

**AFO** 2017

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**AFOS** Asian Federation of  
Osteoporosis Societies

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**PROGRAMME &  
ABSTRACT BOOK**

# The 5th Scientific Meeting of the Asian Federation of Osteoporosis Societies **2017**

6th – 8th October 2017 • Hilton Kuala Lumpur, Malaysia

Theme: *Best Practices, Future Directions.*

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**CONFERENCE SECRETARIAT  
ASIAN FEDERATION OF OSTEOPOROSIS SOCIETIES (AFOS)**

**c/o MEDICAL CONFERENCE PARTNERS**

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## WELCOME MESSAGE FROM THE PRESIDENT OF THE ASIAN FEDERATION OF OSTEOPOROSIS SOCIETIES (AFOS)



On behalf of the Asian Federation of Osteoporosis Societies (AFOS), it gives me great pleasure to welcome you to the 5th Annual Scientific Meeting, in Kuala Lumpur, Malaysia. This biannual meeting would continue the tradition of bringing key opinion leaders, clinicians and scientists from the region to share their knowledge and opinions.

It is predicted that we will see half of all the osteoporotic hip fractures in Asia in the next couple of decades. This is not just due to aging populations in the region but also an ever increasing treatment gap. There is much urgency for us as professionals to prevent and delay fractures in our communities.

The AFOS conference 2017 is fully intended to provide a platform for discussions and interaction so that together we can gain a better insight on osteoporosis and to advance research and collaboration across the region. From the laboratory bench to new therapeutic agents; from epidemiology to clinical outcome; from new ideas to fresh research data; there will be plenty to discuss and impart.

AFOS has moved a long way since its formation a decade ago. Other than the biannual meeting, the AFOS journal, *Osteoporosis and Sarcopenia*, was set up a few years ago to encourage research and publication of such work. AFOS is keen to adopt a programme to encourage the formation of Fracture Liaison Service in its member societies that would manage those who are most at risk of further fractures. AFOS advocacy role has started by working on a declaration statement with wide representation. We are also hoping to expand the AFOS family in Asia as we want to be an inclusive Pan-Asian professional organisation that welcomes any national or regional societies that share our vision.

It is your participation and ideas that would move all of us forward with greater haste and purpose for the betterment of bone health in the region. I hope you enjoy the conference that has been put together for you by an extremely hardworking committee and an equally capable faculty.

### Dr Fen Lee HEW

President

Asian Federation of Osteoporosis Societies (AFOS)



## ASIAN FEDERATION OF OSTEOPOROSIS SOCIETIES (AFOS) COUNCIL MEMBERS



### President

Dr Fen Lee HEW  
*Malaysia*



### Member

Dr Ye-Soo PARK  
*Korea*



### Member

Dr Unnop JAISAMRARN  
*Thailand*



### Vice President

Dr Leilani B. MERCADO-ASIS  
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Dr Akira TAGUCHI  
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### Member

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### Treasurer

Dr Ka Kui LEE  
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### Member

Dr Hou NG  
*Macau*



### Member

Dr Weibo XIA  
*China*



### Secretary

Dr Tang Ching LAU  
*Singapore*



## ABOUT ASIAN FEDERATION OF OSTEOPOROSIS SOCIETIES (AFOS)

1. To make the Asian region excel in education and research in all aspects of osteoporosis, thereby bringing to the region the highest quality of healthcare in this field of medicine for charitable purposes beneficial to the community.
2. To promote the prevention, management and research of osteoporosis and disseminate the result for public benefit.
3. To encourage and support co-operative research in osteoporosis in Asia and beyond and disseminate the result for public benefit.
4. To promote, foster, develop and assist medical and allied profession in the study of and the acquisition, dissemination and application of knowledge and information concerning the causes, diagnosis, prevention and treatment of osteoporosis.
5. To stimulate public interest and provide public education in the prevention and treatment of osteoporosis and related problems and to assist in keeping the medical profession in the Asian region up-to-date in the latest developments in the field of medical and scientific research and methods of diagnosis, prevention and treatment of osteoporosis.



### MEMBER SOCIETIES



The Osteoporosis Society Of Hong Kong



Osteoporosis Society Of Macau



Taiwan Osteoporosis Association



Thai Osteoporosis Foundation



Chinese Society Of Osteoporosis And Bone Mineral Research



Malaysian Osteoporosis Society



Korean Society Of Osteoporosis



Osteoporosis Society Of The Philippines Foundation, Inc



Japan Osteoporosis Society



Osteoporosis Society Of Singapore



### AFOS SCIENTIFIC COMMITTEE



Dr Andrew Yiu Yan HO, Hong Kong  
Dr Joanne LAM, Hong Kong



Dr Hou NG, Macau  
Dr Kit Man VONG, Macau



Dr Keh-Sung TSAI, Taiwan  
Dr Paulo Chih-Hsing WU, Taiwan



Dr Unnop JAISAMRARN, Thailand  
Dr Thawee SONGPATANASILP, Thailand



Dr Weibo XIA, China  
Dr Zhenlin ZHANG, China



Dr Jae-Hyup LEE, Korea  
Dr Sihoon LEE, Korea



Dr Mark Anthony SANDOVAL, The Philippines  
Dr Theresa Marie VALDEZ-FALLER, The Philippines



Dr Akira TAGUCHI, Japan  
Dr Yasuhiro TAKEUCHI, Japan



Dr Seng Bin ANG, Singapore  
Dr Manju CHANDRAN, Singapore



## WELCOME MESSAGE FROM THE ORGANISING CHAIRPERSON OF THE 5TH SCIENTIFIC MEETING OF AFOS 2017



On behalf of the Organising Committee, I would like to warmly welcome you to Kuala Lumpur, Malaysia, for the 5th Scientific Meeting of the Asian Federation of Osteoporosis Societies (AFOS).

The theme of the meeting is “Best Practices, Future Directions”. With this in mind, the programme aims to update you both on the current best practices in osteoporosis, and as well as looking forward to what we can possibly expect in the future. We believe that the programme will be useful to all medical and paramedical professionals who see and manage patients with osteoporosis. For all their efforts put into developing this state-of-the-art programme, I would like to thank the Scientific Committee, ably headed by Professor Dr Siew Pheng Chan. To the speakers, both local and international, we would like to thank you for taking time to provide your valuable insights on the various topics in the programme.

In addition, we have received an increasing number of abstracts submitted by professionals from 16 countries this year. We appreciate the time taken by each submitter to present their research and look forward to listening/viewing the presentations that have qualified for the award sessions.

We would like to take this opportunity to thank the participating pharmaceutical companies for supporting our conference. Such support is crucial to providing high quality continuing education and professional development to healthcare providers in this region on osteoporosis, and is much appreciated.

Finally, after a hard day acquiring knowledge in the conference halls, we hope that you will take advantage of Kuala Lumpur’s vibrant sights and sounds, including its diverse food options. In a 2015 poll of CNN Travel readers, Malaysia came sixth in a list of top 10 culinary hotspots to travel to; so do take advantage while you are here! Let me end by wishing everyone an enjoyable and fruitful meeting both educationally and socially!

**Dr Swan Sim YEAP**  
Organising Chairperson  
5th Scientific Meeting of the Asian Federation of Osteoporosis Societies 2017



### 5TH AFOS LOCAL ORGANISING COMMITTEE

Organising Chairperson	: Dr Swan Sim YEAP	Sponsorship Chairperson	: Dr Fen Lee HEW
Deputy Organising Chairperson	: Dr Boon Ping LIM	Sponsorship Committee	: Dr Siew Pheng CHAN : Dr Swan Sim YEAP : Dr Boon Ping LIM : Dr Premitha DAMODARAN
Treasurer	: Dr Yew Siong SLOW	Social Committee	: Dr Emily Man Lee GOH : Dr Premitha DAMODARAN
Secretary	: Dr Alexander Tong Boon TAN		
Scientific Chairperson	: Dr Siew Pheng CHAN		
Deputy Scientific Chairperson	: Dr Joon Kiong LEE		
Scientific Committee	: Dr Fen Lee HEW : Dr Swan Sim YEAP : Dr Yew Siong SLOW : Dr Winnie CHEE : Dr Fatt Soon LEE : Dr Zanariah HUSSEIN : Dr Jamiyah HASSAN		

**Conference Venue**

HILTON KUALA LUMPUR  
3, Jalan Stesen Sentral 5,  
Kuala Lumpur Sentral,  
50470 Kuala Lumpur, Malaysia  
Tel : +603 2264 2264  
Email : kuala-lumpur@hilton.com  
Website : www.life.hiltonkl.com

**Conference Secretariat**

ASIAN FEDERATION OF OSTEOPOROSIS SOCIETIES (AFOS)  
c/o MEDICAL CONFERENCE PARTNERS  
No. 4, Lengkongan Jenjarom, Taman Seputeh,  
58000 Kuala Lumpur, Malaysia  
Tel : +603 2276 0555 / +6016 335 0036  
Fax : +603 6207 6795

**Registration**

All delegates are required to register at the Registration Counter, located at Level 6 with the official confirmation letter sent via email. Conference collaterals are to be collected at the Registration Counter. All Conference Attendees and Exhibitors must wear their name badges at all times during this conference to access all scientific sessions, meals and exhibitions.

**Registration Counter Opening Hours**

6th October 2017, Friday : 07.00 – 17.30 hrs  
7th October 2017, Saturday : 07.30 – 17.00 hrs  
8th October 2017, Sunday : 07.30 – 13.00 hrs

**Speaker Ready Room**

The Speaker Ready Room is located on Level 6, beside the escalator. All speakers are required to submit their presentation slide(s) at least 4 hours before their scheduled presentation time to ensure a smooth presentation.

**Speaker Ready Room Opening Hours**

6th October 2017, Friday : 07.00 – 17.30 hrs  
7th October 2017, Saturday : 07.30 – 17.00 hrs  
8th October 2017, Sunday : 07.30 – 13.00 hrs

**Abstracts**

The Organising Committee has decided to print the speakers' abstracts in this programme and abstract book. You may also view the abstracts on the conference website at [www.afos2017malaysia.com](http://www.afos2017malaysia.com).

**Poster Panel / Presentation Area**

All poster presenters must put up their posters on the poster panel from 07.30 hrs on 6th October 2017. It should be displayed on the poster panel throughout the duration of the conference and taken down by 10.30 hrs on 8th October 2017.

Please refer to page 36 to 39 in this book for the location of your poster.

**Continuing Professional Development (CPD) Point**

The Malaysian Medical Council has set that all Malaysian doctors need to secure a minimum number of CPD points in order to renew their Annual Practising Certificate (APC).

The Malaysian Medical Association (MMA) has introduced a special mobile application by which you can keep track of your CPD points collected as well as provide a unique QR code by which event organisers can scan and record your attendance at Registration Counter. Your CPD point will be instantly updated. This mobile application is available for both MMA members and non-members.

To download the MMA mobile app, please visit <http://onelink.to/mmaapp> in your mobile's browser.

**Certificate Of Attendance**

Your certificate of attendance will be given.

**Dress Code**

The dress code for the meeting is smart business attire.

**Parking**

The parking fee at the conference venue is RM8 per day. Validation counter is located in front of the Business Center, Level 6.

**Prayer Room**

The prayer room for Muslim delegates is available at Level P1.

**Nearest Mosque**

The nearest mosque is the National Mosque of Malaysia which is an 8-minute drive from the conference venue.

**Getting Around**

Hilton Kuala Lumpur (Conference Venue) is strategically located opposite Sentral Kuala Lumpur, which is the largest transit hub that houses the main railway stations of Kuala Lumpur, the capital of Malaysia. Visitors can make use of the five rail networks (Monorail, LRT, Commuter, ERL and MRT) and public transport to travel around Kuala Lumpur.

In addition to the Malaysian Taxi Services, mobile application based car services such as Grab and Uber are available and widely used in Malaysia.

**Lost And Found**

Please take care of all your belongings. The organiser and secretariat will not be held liable in case of loss, theft or damage to personal belongings. Any misplaced items can be brought to the Registration Counter.

