes), J (4 subtypes), and L (2 subtypes) (10). Thirty-one isolates, one isolate from every subtype, were selected for further microbiologic investigation. Nineteen CA-MRSA isolates collected from January 2003 to December 2003 at NTUH were also enrolled.

Determination of minimum inhibitory concentration (MIC):

All isolates were tested for their MIC levels of oxacillin, gentamicin, clindamycin, erythromycin, ofloxacin, levofloxacin, tetracycline, rifampin, trimethoprim/sulfamethoxazole, vancomycin, and linezolide using agar dilution method proposed by NCCLS (22).

PFGE:

All isolates were typed first by PFGE to determine whether those CA-MRSA belonged to the same molecular types of nosocomial isolates or not. The methods used for undergoing PFGE will be as those described in our previous study (9). The interpretation of PFGE patterns was according to the principals proposed previously (23, 24). Once a CA-MRSA is proved to belong to the same type as a nosocomial isolate, it was not used for further molecular study. All results will be double checked.

PCR and nucleotide sequencing for the analysis of mecA complex:

The chromosomal DNA will be prepared by means of the method described by Hiramatsu et al and Matsuhashi et al (25, 26). PCR amplification was performed using 1 unit AmpliTaq (Perkin-Elmer Cetus, Foster City, Calif.) in 50 μl of reaction mixture (10mM Tris-HCl [pH 8.3], 50 mM KCl, 0.001% [wt/vol] gelatin, 50% [vol/vol] glycerol, 1.5 mM MgCl₂, 200 mM each
deoxynucleoside triphosphatase, 1.0 mM each primer, and template DNA). The reaction was carried out by using a Gene Amp PCR system 9600 (Perkin-Elmer). Thermal cycling was set at 30 cycles (30 s for denaturation at 94°C, 1 min for annealing at 50°C, and 2 min for elongation at 72°C).

Long-range PCR amplification was performed using 2.6 U of Expand high-fidelity PCR system enzyme mix as recommended by the manufacturer (Boehringer Mannheim Biochemica, Mannheim, Germany). A 5-μl portion of the reaction volume was subjected to electrophoresis in a 0.8% agarose gel containing 1 μl of ethidium bromide per ml to detect the amplified DNA fragment. All PCR products were further sequenced using a 377 automated fluorescent DNA sequencing system (Perkin-Elmer, Foster City, Calif.) to compare the nucleotide homology with the published sequence in GenBank. The PCR and sequencing results were double-checked.

**PCR primers and detection of mecA gene complex:**

The primers used for the detection of mecA gene complex include:
- mA2 (5’-AACGTTGTAACCAACCCAAGA-3’),
- mA4 (5’-AGTGTATGATGAGCTATGA-3’),
- mA5 (5’- CGCTCAGGAATTTGTTGTGC-3’),
- mA6 (5’-TATACCAACCCGACAA-3’),
- iS1 (5’-ACATTAGATATTTGGTTGCGT-3’),
- iS3 (5’-TCGGATGCTATTACATTAAGCAT-3’),
- iS4 (5’-ACAATCTGTATTCTCAGGTCGT-3’),
- mI-1 (5’-AATGCGAAAAAGCACAACA-3’),
- mI-2 (5’-GACTTGATTGTTTCCTCTGTT-3’),
- mcR2 (5’-CGCTCAGAAATTTGTTGTGC-3’), and
- mcR3 (5’-ATACTCCACGTTAATTCCATT-3’)

Technical detection of the class A mecA gene complex was based on the positive PCR test results for both sets of primers, mI-1 plus mI-2 and mcR2 plus mcR3. The class B mecA gene complex was detected by long-range PCR using two sets of primers, iS-3 plus mA5 and iS-4 plus mA5. Detection of the Class C mecA gene complex was based on the negative PCR test results for two sets of primer, mI-1 plus mI-2 and mcR2 plus mcR3, as well as a positive long-range PCR test result for one set of primers, iS-1 plus mA6. Detection of the Class D mecA gene complex was based on the negative PCR test results for two sets of primer, mI-1 plus mI-2 and mcR2 plus mcR3, as well as a negative long-range PCR test result for one set of primers, iS-1 plus mA6 (27).
PCR and nucleotide sequencing for the analysis of ccr genes:
The PCR and long-range PCR methods were used to analyze ccr genes as described above. All PCR products were further sequenced using a 377 automated fluorescent DNA sequencing system (Perkin-Elmer, Foster City, Calif.) to compare the nucleotide homology with the published sequence in GenBank. The PCR and sequencing results were double-checked.