Dr Matin Mellor ABDULLAH  
*Malaysia*

Dr Tanawat AMPHANSAP  
*Thailand*

Dr Seng Bin ANG  
*Singapore*

Dr John J. CAREY  
*Ireland*

Dr Siew Pheng CHAN  
*Malaysia*

Dr Wai Sin CHAN  
*Macau*

Dr Manju CHANDRAN  
*Singapore*

Dr Natthinee CHARATCHAROENWITTHAYA  
*Thailand*

Dr Winnie CHEE  
*Malaysia*

Dr Tien-Tsai CHENG  
*Taiwan*

Dr Ching-Lung CHEUNG  
*Hong Kong*

Dr Siok Bee CHIONH  
*Singapore*

Dr Yong Jun CHOI  
*Korea*

Dr Chee Seang CHONG  
*Malaysia*

Dr Peter EBELING  
*Australia*

Dr Serge FERRARI  
*Switzerland*

Dr Fen Lee HEW  
*Malaysia*

Dr Angela Wing Hang HO  
*Hong Kong*

Dr Jawl-Shan HWANG  
*Taiwan*

Dr Tai-Pang IP  
*Hong Kong*

Dr Shahrul Bahyah KAMARUZZAMAN  
*Malaysia*

Dr Bom Taeck KIM  
*Korea*

Dr Tang Ching LAU  
*Singapore*

Dr Dong Ock LEE  
*Korea*

Dr Joon Kiong LEE  
*Malaysia*

Dr Julie T. LI-YU  
*The Philippines*

Dr Mastura MD YUSOF  
*Malaysia*

Dr Leilani B. MERCADO-ASIS  
*The Philippines*

Dr Alejandro V. PINEDA JR  
*The Philippines*

Dr Yew Siong SIOW  
*Malaysia*

Dr Chanika SRITARA  
*Thailand*

Dr Atsushi SUZUKI  
*Japan*

Dr Akira TAGUCHI  
*Japan*

Dr Yasuhiro TAKEUCHI  
*Japan*

Dr Alexander Tong Boon TAN  
*Malaysia*

Dr Paulo Chih-Hsing WU  
*Taiwan*

Dr Weibo XIA  
*China*

Dr Rong-Sen YANG  
*Taiwan*

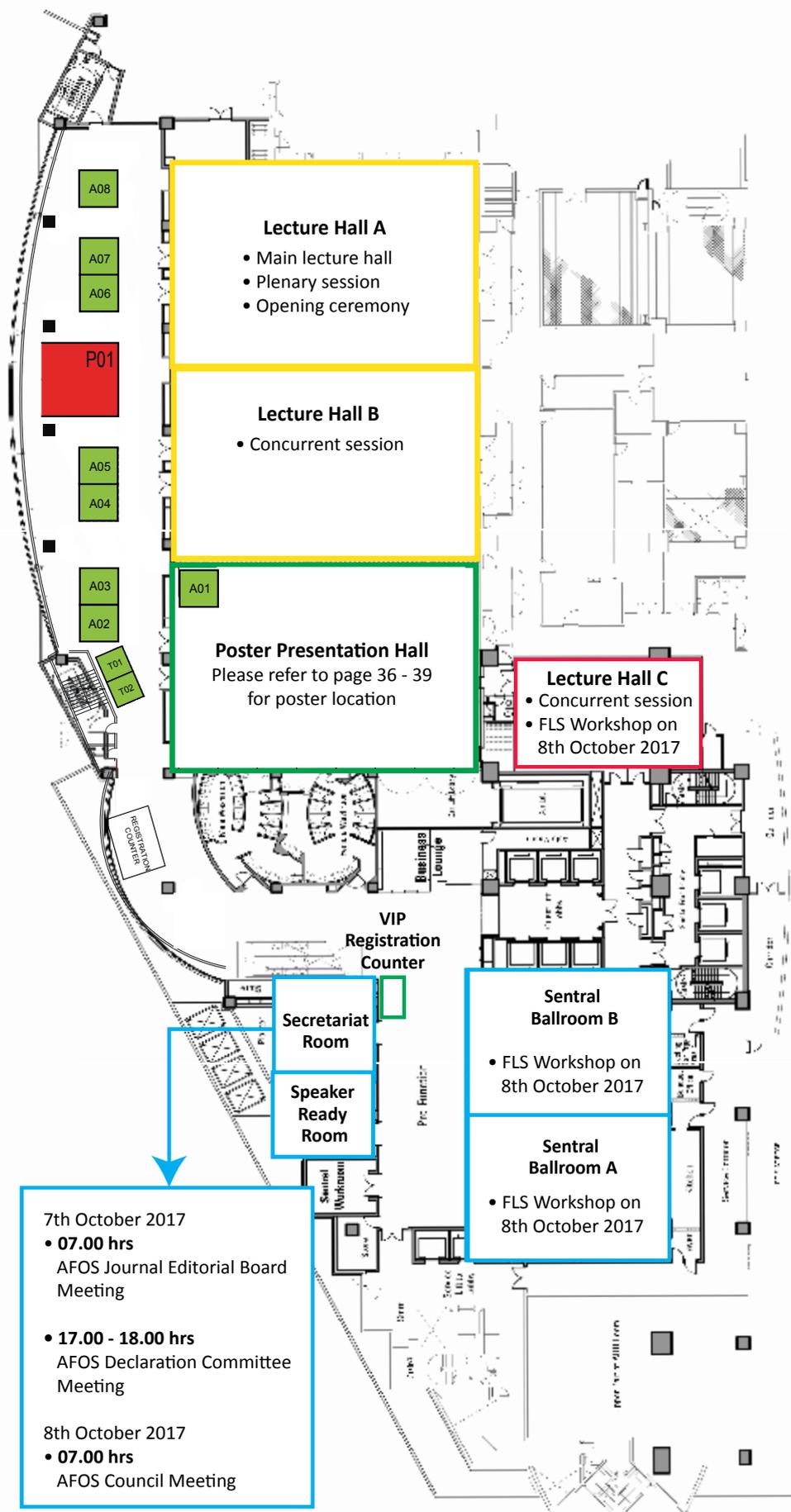
Dr Bu Beng YEAP  
*Australia*

Dr Swan Sim YEAP  
*Malaysia*

Dr Wei YU  
*China*

Dr Ling-Qing YUAN  
*China*

Dr Hua YUE  
*China*





## PROGRAMME OVERVIEW

Day 1 - Friday (6th October 2017)			
Time	Lecture Hall A	Lecture Hall B	Lecture Hall C
07.00 - 17.30	Registration counter opens		
08.45 - 09.00	Introduction and welcome		
09.00 - 09.30	<b>Plenary 1</b> Osteoporosis – Disease burden and treatment gap		
09.30 - 10.00	<b>Plenary 2</b> Unmet need and practical strategy to find the under-diagnosed osteoporotic patients		
10.00 - 10.45	Opening ceremony		
10.45 - 11.15	Tea break & exhibit visit		
11.15 - 12.30	<b>Symposium 1</b> Nutrition, Calcium and Vitamin D: Issues, Controversies	<b>Symposium 2</b> Therapeutics – Part 1	
12.30 - 13.45	Lunch Symposium by Amgen		
13.45 - 14.25	<b>Meet The Expert 1</b> Physical activity: What works, what's safe	<b>Meet The Expert 2</b> Atypical femoral fracture	<b>Meet The Expert 3</b> Thyroid disease/parathyroid disease and bone health
14.25 - 15.40	<b>Symposium 3</b> Imaging – Beyond bone density	<b>Symposium 4</b> Bone biology and biomechanics	Free Paper Oral Presentations 1
15.40 - 16.10	<b>Plenary 3</b> Bone, genes and fractures: Update on genetics of osteoporosis		
16.10 - 17.10	High-Tea Symposium by Mylan / Meda Healthcare		

Day 2 - Saturday (7th October 2017)			
Time	Lecture Hall A	Lecture Hall B	Lecture Hall C
07.30 - 17.00	Registration counter opens		
08.20 - 09.00	<b>Meet The Expert 4</b> Monitoring response to treatment – To scan or not to scan	<b>Meet The Expert 5</b> Glucocorticoid-induced osteoporosis – Difficult cases	<b>Meet The Expert 6</b> Application of body composition assessment by DXA in chinese patients with HIV / AIDS
09.00 - 09.30	<b>Plenary 4</b> Diabetoporosis and obesity		
09.30 - 10.00	<b>Plenary 5</b> Inflammatory disorders and the skeleton		
10.00 - 10.30	Tea break & exhibit visit		
10.30 - 11.45	<b>Symposium 5</b> Therapeutics – Part 2	<b>Symposium 6</b> Sarcopenia	Young Investigator Award Presentations
11.45 - 12.15	<b>Plenary 6</b> Update on osteonecrosis of the jaw (ONJ)		
12.15 - 13.30	Lunch Symposium by A. Menarini Best Poster Award Presentations		
13.30 - 14.10	<b>Symposium 7</b> Mandarin Symposium	<b>Meet The Expert 7</b> ONJ – International consensus	<b>Meet The Expert 8</b> Controversy of calcium & Vitamin D supplementation
14.10 - 15.25	<b>Symposium 8</b> Cancer and bone		Free Paper Oral Presentations 2
15.25 - 15.55	<b>Plenary 7</b> Tumor-induced osteomalacia		
15.55 - 16.55	High-Tea Symposium by MSD		

Day 3 - Sunday (8th October 2017)			
Time	Lecture Hall A	Lecture Hall B	Lecture Hall C
07.30 - 13.00	Registration counter opens		
08.20 - 09.00	<b>Meet The Expert 9</b> Andropause – Debunking the myth	<b>Meet The Expert 10</b> Sarcopenia: The way forward	<b>Meet The Expert 11</b> FRAX – Update on utility
09.00 - 09.30	<b>Plenary 8</b> Therapies past and present – Guiding future directions		
09.30 - 10.45	<b>Symposium 9</b> OP – Hormonal therapies	<b>Symposium 10</b> OP – Special situations	<b>Symposium 11</b> Surgical management
10.45 - 11.15	Tea break & exhibit visit		
11.15 - 11.45	<b>Plenary 9</b> Osteoporosis – New algorithm: Treating to target		
11.45 - 12.15	Presentation of Awards - Young Investigator Award and Best Poster Award		
12.15 - 14.00	Lunch Symposium by Amgen		
14.00 - 17.00	Fracture Liaison Services Workshop by Amgen		

Time	Programme	
07.00 – 17.30	Registration counter opens	
	Lecture Hall A	
08.45 – 09.00	Introduction and Welcome	
09.00 – 09.30	Plenary 1 <i>Chairperson: Dr Swan Sim YEAP</i>	
	Osteoporosis – Disease burden and treatment gap <i>Dr Fen Lee HEW</i>	
09.30 – 10.00	Plenary 2 <i>Chairperson: Dr Swan Sim YEAP</i>	
	Unmet need and practical strategy to find the under-diagnosed osteoporotic patients <i>Dr Paulo Chih-Hsing WU</i>	
10.00 – 10.45	Opening Ceremony	
10.45 – 11.15	Tea break & exhibit visit	
	Lecture Hall A	Lecture Hall B
	Symposium 1 – Nutrition, Calcium and Vitamin D: Issues, Controversies <i>Chairperson: Dr Hou NG</i>	Symposium 2 – Therapeutics – Part 1 <i>Chairperson: Dr Siok Bee CHIONH</i>
11.15 – 11.35	Calcium – What do we recommend our patients? <i>Dr Natthinee CHARATCHAROENWITTHAYA</i>	Bisphosphonates - Short term and long term efficacy <i>Dr Jawl-Shan HWANG</i>
11.35 – 11.55	Vitamin D – Is it really necessary? <i>Dr Siew Pheng CHAN</i>	Therapeutics - Denosumab <i>Dr Tai-Pang IP</i>
11.55 – 12.15	Role of macronutrients, functional foods and beyond <i>Dr Winnie CHEE</i>	Anabolic therapies <i>Dr Manju CHANDRAN</i>
12.15 – 12.30	Q & A	Q & A
12.30 – 13.45	Lecture Hall A	
	Lunch Symposium by Amgen: Making fracture prevention a priority <i>Chairperson: Dr Fen Lee HEW</i>	
	Finding the patient with increased risk of fracture - A call to action! <i>Dr Tai-Pang IP</i>	
	Optimising osteoporosis patient management in the short and long term <i>Dr Serge FERRARI</i>	
	Q&A <i>All above</i>	

Time	Programme		
	Lecture Hall A	Lecture Hall B	Lecture Hall C
13.45 – 14.25	Meet The Expert 1 Physical activity: What works, what's safe <i>Dr Alejandro V. PINEDA JR</i>	Meet The Expert 2 Atypical femoral fracture <i>Dr Joon Kiong LEE</i>	Meet The Expert 3 Thyroid disease/parathyroid disease and bone health <i>Dr Siok Bee CHIONH</i>
	Symposium 3 Imaging – Beyond bone density <i>Chairperson: Dr Joon Kiong LEE</i>	Symposium 4 Bone biology and biomechanics <i>Chairperson: Dr Leilani B. MERCADO-ASIS</i>	Free paper oral presentations 1 (9 minutes per presenter) <i>Chairperson: Dr Manju CHANDRAN Dr Julie T. LI-YU</i>  Please refer to page 16
14.25 – 14.45	Bone mineral density – Best practice <i>Dr John J. CAREY</i>	Genetics and epigenetics of bone development <i>Dr Ching-Lung CHEUNG</i>	
14.45 – 15.05	Bone turnover markers in fracture risk prediction <i>Dr Yong Jun CHOI</i>	Signal transduction involving bone cells coupling <i>Dr Ling-Qing YUAN</i>	
15.05 – 15.25	Clinical utility of TBS in management of osteoporosis <i>Dr Yong Jun CHOI</i>	Predictors of osteoporotic fracture risk <i>Dr Hua YUE</i>	
15.25 – 15.40	Q & A	Q & A	
	<b>Lecture Hall A</b>		
15.40 – 16.10	Plenary 3 <i>Chairperson: Dr Zanariah HUSSEIN</i>		
	Bone, genes and fractures: Update on genetics of osteoporosis <i>Dr Peter EBELING</i>		
16.10 – 17.10	High-Tea Symposium by Mylan / Meda Healthcare <i>Chairperson: Dr Joon Kiong LEE</i>		
	Management of knee osteoarthritis <i>Dr Swan Sim YEAP</i>		

Time	Programme		
07.30 – 17.00	Registration counter opens		
	Lecture Hall A	Lecture Hall B	Lecture Hall C
	Meet The Expert 4	Meet The Expert 5	Meet The Expert 6
08.20 – 09.00	Monitoring response to treatment – To scan or not to scan <i>Dr Chanika SRITARA</i>	Glucocorticoid-induced osteoporosis – Difficult cases <i>Dr Swan Sim YEAP</i>	Application of body composition assessment by DXA in chinese patients with HIV / AIDS <i>Dr Wei YU</i>
	Lecture Hall A		
09.00 – 09.30	Plenary 4 <i>Chairperson: Dr Alexander Tong Boon TAN</i>		
	Diabetoporosis and obesity <i>Dr Serge FERRARI</i>		
09.30 – 10.00	Plenary 5 <i>Chairperson: Dr Alexander Tong Boon TAN</i>		
	Inflammatory disorders and the skeleton <i>Dr Julie T. LI YU</i>		
10.00 – 10.30	Tea break & exhibit visit		
	Lecture Hall A	Lecture Hall B	Lecture Hall C
	Symposium 5 Therapeutics – Part 2 <i>Chairperson: Dr Emily Man Lee GOH</i>	Symposium 6 Sarcopenia <i>Chairperson: Dr Fatt Soon LEE</i>	Young Investigator Award Presentations (9 minutes per presenter) <i>Chairperson: Dr Ching-Lung CHEUNG</i>  Please refer to page 15
10.30 – 10.50	Treatment failure in osteoporosis <i>Dr Rong-Sen YANG</i>	Sarcopenia & frailty: Is it inevitable? <i>Dr Bom Taeck KIM</i>	
10.50 – 11.10	Bisphosphonate – Balancing risks & benefits <i>Dr Atsushi SUZUKI</i>	Frailty screening tools <i>Dr Shahrul Bahyah KAMARUZZAMAN</i>	
11.10 – 11.30	Drug holiday – Getting to grips <i>Dr Leilani B. Mercado-ASIS</i>	Nutrition and exercise – Maintaining intrinsic capacity <i>Dr Alejandro V. PINEDA JR</i>	
11.30 – 11.45	Q & A	Q & A	
	Lecture Hall A		
11.45 – 12.15	Plenary 6 <i>Chairperson: Dr Tang Ching LAU</i>		
	Update on osteonecrosis of the jaw (ONJ) <i>Dr Akira TAGUCHI</i>		
	Lunch symposium by A. Menarini : The past, present and future of osteoporosis management <i>Chairperson: Dr Vijaya Kumar S. L.</i>		
12.15 – 13.30	Revisiting the role of Alendronate in osteoporosis management <i>Dr Siew Pheng CHAN</i>		
	The role of Cholecalciferol for fall prevention in the elderly <i>Dr Maw Pin TAN</i>		
12.15 – 13.30	Best Poster Award Presentations (Please refer to page 15)		

Time	Programme		
	Lecture Hall A	Lecture Hall B	Lecture Hall C
	Symposium 7 (Mandarin Symposium) (13.30 – 15.25) <i>Chairperson: Dr Joon Kiong LEE</i>	Meet The Expert 7	Meet The Expert 8
13.30 – 14.10	Panelists: 1. Dr Weibo XIA 2. Dr Tang Ching LAU 3. Dr Paulo Chih-Hsing WU 4. Dr Wai Sin CHAN  Case Presenters: 1. Dr Ling-Qing YUAN 2. Dr Yew Siong SLOW 3. Dr Tang Ching LAU	ONJ – International consensus <i>Dr Akira TAGUCHI</i>	Controversy of calcium & Vitamin D supplementation <i>Dr Siew Pheng CHAN</i>
14.10 – 14.30		Symposium 8 Cancer and bone <i>Chairperson: Dr Yoon-Sok CHUNG</i>	Free paper oral presentations 2 (9 minutes per presenter) <i>Chairperson:            Dr Unnop JAISAMRARN</i>  Please refer to page 16
14.30 – 14.50		Bone and cancer – Fatal attraction <i>Dr Siew Pheng CHAN</i>	
14.50 – 15.10		Cancer treatment induced bone loss – Size of the problem! <i>Dr Martin Mellor ABDULLAH</i>	
15.10 – 15.25		CTIBL – Prevention and management: New standard of care <i>Dr Mastura MD YUSOF</i>	
	Q & A	Q & A	
	Lecture Hall A		
15.25 – 15.55	Plenary 7 <i>Chairperson: Dr Tai-Pang IP</i>  Tumor-induced osteomalacia <i>Dr Weibo XIA</i>		
15.55 – 16.55	High-Tea Symposium by MSD <i>Chairperson: Dr Alexander Tong Boon TAN</i>  How does diabetic disease affect the choice of antidiabetic agent? <i>Dr Fen Lee HEW</i>		

Time	Programme		
07.30 – 13.00	Registration counter opens		
	Lecture Hall A	Lecture Hall B	Lecture Hall C
	Meet The Expert 9	Meet The Expert 10	Meet The Expert 11
08.20 – 09.00	Andropause - Debunking the myth <i>Dr Bu Beng YEAP</i>	Sarcopenia: The way forward <i>Dr Serge FERRARI</i>	FRAX – Update on utility <i>Dr Tang Ching LAU</i>
	Lecture Hall A		
09.00 – 09.30	Plenary 8 <i>Chairperson: Dr Andrew Yiu Yan HO</i>		
	Therapies past and present – Guiding future directions <i>Dr Peter EBELING</i>		
	Lecture Hall A	Lecture Hall B	Lecture Hall C
	Symposium 9 OP – Hormonal therapies <i>Chairperson: Dr Premitha DAMODARAN</i>	Symposium 10 OP – Special situations <i>Chairperson: Dr Sihoon LEE</i>	Symposium 11 Surgical management <i>Chairperson: Dr Wai Sin CHAN</i>
09.30 – 09.50	Menopausal hormone therapy <i>Dr Seng Bin ANG</i>	Effectiveness and safety of denosumab for osteoporosis in patients with chronic kidney disease (CKD) <i>Dr Yasuhiro TAKEUCHI</i>	Management of painful vertebral fracture <i>Dr Chee Seang CHONG</i>
09.50 – 10.10	SERMs <i>Dr Dong Ock LEE</i>	Guidelines for the management of glucocorticoid-induced osteoporosis <i>Dr Tien-Tsai CHENG</i>	Hip Fracture – Typical and atypical <i>Dr Angela Wing Hang HO</i>
10.10 – 10.30	Hormonal therapies: Testosterone therapy <i>Dr Bu Beng YEAP</i>	Pregnancy induced osteoporosis <i>Dr Alexander Tong Boon TAN</i>	Fracture fixation with augmentation in osteoporotic fracture <i>Dr Tanawat AMPHANSAP</i>
10.30 – 10.45	Q&A	Q&A	Q&A
10.45 – 11.15	Tea break & exhibit visit		
	Lecture Hall A		
11.15 – 11.45	Plenary 9 <i>Chairperson: Dr Boon Ping LIM</i>		
	Osteoporosis – New algorithm: Treating to target <i>Dr Serge FERRARI</i>		
11.45 – 12.15	Presentation of Awards – Young Investigator Award and Best Poster Award		
	Lunch Symposium by Amgen : Fracture Liaison Services: Feel good or good business sense? <i>Chairperson: Dr Peter EBELING &amp; Dr Atsushi SUZUKI</i>		
12.15 – 14.00	Health economics of the aging population in Asia: Who benefits from fracture prevention initiatives? <i>Dr Hector WONG</i>		
	Closing the secondary fracture prevention care gap with FLS <i>Dr Paul MITCHELL</i>		
	FLS: Is it feasible and what has been successful in Asia? <i>Dr Derrick CHAN, Dr Joon Kiong LEE, Dr Tang Ching LAU, Dr Atsushi SUZUKI</i>		
	Discussion: Should osteoporosis be a healthcare priority in Asia? <i>All above</i>		
14.00 – 17.00	Fracture Liaison Services Workshop by Amgen		


**YOUNG INVESTIGATOR AWARD PRESENTATIONS**  
**Day 2 - Saturday (7th October 2017)**

Time	ID	Title	Name	Country
10.30 - 10.39	YIA001	Prevention of subsequent fractures of hip with use of bisphosphonates in Pakistan population	Dr Muhammad Arsalan GHAZI	Pakistan
10.39 - 10.48	YIA002	Prevalence and clinical correlation of Vitamin D inadequacy in traumatic elderly patients underwent orthopaedic surgery	Dr Ong-art PHRUETTHIPHAT	Thailand
10.48 - 10.57	YIA003	Fracture Liaison Service: Four-year performance in a rural hospital in Taiwan	Dr Min-Hong HSIEH	Taiwan
10.57 - 11.06	YIA004	Obesity contributes to low trabecular bone score (TBS) in type 2 diabetes	Dr Reiko WATANABE	Japan
11.06 - 11.15	YIA005	Spine rehabilitation exercise as an adjuvant to bisphosphonates in the treatment of osteoporosis	Dr George Ezekiel A/L SILVANATHAN	Malaysia
11.15 - 11.24	YIA006	Outcomes of kyphoplasty in osteoporotic vertebral fractures: A day care procedure	Dr Syed Faraz Ul Hassan SHAH GILLANI	Pakistan
11.24 - 11.33	YIA007	Bulleyaconitine A prevents Ti particle-induced osteolysis and estrogen deficiency-induced osteoporosis via suppressing NF- $\kappa$ B signal pathway during osteoclastogenesis	Dr Liwei ZHANG	China
11.33 - 11.42	YIA008	The effects of testosterone deprivation induced by buserelin and orchidectomy on bone in a rat model of osteoporosis	Dr Kok Yong CHIN	Malaysia


**BEST POSTER AWARD PRESENTATIONS**  
**Day 2 - Saturday (7th October 2017)**

Time	ID	Title	Name	Country
12.15 - 12.22	BPA001	Canal to diaphysis ratio - A reliable indicator for hip fracture risk or not in elderly population	Dr Yong Lin TAN	Malaysia
12.23 - 12.30	BPA002	Does bisphosphonate based anti-osteoporosis medication affect osteoporotic spinal fracture healing?	Dr Young-Hoon KIM	Korea
12.31 - 12.38	BPA003	Treatment of balloon kyphoplasty for osteoporotic vertebral fractures with middle column compromise	Dr Teppei SENDA	Japan
12.39 - 12.46	BPA004	Usefulness of a bio-absorbable plate for periprosthetic fracture of the femur	Dr Yoshihiro ISHIHAMA	Japan
12.47 - 12.54	BPA005	Urine pentosidine levels are negatively associated with trabecular bone score in patients with type 2 diabetes	Dr Yong Jun CHOI	Korea
12.55 - 13.02	BPA006	Increased risk of osteoporosis in patients with nonalcoholic fatty liver disease: A population-based retrospective cohort study	Dr Hong-Jhe CHEN	Taiwan
13.03 - 13.10	BPA007	Effect of antiepileptic drug therapy on wnt signaling antagonists in Indian women with epilepsy	Bushra PARVEEN	India
13.11 - 13.18	BPA008	A survey on osteoporosis management among Filipino general practitioners	Dr Elaine VENEGAS	The Philippines

 **FREE PAPER ORAL PRESENTATIONS 1**  
**Day 1 - Friday (6th October 2017)**

Time	ID	Title	Name	Country
14.25 - 14.34	FPOP1.1	The use of a pathway checklist to improve the management of osteoporosis in patients with hip fragility fractures	Dr Shu-Lin TEO	Singapore
14.34 - 14.43	FPOP1.2	The relationship between serum lipids versus bone mass, bone metabolism and vertebral fractures in healthy Chinese postmenopausal women	Dr Meiyao WU	China
14.43 - 14.52	FPOP1.3	Secondary prevention of fragility fractures. Are we doing enough?	Dr Juzaily Fekry LEONG	Malaysia
14.52 - 15.01	FPOP1.4	Level of osteoporosis awareness and its related factors in low density distal radius fractures patients aged 50 years and older in Asian country	Dr Muhammad MUZZAMMIL	Pakistan
15.01 - 15.10	FPOP1.5	Effect of nutritional supplement for sarcopenia in community dwelling middle-aged and old people	Dr Chun-Hung KO	Taiwan
15.10 - 15.19	FPOP1.6	Clinical characteristics of elderly hip fracture in Lerdsin Hospital, Thailand	Dr Thitinun ANUSORNVONGCHAI	Thailand
15.19 - 15.28	FPOP1.7	Evaluation of factors influencing bone strength measured by HSA	Dr Kosei YOH	Japan
15.28 - 15.37	FPOP1.8	A study of clinical, epidemiological and environmental risk factors associated with fragility hip fracture in elderly Asian Indians, aged ≥60 years	Dr Shweta VARSHNEY	India

 **FREE PAPER ORAL PRESENTATIONS 2**  
**Day 2 - Saturday (7th October 2017)**

Time	ID	Title	Name	Country
14.10 - 14.19	FPOP2.1	Role of Th-17 lymphocytes in estrogen deficiency mediated bone loss in post-menopausal osteoporosis	Dr Shah WALIULLAH	India
14.19 - 14.28	FPOP2.2	Osteoporosis management protocol increase the awareness of Fracture Liaison Service in orthopaedics department	Fadzleen JOHARI	Singapore
14.28 - 14.37	FPOP2.3	Involvement of lifestyle-related diseases in the development of fragility fracture of the proximal femur	Dr Takashi IWAKURA	Japan
14.37 - 14.46	FPOP2.4	Time to hospital presentation and time to operation among older hip fracture patients in a teaching hospital in Malaysia	Dr Poh Yong CHONG	Malaysia
14.46 - 14.55	FPOP2.5	Specialized hip surgeons and treatment of underlying disease after discharge reduces one-year mortality in elder hip fracture	Dr Chung-Hwan CHEN	Taiwan
14.55 - 15.04	FPOP2.6	Changes of periprosthetic BMD in proximal femur of patients with osteoporosis after cementless total hip arthroplasty	Dr Guangtao FU	China
15.04 - 15.13	FPOP2.7	Hi dietary salt intake induces bone loss in ovx mice by skewing Treg-Th17 balance	Dr Rupesh Kumar SRIVASTAVA	India
15.13 - 15.22	FPOP2.8	Effect of switching from bisphosphonates to denosumab on bone metabolism in aromatase inhibitor-treated breast cancer patients	Dr Masato SHIZUKU	Japan

3

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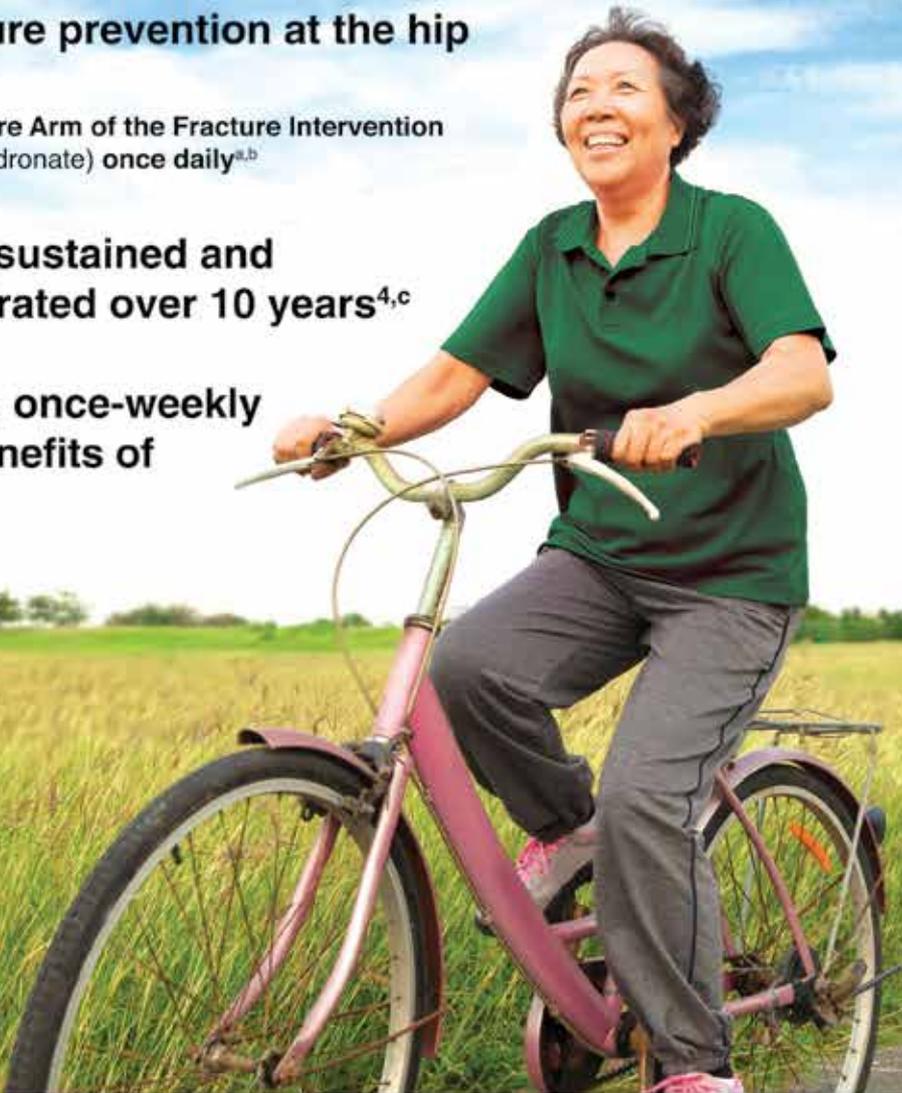