PCR primers and detection of ccr genes:
The primers used for the detection of ccr gene complex include:
\[ \beta_2 \ (5'-ATTGCCTTGATAATAGCCITCT-3') \],
\[ \alpha_2 \ (5'-AACCTATATCATCAATCAGTACGT-3') \],
\[ \alpha_3 \ (5'- TAAAGGCATCAATGCACAAACACT-3') \],
\[ \alpha_4 \ (5'-AGCTCAAAAAGCAAGCAATAGAAT-3') \]
Technical detection of type 1 ccr complex was based on the positive long-range PCR test result for one set of primers, \( \alpha_2 \) plus \( \beta_2 \). Technical detection of type 2 ccr complex was based on the positive long-range PCR test result for one set of primers, \( \alpha_3 \) plus \( \beta_2 \). Technical detection of type 3 ccr complex was based on the positive long-range PCR test result for one set of primers, \( \alpha_4 \) plus \( \beta_2 \) (10, 27).

Determining the types of SCCmec elements:
After determining the types of \( mecA \) gene complex and ccr complex, the types of SCCmec elements was further determined using following criteria:
1. Type I SCCmec element: type 1 ccr complex + class B \( mecA \) gene complex;
2. Type II SCCmec element: type 2 ccr complex + class A \( mecA \) gene complex;
3. Type III SCCmec element: type 3 ccr complex + class A \( mecA \) gene complex;
4. Type IV SCCmec element: type 2 ccr complex + class B \( mecA \) gene complex;
Drug susceptibility determined by MIC and PFGE types:

The drug susceptibility of the 50 MRSA isolates was listed in Table 1 and 2. Among the nosocomial MRSA isolates, both isolates of PFGE type L were only susceptible to vancomycin and linezolid but resistant to all other tested antibiotics. Most isolates of PFGE type C were only susceptible to vancomycin, rifampin, and linezolid. The MRSA isolates of other PFGE types were more susceptible to fluoroquinolones, trimethoprim/sulfamethoxazole, as well as gentamicin and were fully susceptible to vancomycin, rifampin, as well as linezolid. The 19 CA-MRSA belonged to 4 PFGE types, C (5 isolates), D (8 isolate), H (4 isolates), and S (2 isolates). Except the 5 isolates of PFGE type C, nearly all other isolates were susceptible to gentamicin, fluoroquinolones, and trimethoprim/sulfamethoxazole with full susceptibility to vancomycin, rifampin, and linezolid. The PFGE patterns were demonstrated in Figure.

Types of mecA cluster, ccr genes, and SCCmec element:

Our study demonstrated the predominant nosocomial MRSA strain (PFGE type C), which had spread all over Taiwan (10), carried type III SCC\textit{mec} element and the majority of MRSA isolates isolated in Taiwan carried either type III or type IV SCC\textit{mec} element. This is a very interesting finding.

Recently, type IV elements were considered to be a marker of CA-MRSA (21). In our present study, 14 out 19 CA-MRSA carried type IV SCC\textit{mec} element. For the 5 isolates belonging to PFGE type C and classified as CA-MRSA, there were two possibilities. First, the nosocomial strain had been transmitted to community environment. Second, they were just mis-classified and were nosocomial MRSA in fact because that we did not clarify the clinical history in details (patients might have been admitted to another hospital for a period of time before they were admitted to NTUH). In addition, MRSA of PFGE type D accounted for 42.1% of all CA-MRSA. This implied that there was also a predominant MRSA strain, PFGE type C, spread in the community environment in Taiwan.

Among the nosocomial MRSA isolates, there were some isolates (belonging to PFGE type D, G, and H) also carried type IV SCC\textit{mec} element. This might be due to the transmission and spread of CA-MRSA into hospital environment. However, it was also likely that, in fact, MRSA isolates carried type IV SCC\textit{mec} element were originated from their ancestor \textit{S. aureus} isolates which got element and became MRSA in the hospital environment and then transmitted/spread into the community environment. This inference had also been proposed by other researchers (29). However, there was still no solid evidence to verify this hypothesis.