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As seen in the Vertebral Fracture Arm of the Fracture Intervention Trial (FIT) with FOSAMAX (alendronate) once daily<sup>a,b</sup>

### 2 Therapeutic effects sustained and tolerability demonstrated over 10 years<sup>4,c</sup>

### 3 A convenient single, once-weekly tablet with added benefits of 5600IU Vitamin D<sub>3</sub><sup>5</sup>



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<sup>a</sup> FIT was a double-blind, randomized, double-controlled, 3-arm, multicentre study of FOSAMAX (5 mg daily for 2 years and 10 mg daily thereafter) on the risk of vertebral and nonvertebral fracture in 659 postmenopausal women (aged 55 to 81 years). 207 of those women had pre-existing vertebral fractures identified on radiographs at baseline (Vertebral Fracture Study (VFS)). <sup>b</sup> The primary end point in the VFS was the occurrence of new radiographic vertebral fractures at 3 years, defined as a vertebral height reduction of 20% or more from baseline measured at least 4 mm. <sup>c</sup> Hip fracture risk results are based on a pooled data analysis of all women in the VFS and the 1401 women in the Clinical Fracture Study (CFS) who had osteoporosis at baseline. <sup>d</sup> Alendronate is bioequivalent to FOSAMAX PLUS<sup>®</sup> (alendronate) (70 mg) and FOSAMAX PLUS<sup>®</sup> (alendronate/cholecalciferol) (70 mg/5600 IU). The therapeutic equivalence of FOSAMAX 70 mg once weekly and FOSAMAX 10 mg daily has been demonstrated. <sup>e</sup> This long-term study was a third extension (3 years) of an initial 2-year, phase 3, randomized, double-blind, placebo-controlled, multicentre trial that examined the efficacy and tolerability profile of 10 years of continuous alendronate treatment (2 or 10 mg/day) in postmenopausal women with osteoporosis (N=224). The primary end point was the change in bone mineral density at the lumbar spine.

**References:** 1. Black DM et al. Fracture risk reduction with alendronate in women with osteoporosis: the Fracture Intervention Trial. *J Clin Endocrinol Metab*. 2000; 80(11):4115-4124. 2. Black DM et al. Randomized trial of effect of alendronate on risk of fracture in women with existing vertebral fractures. *Lancet*. 1996; 348:1533-1541. 3. Black DM & Thompson DE. The effect of bisphosphonate therapy on osteoporotic fracture in the vertebral fracture arm of the Fracture Intervention Trial. *Int J Clin Pract Suppl*. 1999; 101:46-50. 4. Bone HG et al. Ten years' experience with alendronate for osteoporosis in postmenopausal women. *N Engl J Med*. 2004; 350(12):1189-1199. 5. Malaysia FOSAMAX PLUS<sup>®</sup> Local Product Circular. LFC-MK0217A-T-020105.

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For Medical and Healthcare Professionals only



**PL003**

**Bone, genes and fractures: Update on genetics of osteoporosis**

**Peter R Ebeling**

*Department of Medicine, School of Clinical Sciences, Monash University, Clayton, Australia*

Osteoporosis is a common disease characterized by low bone mineral density (BMD) and microarchitectural deterioration, leading to increased fracture risk with consequently increased morbidity and mortality. The diagnosis of osteoporosis is made clinically either by the presence of a BMD T score  $\leq -2.5$  or by the presence of a fragility fracture. The heritability of BMD is 50-85%, while that of fragility fractures is less, at 25-68%.

Despite this high heritability of BMD, the 95 genes identified in the largest genome-wide association study (GWAS) to date can only explain 5.8% of the genetic contribution to BMD variability. Nevertheless several of the genes identified through GWAS may have important and, hitherto unexplained, roles in bone metabolism and may be potential targets for new anti-osteoporosis therapies.

Other genes have been identified by studying rare monogenic disorders [disorder (gene)] affecting bone such as osteogenesis imperfecta (COL1A1/COL1A2, 13 other genes), Cole-Carpenter syndrome (SEC24D, P4HB), Bruck syndrome (FKBP10, PLOD2), osteopetrosis (CLCN7, TNFSF11, 5 other genes), pycnodystosis (CTSK), sclerosteosis and van Buchem disease (SOST), high bone mass syndrome (LRP5), osteoporosis-pseudoglioma syndrome (LRP5), and Hadju-Cheney syndrome (NOTCH2).

The study of extreme cases of idiopathic osteoporosis has also identified new genes: juvenile osteoporosis (LRP5, DKK1, WNT3A), pregnancy and lactation-associated osteoporosis (LRP5, MTHFR), X-linked osteoporosis (PLS3), and early-onset autosomal dominant osteoporosis (WNT1).

Genetic factors may also be important in the pathogenesis of atypical femur fractures (AFF). The association of AFF with seven rare monogenic bone diseases, including five from above (COL1A1/COL1A2; CTSK; PLS3; LRP5; osteopetrosis genes) supports this hypothesis. In addition, a pilot study in 13 AFF patients and 268 controls identified a greater number of rare variants in AFF cases. A recent whole exome sequencing (WES) study in 3 sisters with AFF showed, among 37 shared genetic variants, a p.Asp188Tyr mutation in the GGPS1 gene in the mevalonate pathway, critical to osteoclast function, which is also inhibited by bisphosphonates. Two studies completed targeted ALPL gene sequencing. An ALPL heterozygous mutation was found in 1 case out of a cohort of 11 AFFs, whilst the second study comprising 13 AFF cases did not find mutations in ALPL. Targeted sequencing of ALPL, COL1A1, COL1A2, and SOX9 genes in 5 cases of AFF identified a variant in COL1A2 in 1 case.

Overall, a deeper understanding of bone biology has been obtained by these genetic discoveries, which may ultimately reveal both novel targets for anti-osteoporosis drugs, and the pathophysiological mechanisms involved in rare associations of anti-osteoporosis therapies such as AFF.

**PL005**

**Inflammatory disorders and the skeleton**

**Asso. Prof. Julie T. Li-Yu, MD, FPRA, MSPH**

*Osteoporosis Society of the Philippines Foundation, Inc.*

Inflammatory rheumatic disorders, most esp rheumatoid arthritis and spondyloarthropathies, cause significant alteration and imbalance in bone remodeling. In both rheumatoid arthritis and AS, the key players that contribute to osteoclastogenesis include interplay of varying pro-inflammatory cytokines, ie. TNF, RANKL, IL-1, IL-17, IL-6, in the maturation, survival, and function of osteoclasts. Newly identified mechanisms on role of autoantibodies (RF and ACPA), and microRNAs as well in RA, while HLAB27 as a genetic marker in AS has also been shown to induce production of cytokine IL-23. Not only do pro-inflammatory cytokines induce osteoclastogenesis, they likewise inhibit osteoblast differentiation. Local microenvironment and mechanical forces in the presence of inflammation results to varying effects on the bone – erosions, periarticular and generalized bone loss in RA, while promoting bone formation at enthesial and periosteal sites in AS. New data are also evolving on the role of Wnt and BMP signaling pathways not only in regulating osteoblast function but also implicated in bone loss in RA and bone formation in AS.



**PL006**

**Update on osteonecrosis of the jaw (ONJ)**

**Akira Taguchi**

*Department of Oral and Maxillofacial Radiology, Matsumoto Dental University, Shiojiri, Japan*

Many factors, such as systemic medication use like steroids, radiation to the jaws, bacterial, viral and deep fungal infections, direct chemical toxicity, trauma, oral ulceration with bone sequestration (OUBS), neuralgia-inducing cavitation osteonecrosis (NICO), osteomyelitis associated with sclerotic osseous disease and malignancies, have been well known as the causes of osteonecrosis of the jaw (ONJ). On the other hand, bisphosphonate (BP)- related ONJ was first reported in oncology patients receiving high dose BPs in 2003 and subsequently in osteoporosis patients receiving low dose BPs in 2004. Since oral surgical procedures, such as tooth extraction, are considered one of the major risk factors for ONJ, there is confusion among physicians, dentists, and patients particularly osteoporosis patients taking low dose BPs. Many papers about BP-related ONJ (BRONJ) have been published to date. Recent studies also have reported an association between ONJ and the antiresorptive therapy denosumab (Dmab; a RANKL-inhibitor). BRONJ and Dmab-related ONJ are together referred to as antiresorptive agent-related ONJ (ARONJ). The pathogenesis of ARONJ still remains unknown.

At 2016, Japanese position paper was revised to disseminate updated information about ARONJ. The international ONJ task force meeting established at 2012 had published consensus paper at 2014 and international recommendation for ARONJ at 2017. In this session, I will present the followings according to the summaries of recent position paper and consensus paper: oral condition specific for osteoporosis patients, definition, staging, and estimated incidence of ONJ, risk factors for ONJ, cooperation between physicians and dentists in the prevention of ONJ, and prevention strategies for ONJ.

International consensus paper as well as other position papers describes that elimination or stabilization of oral disease prior to initiation of antiresorptive agents and maintenance of good oral hygiene are effective for preventing ARONJ. I will present the effect of self-oral health care for the prevention of ARONJ using recent our RCT study.

**PL007**

**Tumor-induced osteomalacia**

**Weibo Xia**, Bo Wu, Juan Feng, Juan Du, Yue Chi, Yan Jiang, Ou Wang, Mei Li, Xiao-ping Xing, Xunwu Meng, Xueying Zhou

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Tumor-induced osteomalacia (TIO), which is usually associated with benign mesenchymal neoplasms, is a kind of metabolic bone disease characterized by hypophosphatemia, low serum 1,25-dihydroxyvitamin D concentrations and bone demineralization. The clinical and biochemical manifestations of TIO are similar to those of patients with autosomal-dominant hypophosphatemic rickets (ADHR), X-linked hypophosphatemic rickets (ARHR). The pathophysiology of TIO is the overproduction of fibroblast growth factor 23 (FGF23) from tumor which suppress renal brush border membrane sodium-dependent phosphate transporter and reduce the activity of renal 1 $\alpha$ -hydroxylase causing renal phosphate wasting and hypophosphatemia. The clinical manifestations often include bone pain, muscle weakness and skeletal deformities. TIO often manifests itself as nonspecific hypophosphatemia so that misdiagnosis and missed diagnosis often occur. Because of the small size, slow growth rate, wide distribution and nonspecific imaging manifestations, the localization of the responsible tumors is challenging. Fortunately, the employment of octreotide scintigraphy increase the detection rate of TIO greatly. And we successfully use the method to locate tumors in Peking Union Medical College Hospital. The most common cause of TIO is phosphaturic mesenchymal tumor (PMT) which is of bone (50%) or soft tissue (50%) origins. The causative tumors are more frequently located in lower extremities, craniomaxillofacial (mandible and maxilla, nasal sinus) and thorax region. Histopathologically, PMTs are composed of spindle-shaped or stellate cells with low nuclear grade embedded in a mesenchymal myxoid with “grungy” calcification. Bone-like structure and cartilage-like tissue is a frequent finding and the tumors are rich in prominent vascularity notably. To date, however, the genetic mechanisms underlying the pathogenesis of PMT remain obscure. Recent study demonstrate that FN1-FGFR1 fusion gene play an important role in tumorigenesis. And 20-60% FN1-FGFR1 fusion gene was identified in PMT. Complete surgical resection of causative tumors is the definitive treatment of TIO. After tumor resection, FGF23 level decreased, serum phosphorus level normalized and symptoms relieved. For patients without tumor complete resection or fail to localization of the tumors, medical therapy consisting of phosphorus supplementation and calcitriol is essential. In addition, the recently developed FGF23 antibody has the potential to normalize serum phosphorus level and relieve osteomalacia.



**PL008**

**Therapies past and present – Guiding future directions**

**Peter R Ebeling**

*Department of Medicine, School of Clinical Sciences, Monash University, Clayton, Australia*

Patients at high near-term risk for fracture including the elderly, women, and those with a recent vertebral or non-vertebral fracture, including hip fracture, represent an extremely vulnerable group in whom anti-osteoporosis treatment should be expedited. Unfortunately, despite the existence of effective treatments to reduce subsequent fractures, the majority of patients presenting with a minimal trauma fracture do not receive treatment.

The first-line anti-osteoporosis therapies are antiresorptive drugs, which reduce vertebral, non-vertebral and hip fractures. Bisphosphonates can be administered orally in daily, weekly or monthly doses. The HORIZON study evaluated the effect of treatment in patients with an increased risk of fracture immediately following a hip fracture, a period during which they are at highest risk. It demonstrated that an intravenous zoledronic acid infusion administered  $\geq 2$  weeks after a hip fracture resulted in hip BMD increases, significant reductions in subsequent vertebral, non-vertebral and clinical fracture risk and reduced mortality. An alternative to the bisphosphonates is the human monoclonal antibody against a potent osteoclast growth and differentiation factor, receptor activator of nuclear factor kappa-B ligand (RANKL), denosumab. This is also effective at reducing vertebral, non-vertebral and hip fractures and it is a first-line drug in some, but not all countries. The benefits of antiresorptive drugs in reducing fractures far outweigh the risks of rare associations, such as jaw osteonecrosis or atypical femur fractures. However, the use of antiresorptive drugs has declined in some countries (e.g. USA, Singapore) by 50% or more due to concerns about these associated rare adverse events.

Teriparatide has been the prototype anabolic drug to treat patients with severe osteoporosis and has usually been reserved for second-line treatment, following failure of antiresorptive drugs. Teriparatide treatment reduces vertebral and non-vertebral fractures. New anabolic agents, including abaloparatide and romosozumab, currently under development, also appear to be promising treatments for patients at high near-term fracture risk. These drugs increase bone formation and, like teriparatide, need to be followed by an antiresorptive drug to maintain anti-fracture efficacy. Sequential therapy with an anabolic drug followed by an antiresorptive drug appears to be promising, but not all reverse sequential therapies with an antiresorptive drug followed by an anabolic drug are effective at maintaining BMD (e.g. denosumab followed by teriparatide).

In conclusion, identification of patients at particularly high risk of fracture during the year after a fracture helps clinicians target appropriate treatment more precisely and cost-effectively. However, despite the availability of several anti-osteoporosis drugs that reduce vertebral, non-vertebral and hip fractures, the majority of patients presenting with a minimal trauma fracture are not being treated. This evidence-treatment gap is a global problem that now needs to be closed.

### **SA1.1**

#### **Calcium – What do we recommend our patients?**

**Natthinee Charatcharoenwithaya**

*Endocrinology and Metabolism, Department of Medicine, Thammasat University, Pathumthani, Thailand.*

Maintaining an adequate calcium intake is the fundamental step in the prevention and treatment of osteoporosis. However, the optimal intake of calcium is uncertain. Most osteoporosis guidelines recommend a daily calcium intake between 700 and 1,200 mg. All patients should be advised to get as much as possible of calcium from dietary sources particularly dairy product. Calcium supplementation should be considered in patients with inadequate dietary calcium intake.

The most widely used calcium supplements are calcium carbonate and calcium citrate. Calcium carbonate is the least expensive and contains up to 40% of calcium content. However, it may cause more gastrointestinal complaints including constipation and bloating than calcium citrate. Calcium citrate is more expensive than calcium carbonate and contains only 21% of calcium content. However, its absorption is not dependent on gastric acid and it may be less likely to cause gastrointestinal complaints. Calcium supplementation more than 500 - 600 mg/dose should be given in divided doses for optimal absorption. The total calcium intake (diet plus supplements) should not exceed 1,500 mg/day, because of lack of benefits beyond this recommended level and the possibility of adverse effects. Patients with a history of nephrolithiasis should be evaluated for hypercalciuria prior to starting calcium supplementation.

Although several clinical trials have reported a beneficial effect of calcium or calcium plus vitamin D on bone mineral density in postmenopausal women and older men with osteoporosis, the data on fracture risk reduction are inconsistent. In addition, it is difficult to differentiate the effect of calcium from that of vitamin D. A recent meta-analysis of randomized controlled trials of calcium plus vitamin D supplementation from the National Osteoporosis Foundation demonstrated a significant 15% reduced risk of total fractures (summary relative risk estimate [SRRE], 0.85; 95% confidence interval [CI], 0.73-0.98) and a 30% reduced risk of hip fractures (SRRE, 0.70; 95% CI, 0.56-0.87).

### **SA1.3**

#### **Role of macronutrients, functional food and beyond**

**Winnie Chee Siew Swee**

*Dept of Nutrition & Dietetics, International Medical University, Bukit Jalil, Kuala Lumpur, Malaysia*

Research in lifestyle approaches to build peak bone mass during growth to prevent osteoporosis and treat the disease later in life has gained momentum over the years. Nutrients beyond calcium and vitamin D appear to play an important role in bone health. Excessive, as well as insufficient, levels of retinol intake may be associated with compromised bone health. Deficiencies in vitamin B, along with the consequent elevated homocysteine level, are associated with bone loss, decreased bone strength, and increased risk of fracture. Deficiencies in vitamins C, E, and K are also associated with poor bone health. Beyond nutrients, the role of functional foods has become a niche area in bone health nutrition. Other than foods fortified with calcium, plant proteins and phytoestrogens were touted to promote bone health. Currently research in enhancers of calcium absorption such as prebiotic fibers and whey proteins have gained much attention. The use of sophisticated methodologies for evaluating the effectiveness for calcium absorption such as measures of bone quality, bone densitometry and stable isotopes further strengthen the evidence for bone health benefits. These findings highlight the importance of adequate nutrition in preserving bone mass and reducing the risk of osteoporosis and fractures.



**SB2.1**  
**Bisphosphonates - Short term and  
long term efficacy**

**Jawl-Shan Hwang**

*Division of Endocrinology and Metabolism, Department of Internal Medicine, Chang Gung Memorial Hospital, Taipei, Taiwan*

Osteoporosis is a major public health problem in many countries, as well as in Asia. It's the most common metabolic bone disorder, characterized by a decrease in bone mass and deterioration in skeletal microarchitecture, which lead to increased fragility and susceptibility to fractures. The fractures associated with osteoporosis have been proven to cause considerable disability, loss of quality of life and mortality. Patients who have one osteoporotic fracture are at increased risk for having another osteoporotic fracture develop.

Bisphosphonates are widely used for osteoporosis all over the world, bisphosphonates decreasing osteoclast differentiation, activity, and survival. These class medications strongly bind to the hydroxyapatite component of bone, and remain near the bone surface for months to years, inhibit bone resorption and improve the structural biomechanical properties of bone by increasing bone mass while maintaining bone microarchitecture. Most of the bisphosphonates have well-established antifracture efficacy in both men and menopausal osteoporosis women. Alendronate, risedronate, ibandronate, and zoledronic acid have been shown to be both effective and safe in the short-term trial and long-term beyond 3 years of treatment. No unexpected adverse events were identified in these studies and the long-term tolerability profiles of bisphosphonates remain favorable. Data from the withdrawal extension studies of alendronate and zoledronic acid also showed that residual fracture benefits were seen in patients who discontinued treatment for 3 to 5 years after an initial 3- to 5-year treatment period. The duration and potential discontinuation of treatment should be personalized for individual patients based on their response to treatment, fracture risk and comorbidities.

**SB2.2**  
**Therapeutics - Denosumab**

**Tai-Pang Ip**

*Department of Medicine, Tung Wah Hospital, Hong Kong*

Denosumab is a fully humanized monoclonal antibody which acts as a RANK ligand inhibitor with similar physiological actions as the endogenous osteoprotegerin. Not only does it inhibit the differentiation and maturation of the osteoclasts from its precursors, denosumab also inhibits the function and survival of the osteoclasts, thus achieving a very potent clinical antiresorptive effect at the skeleton. The 3-year double-blinded randomised control trial, the FREEDOM study, has demonstrated that denosumab significantly decreased the risk of vertebral, non-vertebral and hip fractures in postmenopausal osteoporotic women leading to its approval by the United States FDA in 2010 for the treatment of postmenopausal osteoporosis. It is subsequently also approved for use in men at high risk for fracture receiving androgen deprivation therapy for non-metastatic prostate cancer and in women at high risk for fracture receiving adjuvant aromatase inhibitor therapy for breast cancer.

Osteoporosis being a chronic disease logically requires long-term medical treatment. The long-term efficacy and safety of denosumab has been clearly demonstrated by its ten-year long-term follow up study showing a continual increase of bone mineral densities (BMDs) at both the spine and hip without a plateauing effect as shown by the hip BMD upon long-term alendronate treatment. The improvement in BMDs was paralleled by the clinical observation of the maintenance of antifracture efficacy.

Although serious adverse effects such as osteonecrosis of the jaw (ONJ) and atypical femur fracture (AFF) have also been reported with the use of denosumab, the incidences of such adverse effects are much lower as compared with those of bisphosphonates.

To balance the potential risks and benefits of long-term treatment, there has been a recommendation to have a drug holiday after five years of treatment with bisphosphonates in patients with a relative low risk of fracture. However there are fundamental differences in the mechanism of action between denosumab and bisphosphonates such that rapid bone loss would occur upon cessation of denosumab treatment. Based on the latest literature evidence, the notion of drug holiday with denosumab is considered inappropriate.

### **SB2.3**

#### **Anabolic therapies**

**Manju Chandran**  
*Singapore General Hospital*

Stimulating new bone growth requires an anabolic agent. Teriparatide (Recombinant human parathyroid hormone 1-34) has long been approved for the treatment of postmenopausal osteoporosis, osteoporosis in men and glucocorticoid-induced osteoporosis. Another PTH analog, rhPTH (1-84) is currently licensed by the FDA for the treatment of hypoparathyroidism only. PTH exerts its effects by binding to the parathyroid hormone receptor1 (PTH1R) expressed on osteoblasts, osteocytes and renal tubular cells. Constant activation of the receptor by parathyroid hormone as seen in patients with primary hyperparathyroidism results in bone loss. However, intermittent administration of PTH has been shown to exert anabolic effects on bones and this is the basis for its use in the management of osteoporotic bone loss. The N-terminal PTHrP analogue, Abaloparatide also activates the PTH1R. It has 76% homology to Parathyroid hormone related peptide (PTHrP 1-34). It has recently been approved by the FDA for the treatment of postmenopausal osteoporosis. It has been shown to reduce new morphometric vertebral fractures by 86% and non-vertebral fractures by 43% compared to placebo. The Wnt/Beta -catenin signaling pathway is critical in the bone mass regulatory pathway. It is under the influence of several stimulatory and inhibitory molecules. Attempts to block its inhibition through monoclonal antibodies has resulted in the development of the Sclerostin inhibitor -Romosozumab. It was recently considered for approval by the FDA based on the results of the FRAME study that showed compared to placebo a 73% reduction in the risk of new vertebral fractures and a 36% reduction in clinical fractures in postmenopausal women. In the BRIDGE study, administration of romosozumab in men with osteoporosis resulted in significant increases in BMD at the lumbar spine, total hip as well as the femoral neck. However, a higher rate of adjudicated cardiovascular adverse events was noted in the Romosozumab group compared to Alendronate in a phase 3 active comparator study. More data regarding this will be needed before approval for the use of Romosozumab in osteoporosis is granted by the FDA. Calcium-sensing receptor (CaSR) antagonists (Calcilytics) target the CaSR on the surface of the parathyroid cells and induce endogenous PTH production. They showed initial promise in animal studies as an alternative to PTH analogues, however they have not been proven to be useful to the treatment of osteoporosis in humans. This presentation will provide an overview of the currently available anabolic osteoporosis therapies as well as an update on agents currently in development and that hold potential for use in the treatment of osteoporosis.

### **SA3.1**

#### **Bone mineral density - Best practice**

**John J. Carey**  
*Galway University Hospitals, National University of Ireland, Galway, Ireland. President,  
International Society for Clinical Densitometry.*

Bone Mineral Density (BMD) is the best single surrogate of bone strength. BMD can be measured at various skeletal sites, performed by various technologies for over 100 years. Dual-energy X-ray Absorptiometry (DXA) was introduced in 1987, which soon became the accepted gold standard for non-invasive measurement of BMD. Soon after the society for clinical densitometry was formed, later becoming the International Society for Clinical Densitometry (ISCD) in 1993. The goal of ISCD was to establish and teach standards and best practice for measuring BMD and other densitometric parameters in clinical practice. Today BMD can be used to assess fracture risk, diagnose low bone mass or osteoporosis, assess prognosis, and monitor disease progression and/or response to therapy. Measurements must be accurate and reliable to have validity, and standards are in place which must be adhered to. In 1994 the World Health Organisation established DXA criteria for diagnosis of osteoporosis in postmenopausal women, using a standard deviation referenced scale. The ISCD have clarified, modified and adapted these criteria over time to enable practitioners to understand their strengths and limitations, how to apply them, and established criteria for other populations including men, premenopausal women and children. ISCD positions are published periodically and available on the website. ISCD states the preferred skeletal sites for measuring BMD in children are the whole body-less head and lumbar spine. In adults the recommended sites are total hip, femoral neck, lumbar spine (L1-L4) and in some instances distal 1/3 radius. Correct positioning of patients and correct use of technology, software and reference data are critical. A quality scan is the best way to assess fracture risk, make a diagnosis and establish a baseline for all subsequent scans. Repeated measures require further refinements and knowledge of the least significant change (LSC). Acceptable standards for LSC are <5.3% for the lumbar spine, <5.0% for total proximal femur and <6.9% for femoral neck. A recent publication by international authors for the ISCD outlines 14 best practice standards for DXA scan acquisition, analysis, interpretation and reporting which is available through the society's website.



### **SA3.2**

#### **Bone turnover markers in fracture risk prediction**

**Yong Jun Choi**

*Department of Endocrinology and Metabolism,  
Ajou University School of Medicine, Suwon, South Korea*

Bone turnover markers (BTMs) provide us with a noninvasive approach to studying bone turnover, and they can be measured easily and with good precision. N-terminal propeptide of type I procollagen (PINP) and C-telopeptide of type I collagen (CTX-I) are markers of bone formation and resorption, respectively, that are recommended for clinical use. However, these markers are subject to several sources of variability, including feeding (resorption decreases) and recent fracture (all markers increase for several months). BTMs increase at menopause and these higher levels are associated with more rapid bone loss. In some but not all studies, they are also associated with greater risk of fracture. However, the evidence base for use as predictors of fracture is not robust, and so BTMs have not been included in fracture prediction models. In people with osteoporosis, bone turnover markers might be useful to assess the response to anabolic and antiresorptive therapies, to evaluate compliance with treatment, or to indicate possible secondary osteoporosis. In untreated women, very high bone turnover marker concentrations suggest secondary causes of high bone turnover (eg, bone metastases or multiple myeloma). Much remains to be learned about how bone turnover markers can be used to monitor the effect of stopping bisphosphonate therapy (eg, to identify a threshold above which restarting therapy should be considered).

### **SA3.3**

#### **Clinical utility of TBS in management of osteoporosis**

**Yong Jun Choi**

*Department of Endocrinology and Metabolism,  
Ajou University School of Medicine, Suwon, South Korea*

Dual-energy X-ray absorptiometry (DXA) is indispensable for clinical practice in osteoporosis. DXA is the reference method for measuring bone mineral density (BMD) at the lumbar spine and proximal femur. However, bone strength mostly reflects the integration of bone density and bone quality. BMD accounts for only 70% of bone strength. Refinements in image quality, resolution and acquisition time, combined with more advanced computation power, have extended the utility of DXA from BMD to other functions. It can evaluate bone quality by indirect analysis of micro- and macro-architecture of the bone, which improves the prediction of fracture risk. TBS is one the most recently developed diagnostic tools using DXA that could be substantial in osteoporosis. TBS is a novel imaging technique, based on standard DXA images that could prove to be a useful index of bone texture to provide skeletal information in addition to the standard BMD results. TBS uses experimental variograms of 2D projection images, quantifying variation in gray-level texture from pixel to the adjacent pixels. TBS is not a direct measurement of bone microarchitecture but it is related to bone characteristics that include the trabecular number, trabecular separation, and the connectivity density. An elevated TBS indicates a robust and fracture-resistant microarchitecture. A low TBS reflects weak, fracture-prone microarchitecture. Another advantage of TBS is that it can be obtained by re-analysis of past lumbar spine DXA images without taking another scan. The usefulness of TBS in osteoporosis is becoming increasingly evident. Low TBS is associated with both a history of fracture and incidence of new fracture. The effect is independent of BMD and is of sufficient magnitude to enhance risk stratification with BMD. The effect is also partly independent of the FRAX fracture risk assessment tool, with likely greatest utility for those individuals who lie close to an intervention threshold. TBS adjusted FRAX probabilities were developed using the Manitoba data. Several smaller investigations have suggested a role for TBS in particular causes of increased fracture risk, such as glucocorticoid excess, type 2 diabetes, and rheumatoid arthritis.