Previous studies had point out that MRSA isolates carring type III SCC\textit{mec} element were more resistant to antibiotics than MRSA isolates carring other type (especially type IV) SCC\textit{mec} element (20). Our study also demonstrated the same results. In the present study, MRSA isolates belonging to PFGE type B, C, J, and L, which carried type III SCC\textit{mec} element, usually were only susceptible to vancomycin, rifampin, and linezolid with little exception. However, MRSA isolates belonging to PFGE type D, G, H, and S, which carried type IV element, usually were susceptible to gentamicin, fluoroquinolone, and trimethoprim/sulfamethoxazole in addition to rifampin, vancomycin, and linezolid.

In conclusion, our study found that the predominant MRSA strain in Tai\text{"}wan carried type III SCC\textit{mec} element and the majority of CA-MRSA carried type IV SCC\textit{mec} element. There was also a predominant MRSA strain among the CA-MRSA isolates.
Another recent important finding about CA-MRSA was that some CA-MRSA carried Panton-Valentine Leukocidin (PVL) gene (30). The prevalence of PVL gene among CA-MRSA isolates, especially those carried type IV element, in Taiwan was worthy of further study. The origin of so-called CA-MRSA was also another important and interesting issue. Studies on the longitudinal evolution of MRSA in Taiwan might offer help to illuminate this unsolved problem.