### **SB4.1**

#### **Genetics and epigenetics of bone development**

**Ching-Lung Cheung**

*The University of Hong Kong, Pokfulam, Hong Kong*

The goals of genome-wide association studies (GWAS) are to discover new therapeutic targets of osteoporosis and improve our limited understanding of bone metabolism. For example, RANKL and SOST are susceptibility genes of osteoporosis, and monoclonal antibodies against these two genes have been developed as therapeutic targets of osteoporosis (denosumab and romosozumab). Although more than 60 susceptibility genes have been identified by GWAS, the role of these genes in bone metabolism remains large unknown. We recently performed candidate gene resequencing and identified novel rare variants of susceptibility genes in bone mass regulation, while in vitro and in vivo assays confirmed the involvement of the gene in bone development and mineralization. In addition to genetic variation, epigenetics also plays an important role in bone development. Non-coding RNA is abundant in the genome and involved in regulation of gene expression. In vivo and in vitro studies have demonstrated their roles in bone development, while circulating micro-RNAs may also involve in bone mass regulation in human. In this symposium, the role of genetics and epigenetics in bone development will be discussed.

### **SB4.2**

#### **Signal transduction involving bone cells coupling**

**Ling-Qing Yuan**

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The Second Xiangya Hospital, Central South University, Changsha, China*

Bone is constantly remodeled throughout life to maintain robust structure and function. Intercellular communication within the bone microenvironment is critical for the maintenance of normal bone structure. Bone remodelling requires cross-talk between different bone cells. Cytokines secreted by different bone cells or direct cell-cell physical engagement are involved in regulation of coupling of bone resorption and bone formation. Moreover, cytokine released from extracellular matrix also could regulate osteoblast-lineage cells and osteoclast-lineage cells. Recent study reveal exosome secreted by bone cells play an important role in regulating bone remodelling. Osteoporosis and other metabolic bone disease is a result of abnormality of bone remodeling. Reveal the cellular and molecular basis of bone remodelling and finding of novel paracrine or coupling factors, will lay a good foundation for drug development anti-bone disease.

### **SB4.3**

#### **Predictors of osteoporotic fracture risk**

**Hua Yue**

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With the process of aging society, the prevalence of osteoporosis increases year by year. As we all know that osteoporotic fractures have great harm with high mortality and disability rate. All our efforts have been made is try our best to reduce the incidence of osteoporotic fractures. Therefore, it is critical for us to find out osteoporotic fracture risk factors. The objective of this review (including our own study) was to identify risk factors for osteoporotic fractures. We included WHO-Fracture Risk Assessment Tool (FRAX®) and other predictors (BMD, prior fracture, bone geometry parameters and body mass, etc). In this review, some of the risk factors are known to contribute to long-term fracture risk, some factors predict fracture risk over a shorter time, such as over a 1-year period. Some clinical characteristics predict hip and non-vertebral fracture among elderly with osteoporosis, while other factors predict vertebral fracture more accurately. Assessment of these risk factors may help us to identify those osteoporosis patients with high fracture risk and help us to give them proper and accurate treatment.



**SA5.1  
Treatment failure in osteoporosis**

**Rong-Sen Yang**

*Professor and Chairman, Department of Orthopaedics, National Taiwan University & Hospital, Taipei, Taiwan*

Management of osteoporosis, especially for the elderly patients with associated fragility fractures, is an important issue worldwide. In a patient receiving treatment for 12 months at least, the criteria for good response include no new incident fracture, increased BMD and suppressed bone markers with anti-resorptive treatment. All these indicate a decreased fracture risk and the treatment should be maintained. Despite current effective anti-osteoporotic treatments, many patients are not managed appropriately, even following a fragility fracture. Ignorance of diagnosis, treatment and consequences, by physicians and patients, as well as poor adherence, remain important challenges for management of osteoporosis.

On the other hand, some patients with high compliance fail with available treatments. Treatment failure in osteoporosis was proposed by a working group in IOF as (1) two or more incident fragility fractures; (2) one incident fracture and elevated bone turnover markers at baseline with no significant reduction during treatment (with an antiresorptive), a significant decrease in BMD, or both; (3) both no significant decrease in bone markers and a significant decrease in BMD.

The causes of treatment failure remain uncertain, including poor bone responders and inappropriate treatment. Since no treatments can completely eliminate the risk of fragility fractures so that we may need to detect those with poor response to treatment before occurrence of new incident fragility fracture. First of all, adequate supplement of calcium and vitamin D3 can not be overemphasized. A comprehensive review of the treatment protocol, especially through the Fracture Liaison Service (FLS), can help much the adherence of treatment. Regular assessment of bone turnover markers and BMD may provide some clues to early detection of poor responders.

After optimizing the compliance and eliminating other causes of secondary osteoporosis, the working group in IOF recommends change of treatment. Some recommendations include shifting from a medication with weaker potency to a stronger one, from oral agent to parenteral one, or from an antiresorption to bone forming agent. A combination therapy does not reach consensus currently. However, the evidence is not solid. More comprehensive review and clinical studies are needed to address such issues of treatment failure in osteoporosis.

**SA5.2  
Bisphosphonate - Balancing risks & benefits**

**Atsushi Suzuki**

*Division of Endocrinology and Metabolism,  
Fujita Health University, Toyoake, Aichi, Japan*

Bisphosphonate (BP) is a potent anti-resorptive agent, and widely used in the treatment of diseases relating to bone loss including primary osteoporosis, Paget's disease and malignancy-associated hypercalcemia. As for anti-fracture effect, BP can decrease the risk of vertebral fractures (by 40% to 70%), hip fractures (by 20% to 50%), and nonvertebral fractures (by 15% to 39%) according to the clinical trials during 3 to 4 years in primary osteoporosis. In glucocorticoid-induced osteoporosis, many guidelines recommend that adults at moderate to high risk of fracture should be treated with oral BP for its high anti-fracture evidence and safety. Moreover, intravenous BP has been reported to decrease mortality rate after hip fracture.

Common adverse symptoms of BPs are upper gastrointestinal. However, most concern about the long-term BP therapy is considered over-suppression of bone remodeling. Several reports have documented that patients receiving BPs have higher risk of osteonecrosis of the jaw (ONJ) and diaphyseal and subtrochanteric femoral fractures called as atypical femoral fractures (AFF). The most ONJ cases have occurred in patients with cancer who received high doses of intravenous BP, and other medications including denosumab and anti-cancer agents are also associated with ONJ. Therefore, ONJ is not specific for BP, and such rare events are outweighed by fracture risk reduction by BPs especially in high fracture risk patients. The relative risk of patients with AFFs taking BPs is high, but the absolute risk of AFFs in patients on BPs is low, ranging from 3.2 to 50 cases per 100,000 person-years. The risk of AFF increases with BP therapy duration, and long-term BP use may be associated with higher risk of AFF (100 per 100,000 person-years). Therefore, A drug holiday of 2 to 3 years could be considered in low fracture risk patients after 3 to 5 years of BP treatment. BPs are still one of the first-line drug for osteoporosis treatment, but long-term BP use should be considered on balance between their anti-fracture efficacy and additional risk of AFF.



### **SB6.3**

#### **Nutrition and exercise: Maintaining intrinsic capacity**

**Prof. Alejandro V. Pineda, Jr., MD, DFM, FPAFP**

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Aging is a process which starts from the time we are born and as time passes. Up until physical maturity, anabolic processes surpass the catabolic actions of body cells. After the body reaches physiologic maturity, degenerative mechanisms prevail. A natural decrease in lean body mass is experienced as energy needs decline. Physical inactivity decreases energy needs even further, not only as a result of the reduced energy output but also because minimal physical activity leads to greater loss of lean body mass. More commonly, usual ageing predominates whereby age-related physiologic decline in bodily functions are observed. Up until physical maturity, anabolic processes exceed the catabolic processes of body cells. After the body reaches physiologic maturity, degenerative mechanisms prevail. Physical inactivity at this point decreases energy needs even further, not only because of the decrease in energy output but also because inactivity will cause greater loss of lean body mass.

SARCOPENIA has been described as a syndrome characterized by progressive, involuntary and generalized loss of muscle mass and strength that affect elderly adults. Primary Sarcopenia is age-related while secondary Sarcopenia can be activity-related, disease associated and nutrition-correlated. The diagnostic criteria set by the ESPEN Special Interest Groups require presence of low muscle mass (percentage of muscle mass > 2 SDs below the mean in individuals aged 18-39 in the National Health and Nutrition Examination Survey III Cohort) and walking speed <0.8 m/s in the 4-meter walk test or reduced performance in any functional test used to assess the geriatric population. The mechanisms that trigger Sarcopenia are continuously being explored but are likely to be multi-factorial. Intrinsic factors for development: metabolic resistance or endocrine dysfunction, oxidative stress, inflammation, impairment of  $\alpha$  motor neuron, and impaired satellite cell function. Extrinsic factors include: reduced physical activity, inadequate nutrition due to loss of appetite, depression, poverty, and isolation. Consequences of Sarcopenia can be anywhere from loss of functional status, increase fall risk, morbidity and mortality. Nutrient intake is said to be the most critical anabolic stimulus for skeletal muscle development. Resistance exercise is another anabolic stimulus that has been identified to increase muscle protein synthesis in younger and older individuals. Recent publications point to a cumulative effect of resistance exercises with nutrition supplementation on protein metabolism.

### **SB8.1**

#### **Bone and cancer – Fatal attraction**

**SP Chan**

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Bony metastases or “bone mets” occur when the primary malignant cells spread and relocate to bone. Bone metastases are common and have devastating effects on cancer patients. The most likely cancers to metastasize to bone are, prostate, breast and lung. 65-80% of metastatic breast and prostate cancers are to the bone. Once malignancies have metastasized to bone, they are generally incurable.

The bone microenvironment is a unique and fertile soil for cancer metastasis. There are components of the bone microenvironment that influence tumour localization; in addition, tumour-derived factors recruit osteoclasts and osteoblasts, modulate cellular and protein matrix components, resulting in the liberation of growth factors from the bone matrix, which then feedback to enhance tumour growth, expansion and invasion. This is the ‘vicious cycle’ of bone metastases.

In addition, bone marrow serves as a reservoir for dormant tumour cells that can resist chemotherapeutic attack, and these tumour cells will emerge later as full-blown metastases in bone or other organs.

Consequences of bone metastases are bone pain, pathological fractures and hypercalcaemia. Understanding the interaction between tumour and bone, has uncovered therapeutic opportunities, e.g. bisphosphonates and RANK-ligand inhibitors, for the prevention, treatment of bone metastases; as well as bone loss associated with chemotherapies for malignancies.



### SB8.2

#### **Cancer treatment induced bone loss – Size of the problem!**

**Matin Mellor Abdullah, MBBS (Mal) FFRRCS (Ire) AM (Mal)**

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Cancer and its treatment have significant effects on bone integrity. There are 3 distinct areas of interaction. Firstly, bone metastases are common in many solid cancers like breast, lung and colorectal cancers. Secondly, many cancer treatments adversely affect bone health that may cause fractures if left unattended. Thirdly, bone marrow micro-environment is involved in the metastatic process and emerging data suggest that the use of bone targeted treatment can reduce metastases to bones. Major cancer treatment modalities include surgery, chemotherapy, radiotherapy, hormonal manipulation, targeted therapies, immunotherapies and others. Chemotherapy, radiotherapy and hormonal manipulation are associated with significant bone loss that may lead to fractures that will significantly impair the patient's quality of life. Bone loss increases with age.

### SB8.3

#### **CTIBL – Prevention and management: New standard of care**

**Mastura Md Yusof**

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Osteopenia and osteoporosis are often long-term complications of anti-neoplastic treatments, defined as cancer treatment induced bone loss (CTIBL). CTIBL increases the risk for skeletal morbidity and impact negatively on the quality of life of oncology patients. As cancer cure rates increase with improved management, late toxicity such as changes in bone metabolism becomes increasingly relevant and challenging. Common cancers such as breast and prostate cancer are often treated with therapies that can adversely affect bone health. As cancer cure rates increase with improved management, the late toxicity of changes in bone metabolism becomes increasingly relevant and challenging. Hormone treatment such as gonadotropin-releasing hormone, luteinizing hormone-releasing hormone agonists, anti-androgens and aromatase inhibitors can cause significant reduction in the levels of estrogen or testosterone, the hormones responsible for bone mass maintenance in women and men respectively. Corticosteroids, effective for treatment of multiple myeloma increase the risk of fracture over that expected for any given bone mineral density. Chemotherapies, radiotherapy and targeted therapies in cancer may contribute to deregulate bone remodeling to some degree via different mechanisms. Improved understanding of the pathophysiology of CTIBL, streamlined risk assessment for identification of patients at increased risk for fragility fractures is essential. A multidisciplinary approach involving appropriate intervention that include both lifestyle modifications and tailored pharmacological strategies to prevent bone metabolism failure during anti-tumour treatments and survival period will reduce the risk for skeletal complications and thence improve quality of life. Among the bone modifying agents that are approved for hormone deprivation therapy induced osteoporosis are bisphosphonates, which reduces bone resorption by inducing osteoclast apoptosis and denosumab, the receptor activator of nuclear factor- $\kappa$ B ligand (RANKL) ligand. The efficacy and safety profile of these drugs have been established in large patient population.



**SA9.2  
SERMs**

**Dong Ock Lee**

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Ideal SERMs (Selective Estrogen Receptor Modulators) should be an ER (Estrogen Receptor) agonist in bone, vagina, urogenital atrophy, brain, heart and ER antagonist in breast, endometrium. But such an ideal SERM is not yet developed.

For osteoporosis, raloxifene and bazedoxifene are clinically available now, their use in postmenopausal women with osteoporosis increased BMD and prevent vertebral fractures. But they have a lack of efficacy in prevention of non-vertebral fracture. Osteoporosis is a chronic disease and patients should be treated for a long time. Safety of bisphosphonate after long-term use has been concerned recently. Specifically, atypical fracture after long-term use of bisphosphonate develop more frequently in Asians. So in the aspect of long-term treatment, younger postmenopausal women with low risk of femur fracture should be considered as a candidate of SERMs.

Raloxifene prevents invasive breast cancer in high risk patients and bazedoxifene reduced the incidence of endometrial cancer. Both drugs improve the lipid profile. Side effects include venous thromboembolism and stroke. Venous thromboembolism shows relatively low incidence in Asians and risk of stroke increase depending on the age.

Adequate candidates for SERMs especially in Asians will be reviewed.

**SA9.3**

**Hormonal therapies: Testosterone therapy**

**Bu Beng Yeap**

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and Department of Endocrinology and Diabetes,  
Fiona Stanley Hospital, Perth, Western Australia, Australia*

Older men have lower testosterone concentrations compared with younger and middle-aged men, and are the age-group most vulnerable to osteoporosis. Lower testosterone concentrations in older men are associated with increased fracture risk. Androgen deficiency should be investigated as an underlying cause of osteoporosis in men with suggestive symptoms and signs. In men with androgen deficiency due to disorders of the hypothalamus, pituitary and testes, testosterone treatment improves symptoms and signs of androgen deficiency and is expected to improve bone mineral density. In randomised controlled trials in older men with low-normal baseline testosterone concentrations without known pituitary or testicular disease, testosterone intervention improved sexual function and also improved bone density, estimated bone strength and haemoglobin concentrations, but its effect on fracture risk remains to be clarified. Testosterone treatment is indicated in men with pathological hypogonadism. Men at high risk of fracture should be treated with a pharmacological agent for osteoporosis with evidence for anti-fracture benefit.



### SB10.1

## Effectiveness and safety of denosumab for osteoporosis in patients with chronic kidney disease (CKD)

Yasuhiro Takeuchi

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Chronic kidney disease (CKD) at stage 3 and 4 is an established risk factor for fragile fracture. Elderly people are prone to develop not only osteoporosis but also CKD. Furthermore, both diseases are getting worse with advancing age. Therefore, elderly patients with osteoporosis are preferentially to be treated if they also have CKD at stage 3/4. Unfortunately, however, drug regulatory authorities caution physicians who prescribe several anti-osteoporosis drugs, such as bisphosphonates and anti-RANK ligand antibody (denosumab), to patients with impaired renal function, in particular stage 4 CKD. There are several possible mechanisms whereby bone integrity is deteriorated in patients with CKD. Among them, secondary hyperparathyroidism is important because it directly impairs bone metabolism and because it is sometimes difficult to be appropriately treated. Anti-bone resorptive agents including denosumab suppress calcium mobilization from bone to circulation and induce compensatory secretion of parathyroid hormone. Then, treatment of CKD patients with denosumab provokes not only immediate severe hypocalcemia but also augmentation of secondary hyperparathyroidism. Here I present clinical data of patients with CKD stage 3/4 who were treated with denosumab and would demonstrate preventive effects of active vitamin D drugs on secondary hyperparathyroidism as well as hypocalcemia. Based on our clinical observations, denosumab is an option for osteoporosis patients with CKD stage 3/4 when treated along with active vitamin D with strict and careful attention.

### SB10.2

## Guidelines for the management of glucocorticoid-induced osteoporosis

Tien-Tsai Cheng

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Taiwan*

Glucocorticoids use can lead to several well-known adverse events. Osteoporosis is one of the complications of glucocorticoid therapy and is associated with subsequent fracture. Although awareness of glucocorticoid therapy induced osteoporosis grown recently, it remains under-diagnosed and under-treated by health-care providers. Glucocorticoid-induced osteoporosis (GIOP) has several unique characteristics; including rapid bone loss and increased fracture risk occur soon after therapy is initiated. GIOP is second in frequency only to the osteoporosis that occurs after menopause and is the most common form of drug-induced osteoporosis. GIOP is also one of the most devastating complications of prolong glucocorticoid therapy in rheumatoid arthritis (RA). Primary prevention of fracture in individuals on high dose and long-term glucocorticoid therapy is essential.

Several available guidelines for the management of GIOP had been developed. In 2017, the American College of Rheumatology (ACR) revised its 2010 recommendations to incorporate advances in risk assessment and to include all currently approved treatments. In 2012, the International Osteoporosis Foundation (IOF) and the European Calcified Tissue Society also provided a framework for the development of guideline from which country-specific recommendations could be derived. The framework covered the management of GIOP in men and women aged 18 years or over, in whom continuous oral glucocorticoid therapy at any dose is considered for 3 months or longer. It had been well recognized that guidance should be varied between countries because of differences in resources, availability and cost of treatments and health care policies. In the presentation, we will demonstrate the Taiwan's guideline, endorsed by Taiwan Osteoporosis Association (TOA), for management of GIOP.

### SB10.3

## Pregnancy induced osteoporosis

Dr Alexander Tan

*Associate Professor & Consultant Endocrinologist, Head of Diabetes Care Unit, University of Malaya Medical Centre*

The physiology of pregnancy and lactation increases the risk of net bone resorption and in a very small number of women, this results in severe osteoporosis with one or more vertebral compression fractures. Pregnancy-lactation associated osteoporosis (PLO) usually presents post-partum or in the third trimester. As the vast majority of pregnant women do not have baseline bone mineral density evaluations, it is unclear if PLO occurs in women who already have low bone mineral density prior to pregnancy or due to an exaggerated bone loss during pregnancy and lactation. Several mechanisms have been proposed including high fetal calcium demand, calcium loss in breast milk and increased parathyroid related peptide secretion by the placenta and breast. The use of anti-osteoporotic agents in pregnancy and lactation is controversial. This lecture will present a case of pregnancy-lactation associated osteoporosis and discuss the risk factors and management.

## **SA11.2 Hip fracture - Typical and atypical**

**Angela Wing-Hang Ho**  
Caritas Medical Centre, Hong Kong

Fragility fracture is one of the common chronic diseases in geriatrics. The prevalence of femoral neck osteoporosis based on a hip T-score of  $< -2.5$  was 47.8% in males and 59.1% in females in our previous study of 239 hip fractures. The incidence of hip fracture increases with age, and the incidence is high in the elderly (1639 per 100 000 in men and 3012 per 100 000 in women for the age-group of  $\geq 85$  years). A 2015 study of geriatric hip fracture showed that there was a steady increase in the incidence of geriatric hip fracture in Hong Kong. The overall 30-day and 1-year mortalities were 3.01% and 18.56%, respectively. Advancing age and male sex were associated with an increased mortality and a higher excess mortality rate following surgery.

One uncommon but alarming side effects of anti-resorptives is atypical fracture. Surgical treatment of atypical fracture is difficult, eg difficult reduction and complicated union. Prevention measures such as surveillance, patient education and prophylactic fixation will also be reviewed.

## **SA11.3 Fracture fixation with augmentation in osteoporotic fracture**

**Pol.Col.Lt. Dr. Tanawat Amphansap**  
*Police General Hospital, Bangkok, Thailand*

There are three key elements to be considered when defining osteoporosis: Bone mass, Loss of bone mass and Microstructural changes. The pattern of age-related cortical bone loss involves decreases in cortical thickness, bone mineral content and cortical bone density with concomitant increase in medullary diameter and also increase in cortical porosity. The osteoporotic bone has thinner trabeculae with fewer interconnections. Many of the interconnections may have become broken with time. Clinically, implants may “cut through” the softer bone in metaphyseal areas or may get loose and fail. It is difficult to produce secure fixation of the implant to the bone. The common mode of failure of internal fixation is bone failure rather than implant breakage. Furthermore, the healing process may be prolonged because of decreased healing capacity in osteoporosis.

The specific implant feature for osteoporotic bone is angular stability. Angular stability can now be achieved with plates and nails. 1) Locking Compression Plate with combination hole consists of two proven elements: One half of the hole has the design of the dynamic compression unit for conventional screws. One half is conical and threaded to accept the matching thread of the locking head screw providing angular and axial stability. The fixed angle created between the plate and screw is not disturbed by poor bone quality. 2) Angular Stable Locking System (ASLS); This new system allows for the first time angular stable interlocking of intramedullary nails.

The most important rules of fracture fixation in osteoporotic bone are Relative rather than absolute stability; Indirect rather than anatomical reduction; Locked splinting with long plates or nails; Load distribution; No interfragmentary compression; Secondary bone healing; and no mixture of principles and methods.

The use of bone cement augmentation has been reported for plate, screw and nail osteosynthesis in elderly patients that demonstrating increased bone-implant interface, improved implant anchorage, reduced screw cut-out and improved early full-weight bearing. Properties for an ideal bone substitute include: void filling capacity, structural support, osteoconductivity, osteoinductivity, osteogenicity, minimal morbidity, cost-effectiveness and unlimited availability. There is currently no bone substitute that fulfills all of these requirements.



### MTE001

#### Physical activity: What works, what's safe

**Prof. Alejandro V. Pineda, Jr., MD, DFM, FPAFP**

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OSTEOPOROSIS as defined by WHO is a systemic skeletal disease characterized by low bone mass and micro-architectural deterioration of bone tissue, with a consequent increase in bone fragility and susceptibility to fractures. The condition is characterized by progressive loss of bone density and quality, thinning of bone, increased susceptibility for fracture (fragility), and a DEXA Scan T score  $\leq -2.5$ . Numerous evidence point to the critical importance of physical activity to bone health in primary (at risk for) and secondary (treatment) prevention of osteoporosis. Indulgence redounds to the following benefits: reduces risk for osteoporotic fractures; enhanced bone peak mass; slowed rate of bone loss and decline with aging; reduced risk of falls by improving muscle strength and balance.

The Consensus Statements on Osteoporosis Diagnosis, Prevention, and Management in The Philippines 2010 explored responses to several questions. Q2: What forms of exercise can improve bone density? Tai Chi Chun (TCC) is primarily for balance, muscle strength, fall prevention, flexibility, and performance of activities of daily living. (However, there is insufficient evidence to recommend Tai-Chi for the prevention of osteoporosis.) Supervised high intensity resistance training such as muscular strength training loads consisting of single or multiple sets of 8-12 repetitions of exercises done 2-3 days/week is recommended for the prevention of osteoporosis. It is suggested that exercise therapy, e.g., aerobics, resistance, and weight bearing exercises, and walking be regularly done to increase bone density at the lumbar spine and hip. Both Tai Chi and resistance exercises maybe incorporated in regular community center activities. Q19: What form of exercise should be recommended among the high risk individuals to increase BMD and decrease future fracture risk? It is suggested that exercise be encouraged among both the housebound elderly and those in the community due to its benefit on balance and indirectly on fracture prevention. A 3x a week, 6 month Tai Chi Chuan exercise program can be considered an option in enhancing an individual's balance and prevention of fall. Exercise appears to have statistically significant beneficial effects on balance ability compared to usual activity. Interventions including gait, balance, coordination, functional exercises; muscle strengthening; and muscle exercise types appear to have the greatest impact on indirect measures of balance.

### MTE004

#### Monitoring response to treatment - To scan or not to scan

**Chanika Sritara**

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Mahidol University, Bangkok, Thailand*

Bone mineral density assessment by dual-energy X-ray absorptiometry (DXA) is the most widely available and best validated clinical tool to diagnose osteoporosis, assess fracture risk, and monitor the skeletal effects of medications that reduces fracture risk (1). For the monitoring purpose, the ISCD recommends the use of value of least significant change (LSC), which is the upper limit of the 95%CI of the root mean square of standard deviation (RMS SD) or coefficient of variation (RMS CV). The change in the BMD greater than this value is regarded as significant. In addition, the ISCD suggests the monitoring time interval (MTI) using the formula:

$MTI \text{ (years)} = LSC / \text{expected change per year}$

However, the American College of Physicians (ACP) recently published a weak recommendation against bone density monitoring during the 5-year pharmacologic treatment period for osteoporosis in women based on low-quality evidence (2).



## MTE005

### Glucocorticoid-induced osteoporosis – Difficult cases

Swan Sim YEAP

*Subang Jaya Medical Centre, Selangor, Malaysia*

Glucocorticoid-induced osteoporosis (GIO) is one of the most common causes of secondary osteoporosis. This session will discuss some of the management issues when dealing with patients. Below are some useful references on the management of GIO:

1. Buckley L, Guyatt G, Fink HA, et al. 2017 American College of Rheumatology Guideline for the Prevention and Treatment of Glucocorticoid-Induced Osteoporosis. *Arthritis Care Res (Hoboken)* 2017 Aug; 69(8): 1095-1110. doi: 10.1002/acr.23279.
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## MTE006

### Application of body composition assessment by DXA in chinese patients with HIV/AIDS

Wei Yu, JingPeng Yao, Xiaohong Zhou, Mengtao Sun, Taisheng Li

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Body composition consists mainly of fat, lean, as well as bone mineral content. A variety of techniques are available for evaluating the changes of different body composition respectively, such as Dual energy X-ray absorptiometry (DXA), Bioelectrical impedance analysis (BIA), computerized tomography (CT) and magnetic resonance imaging (MRI) etc.

Acquired Immune Deficiency Syndrome (AIDS) is caused by Human Immunodeficiency Virus (HIV). Subjects who are infected with HIV may develop AIDS over a period of years. Although the treatment with highly active antiretroviral regimens (HAART) has led to reductions in the development of HIV-related illness and increases patients survival time remarkably, it has also led to metabolic changes in the patients. Changes in body composition for HIV-infected patients may occur and are associated with important effects during the treatment of highly active antiretroviral therapy (HAART). Also, some of the changes included lipodystrophy, sarcopenia, as well as changes of both bone mass and bone turnover, which have been reported as a consequence of HIV infection itself. Early detection for these changes has become increasingly important for HIV-infected patients. Authors will review some evaluating methods for body compositions changes, including fat, lean mass and bone mass, and focus on the application Dual-energy X-ray absorptiometry (DXA) in some pilot studies for Chinese HIV-Infected patients. Further discussing some features of the body composition changes in Chinese HIV-Infected patients have indicated that applying DXA to assess HIV-infected patient's body composition changes could provide objective information for physicians to prevent lipodystrophy, sarcopenia as well as osteoporosis.