25. Hiramatsu K, Kihara H, Yokota T. Analysis of borderline-resistant strains of


<table>
<thead>
<tr>
<th>PFGE</th>
<th>Oxa</th>
<th>Gen</th>
<th>Clin</th>
<th>Ery</th>
<th>Off</th>
<th>Levo</th>
<th>Tet</th>
<th>Rif</th>
<th>BXT</th>
<th>Van</th>
<th>Lin</th>
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<tr>
<td>B</td>
<td>≥128</td>
<td>32 ~ 128</td>
<td>≥128</td>
<td>&gt;128</td>
<td>64 ~ 128</td>
<td>2 ~ 4</td>
<td>128</td>
<td>&lt;0.03 ~ 1</td>
<td>≥128</td>
<td>1 ~ 2</td>
<td>0.5</td>
</tr>
<tr>
<td>C</td>
<td>&gt;128</td>
<td>16 ~ &gt;128</td>
<td>≥128</td>
<td>&gt;128</td>
<td>64 ~ &gt;128</td>
<td>4 ~ 16</td>
<td>64 ~ &gt;128</td>
<td>&lt;0.03 ~ 16</td>
<td>≥128</td>
<td>0.5 ~ 2</td>
<td>0.125 ~ 1</td>
</tr>
<tr>
<td>D</td>
<td>&gt;128</td>
<td>0.5 ~ 1.0</td>
<td>≥128</td>
<td>128</td>
<td>2 ~ 4</td>
<td>0.125</td>
<td>0.125 ~ 16</td>
<td>&lt;0.03</td>
<td>2</td>
<td>0.5</td>
<td>0.25 ~ 1</td>
</tr>
<tr>
<td>G</td>
<td>4 ~ 8</td>
<td>0.5 ~ 128</td>
<td>32 ~ ≥128</td>
<td>≥128</td>
<td>2 ~ 4</td>
<td>0.125</td>
<td>8 ~ 16</td>
<td>&lt;0.03</td>
<td>2</td>
<td>0.125 ~ 0.5</td>
<td>0.25 ~ 1</td>
</tr>
<tr>
<td>H</td>
<td>4 ~ 128</td>
<td>1 ~ 128</td>
<td>&gt;128</td>
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<td>2 ~ 128</td>
<td>0.125 ~ 4</td>
<td>8 ~ 32</td>
<td>&lt;0.03</td>
<td>2</td>
<td>0.25 ~ 0.5</td>
<td>0.5 ~ 1</td>
</tr>
<tr>
<td>J</td>
<td>≥128</td>
<td>8 ~ 16</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>64 ~ 128</td>
<td>2 ~ 4</td>
<td>16 ~ 64</td>
<td>&lt;0.03</td>
<td>&gt;128</td>
<td>0.5 ~ 1</td>
<td>0.5</td>
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<td>L</td>
<td>&gt;128</td>
<td>64</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>&gt;128</td>
<td>16</td>
<td>32 ~ 64</td>
<td>16</td>
<td>8</td>
<td>1</td>
<td>0.5</td>
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Abbreviation: MIC, minimum inhibitory concentration; Oxa, oxacillin; Gen, gentamicin; Clin, clindamycin; Ery, erythromycin; Off, ofloxacin; Levo, levofloxacin; Chl, chloramphenicol; Tet, tetracycline; Rif, rifampin; BXT, trimethoprim/sulfamethoxazole; Van, vancomycin; Lin, linezolid.
<table>
<thead>
<tr>
<th>PFGE</th>
<th>Oxa</th>
<th>Gen</th>
<th>Clin</th>
<th>Ery</th>
<th>Ofi</th>
<th>Levo</th>
<th>Tet</th>
<th>Rif</th>
<th>BXT</th>
<th>Van</th>
<th>Lin</th>
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<tbody>
<tr>
<td>C</td>
<td>≥128</td>
<td>16 ~ 32</td>
<td>&gt;128</td>
<td>4 ~ &gt;128</td>
<td>≥128</td>
<td>4 ~ 16</td>
<td>32 ~ 128</td>
<td>≤0.03</td>
<td>≥128</td>
<td>0.5 ~ 1</td>
<td>0.25 ~ 1</td>
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<tr>
<td>D</td>
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<td>≥128</td>
<td>8 ~ &gt;128</td>
<td>1 ~ 4</td>
<td>0.06 ~ 0.125</td>
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<td>2 ~ 4</td>
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<td>1</td>
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<tr>
<td>H</td>
<td>8 ~ 16</td>
<td>0.5</td>
<td>&gt;128</td>
<td>64 ~ &gt;128</td>
<td>2 ~ 4</td>
<td>0.125</td>
<td>0.25 ~ 16</td>
<td>&lt;0.03</td>
<td>2 ~ 4</td>
<td>0.5</td>
<td>1</td>
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<tr>
<td>S</td>
<td>4 ~ 16</td>
<td>0.5</td>
<td>&gt;128</td>
<td>≥128</td>
<td>2 ~ 4</td>
<td>0.125</td>
<td>16</td>
<td>&lt;0.03</td>
<td>2</td>
<td>0.5</td>
<td>1</td>
</tr>
</tbody>
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Table 3. The types of *mecA* gene cluster, *ccr* gene, and SCC*mec* element of MRSA isolates

<table>
<thead>
<tr>
<th>PFGE type</th>
<th>B</th>
<th>C</th>
<th>D</th>
<th>G</th>
<th>H</th>
<th>J</th>
<th>L</th>
<th>S</th>
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</thead>
<tbody>
<tr>
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<td>A</td>
<td>A</td>
<td>B</td>
<td>B</td>
<td>B</td>
<td>A</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td><em>ccr</em> gene</td>
<td>3</td>
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<td>2</td>
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</tr>
<tr>
<td>SCC<em>mec</em></td>
<td>III</td>
<td>III</td>
<td>IV</td>
<td>IV</td>
<td>IV</td>
<td>III</td>
<td>III</td>
<td>IV</td>
</tr>
</tbody>
</table>
Figure. The PFGE banding patterns of some PFGE types.
計畫成果自評

由上述的研究報告中，可以清楚的發現我們已釐清了台灣地區 MRSA 菌株，不管是院內或社區性，SCCmec element 的分型；闡明了院內 MRSA 和社區性 MRSA 所攜帶 SCCmec element 的之差異；發現了社區性 MRSA 菌株也有一個 predominant strain。這些重要的發現，都讓我們對台灣本土的 MRSA 菌株的細菌特性有了進一步的了解。我們的研究結果，符合原先所設定的個個目標，也提出了若干進一步的問題、刺激我們進行更深一層的研究。

整體而言，我們的研究結果清楚而重要，不失為台灣本土針對 MRSA 抗藥性基因的一個好的研究。