**MTE007**  
**ONJ- International consensus**

**Akira Taguchi**

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Bisphosphonate (BP)-related osteonecrosis of the jaw (ONJ) was first reported in oncology patients in 2003 and subsequently in osteoporosis patients in 2004. Since oral surgical procedures, such as tooth extraction, are considered one of the major risk factors for ONJ, there is confusion among physicians, dentists, and patients particularly osteoporosis patients taking low dose BPs. Recent studies also have reported an association between ONJ and the antiresorptive therapy denosumab (Dmab; a RANKL-inhibitor). BP-related ONJ (BRONJ) and Dmab-related ONJ (DRONJ) are together referred to as antiresorptive agent-related ONJ (ARONJ).

Many papers regarding ARONJ have been published to date. However, the pathogenesis of ARONJ still remains unknown. The international ONJ task force members were selected and gathered from 12 countries at 2012 to update previous guidelines as well as the ASBMR 2007 Task Force report and oral surgery recommendations with an evidence-based review. This task force was supported by National and International societies including; CAOMS, OC, ASMBR, AAOMS, ECTS, The Endocrine Society, IOF, ISCD, IBMS, IAOMS, PAOS, FIRMO, JSBMR. In the first meeting that was held in Minneapolis, Minnesota, the international ONJ task force had listed 9 questions regarding ARONJ that should be clarified as soon as possible. Based on these 9 questions, international consensus paper was published online on JBMR at the end of 2014. However, there were differences in some issues regarding ARONJ among international consensus paper and other position papers such as American Association of Oral and Maxillofacial Surgeons (AAOMS) position paper 2014 revision. AAOMS position paper 2014 seems to be widely believed and used in Asian countries.

In this meet-the-experts session, I will consider these differences based on my own ARONJ cases. Especially, I will focus on the definition, staging, early sign on imaging for stage 0, true condition (ONJ or osteomyelitis), and merit of drug discontinuation before tooth extraction. I would like to discuss with the audience about these issues.

**MTE009**  
**Andropause – Debunking the myth**

**Bu Beng Yeap**

*School of Medicine, University of Western Australia,  
and Department of Endocrinology and Diabetes,  
Fiona Stanley Hospital, Perth, Western Australia, Australia*

As men grow older, circulating testosterone declines while ill-health accumulates. In epidemiological studies, lower testosterone concentrations are associated with poorer health outcomes including frailty, osteoporosis, cardiovascular disease and mortality. Higher concentrations of the bioactive metabolites of testosterone, dihydrotestosterone and estradiol, are associated with longer leucocyte telomere length in men, indicative of slower biological ageing. Therefore the question arises as to whether lower concentrations of testosterone or its bioactive metabolites might be contributory factors to, as well as biomarkers for, ill-health in ageing men. In middle-aged and older men, obesity and medical comorbidities are closely associated with lower circulating testosterone and loss of excess weight results in increased testosterone concentrations. However, in the oldest old men, there is a progressive impairment of testicular endocrine function as evidenced by progressive increases in luteinising hormone correlated with decreases in testosterone and dihydrotestosterone. In randomised controlled trials in older men with low-normal baseline testosterone concentrations, testosterone intervention improved sexual function and also improved bone density, estimated bone strength and haemoglobin concentrations. Testosterone treatment is indicated in men with symptoms and signs of androgen deficiency due to disorders of the hypothalamus, pituitary or testes. Further research is needed to clarify the benefits and risks of testosterone treatment in older men with lower testosterone concentrations in the absence of pathological hypogonadism.

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References: 1. Vitamin B2 is needed for the release of energy from protein, fats and carbohydrate. 2. Protein helps build and repair body tissues. 3. Vitamin D is necessary for the absorption and utilization of calcium and phosphorus. 4. Edington J, Barnes R, Bryan F et al. Clinical Nutrition (2004) 23, 195-204. (Handgrip strength) \* Individual results may vary. † Compared with previous formulation.

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ID	Title & Authors
PC01	A review of osteoporosis treatment with strontium ranelate in Hospital Melaka <b>Sahadun MAA</b> , Asikin MZ, Teo GH, Minghat AH, Sivapathasundaram N
PC02	Inadequate Vitamin D level: Association with low energy fractures of distal radius in young patients and its predictors in Karachi Pakistan <b>Muhammad M</b> , Ranjeet K, Muhammad Saeed M, Syed Jahanzeb, Vinod K, Anisuddin B
PC03	Determinants of insulin resistance and bone mass: Area, strength, or composition of muscle? <b>Lim SK</b> , Park HS, Lim JS
PC04	Fracture Liaison Service in Ajou University Hospital <b>Chung YS</b> , Won YY
PC05	Risk factors analysis in postmenopausal osteoporosis fragility fracture patients after treated with zoledronic acid <b>Fu GT</b> , Ding Y, Liu W, Hu XM, Li CC, Li CH
PC06	A case of vertebra fractures during weekly PTH therapy; to stop fracture-domino <b>Otsuka Y</b> , Otsuka A, Suzuki N, Otsuka S, Iwata T, Endo K, Otsuka Y
PC07	The workloads and job satisfaction of a fracture liaison nurse <b>Hsieh MH</b> , Liu CY, Lin CY, Yang CC, Chien JT
PC08	Effect of sarcopenia and central obesity on nutritional status in the elderly <b>Chang CS</b> , Chang YF, Liu PY, Wu SJ, Chiu CJ, Chen CY, Wu CH
PC09	Clinical results of combination therapy with teriparatide and denosumab <b>Yuki I</b> , Murase S, Iwasaki H, Takaha Y, Tamagawa Y, Tanigawa H
PC10	Effect of denosumab on bone formation marker P1NP <b>Tanigawa H</b> , Imoto Y, Nishiwaki S, Tamagawa Y, Imai S
PC11	Vitamin D, body fat and their associations in a healthy Malaysian population <b>Yeap SS</b> , SC Thambiah, IN Samsudin, SY Zahari-Sham, N Zainuddin, S Mohamad-Ismuddin, N Shahifar, S MD-Said, K Feiza, Lim HK, Lee LF, Lim J, Hew FL
PC12	Vitamin D deficiency and possible link with bony pain and onset of osteoporosis <b>Ghazi MA</b> , Ali SK, Najjad KR, Siddiqi MA, Taufiq I
PC13	Osteonecrosis of the jaw and bilateral atypical femoral fracture both occurring during treatment for osteoporosis <b>Payumo EF</b> , Crisostomo TD, Cudal ID
PC14	Characteristics of falls and fall-related fractures in hospitalized patients <b>Won YJ</b> , Kim YM, Kim SW, Kim BR, Kim JG, Kim SW
PC15	Calculation of free and bioavailable Vitamin D and its association with bone mineral density in Malaysian women <b>Yeap SS</b> , SC Thambiah, Wong TH, E Das Gupta, AK Radhakrishnan, Gun SC, G Chembalingam



<b>ID</b>	<b>Title &amp; Authors</b>
PC16	Osteoporotic awareness in patients with proximal femur fractures <b>Maniventhan N, Noor Mahazrinna, Vicknesh</b>
PC17	Alkaline phosphatase is a predictor of BMD in postmenopausal females <b>Saba T, Sundus T</b>
PC18	Interplay of Vitamin D, Vitamin B12, Homocysteine and bone mineral density in postmenopausal females <b>Sundus T, Saba T, Khalid PL</b>
PC19	Big data and osteoporosis: Using information mining to gather representative diagnostic data <b>Yan YH, Lin GL, Liu YT, Chang ST, Hong JJS</b>
PC20	Positive effect of Strontium Ranelate in complex non-union femur fracture <b>Suresh SR, Nadzirah HA</b>
PC21	Osteoporosis was considerably underdiagnosed in women: A single-center study from Taiwan <b>Yan YH, Lin GL, Chang ST, Liu YT</b>
PC22	Efficacy of Zoledronic Acid in post menopausal osteoporosis treatment <b>Shah W, Vineet S, RN Srivastava, AA Mahdi, Yashodhara P, Santosh K</b>
PC23	Trabecular Bone Score (TBS) and fracture risk prediction in Asian Type 2 diabetic subjects <b>Yeap SS, Lee LF, Feiza K, Hew CKT, Lim HK, Lim J, Hew FL</b>
PC24	Association of early hysterectomy as a risk for fracture in young females <b>Siddique T, Gillani SFuHS, Durrani AA</b>
PC25	Prevalence of osteoporosis in males: A retrospective study in a tertiary referral center <b>Esquibel MIL, Li-Yu JT</b>
PC26	Fracture Liaison Services improve outcomes of patients with osteoporosis-related fractures: A systematic literature review and meta-analysis <b>Wu CH, Tu ST, Chang YF, Chan DC, Chien JT, Lin CH, Sonal S, Manikanta D, Chen JF, Tsai KS</b>
PC27	Surgical outcomes in nonagenarians following hip fracture <b>Liu HC, Lin WT</b>
PC28	What makes your bones brittle? Beverages for bone health—a systematic review <b>Chang HJ, Lin IC, Chang YF, Wu CH</b>



## POSTER ABSTRACTS (ORTHOPAEDIC)

ID	Title & Authors
PO01	Complicated osteoporotic spinal fractures with neurologic compromise: Its clinical characteristics and clinical outcomes <i>Kim YH, Ha KY, Kim SI, Park HR</i>
PO02	Cervical spine myelopathy and osteoporosis <i>Truc Vu, Si Vo</i>
PO03	Risk factors including age, gender, osteoporosis in spine or hip, and local osteoporosis for more severe pattern of fracture in proximal humerus fracture <i>Kim JH, Koh KH</i>
PO04	Pharmacologic treatment improved mortality and quality of life in osteoporotic hip fracture without increasing cost: 12 months follow-up <i>Ong-art P</i>



## POSTER ABSTRACTS (BASIC SCIENCE)

ID	Title & Authors
PB01	Activation of ASIC1a by acidosis increases osteoclast migration and adhesion by modulating integrin/Pyk2/Src signaling pathway <i>Yuan FL, Li X</i>
PB02	<i>Lactobacillus acidophilus</i> enhances trabecular and cortical bone microarchitecture in ovx mice via modulating host immune system <i>Dar Hamid Y, Srivastava Rupesh K</i>
PB03	Development of a new osteoclast inhibitor targeting integrin-osteopontin interaction <i>Lee SY, Park D</i>
PB04	Liposomal encapsulation for systemic delivery of propranolol via transdermal iontophoresis improves bone microarchitecture in ovariectomized rats <i>Chen CH, Teong B, Lin SY, Huang HH, Ho CJ</i>
PB05	The effect of anti-osteoporosis treatment on titanium particle-induced osteolysis in osteoporotic mouse calvarial model <i>Fu GT, Ding Y, Li CC, Liu W, Peng P</i>
PB06	Exogenous PTHrP repairs the damaged fracture healing of osteoporotic mice and accelerates fracture healing of wild mice <i>Wang YH, Lin H</i>
PB07	Role Of miR-672-5p in postmenopausal induced osteosarcopenia and its mechanism of action in sustaining musculoskeletal health <i>Ahmad N, Trivedi R</i>
PB08	<i>Marantodes Pumilum</i> leaves increased callus volume and connective density of osteotomized tibia in osteoporosis rat model <i>Tijjani RG, Ahmad NS, Ima Nirwana S, Norliza M, Jamia Azdina J, Norazlina M</i>



ID	Title & Authors
PM01	Effects of epidural steroid injections to bone mineral density and anti-osteoporotic medications <i>Lee JH, Nah SY</i>
PM02	Use of denosumab in a rheumatology centre <i>Hazlyna B, Ch'Ng SS, Abidin MZZ, Zain MM, Lau IS</i>
PM03	Disuse osteoporosis in heterotopic ossification resection complicated with femoral neck fracture: A case report <i>M Rajaei AMZ, Sahran Y, Faisham WI</i>
PM04	Identification of novel Vitamin D receptor gene mutations in Chinese sporadic patients with Paget's disease of bone <i>Yue H, Zhang ZL</i>
PM05	Decreased risk of fracture in patients receiving traditional chinese medicine for osteoporosis: A Taiwan nationwide population-based cohort study <i>Hsieh MH, Yeh MH, Cheng CY, Chen WJ</i>
PM06	The relationship between coronary atherosclerosis in coronary computed tomography angiograph and Vitamin D level in health check- up <i>Yu BY, Lee AY</i>
PM07	Implant of choice for osteoporotic basicervical femoral neck fracture in a young patient with demineralised bone disease <i>Choo PT, Kasim NS, Chopra S</i>
PM08	Quetiapine ameliorates bone resorption in collagen - induced arthritis in mice via the suppressions of the AKT and ERK signaling pathways <i>Wang WH, Huang JH</i>



## Relationships Between Different Types Of Physical Activity And Metabolic Syndrome

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### INTRODUCTION:

Metabolic syndrome (MetS) is a clustering of at least three of the five following metabolic disturbances: abdominal obesity, elevated blood pressure, elevated fasting blood glucose, high triglycerides, and low high-density lipoprotein cholesterol. MetS and its components are related to the risk of developing cardiovascular disease, diabetes, and several cancers. In Taiwan, encouraging workers' physical activity is one of the main workplace-health-promoting programs to prevent MetS. We hypothesized that not only leisure-time PA but also occupational and commuting PA at an appropriate intensity may reduce the risk of MetS and its components. The aim of this study was to investigate the relationships of different physical activity types and metabolic syndrome in workers.

### MATERIALS & METHODS:

In this cross-sectional study, 3296 Taiwanese workers aged 20 years and above were enrolled. A self-reported questionnaire was used to assess nutritional health behavior and PA levels related to occupation, leisure time, and commuting. Blood pressure, anthropometric measures, and biochemical determinations of the blood were also obtained.

### RESULTS:

Multiple logistic regression was used to evaluate the adjusted odds ratios (ORs) and 95% confidence intervals (CI) of MetS and its components associated with different types of PA. The prevalence of MetS was 16.6% in workers. Compared with a low level of leisure-time PA, a high level of leisure-time PA showed a significantly lower risk of high triglycerides (OR 0.73, 95% CI 0.61–0.87) and MetS (OR 0.76, 95% CI 0.62–0.95). Compared with a low level of occupational PA, a high level of occupational PA represented a significantly lower risk of both abdominal adiposity (OR 0.64, 95% CI 0.49–0.84) and high triglycerides (OR 0.71, 95% CI 0.55–0.90).

### DISCUSSIONS AND CONCLUSIONS:

This study suggests that high levels of leisure-time were associated with reduce risk of MetS. Moreover, we emphasize that not only leisure-time PA but occupational PA could be important for the prevention of MetS. Therefore, it is recommended that leisure-time and occupational PA should be considered an essential part of workplace health promotion and may reduce entire cluster of metabolic risk factors. Not only leisure-time PA but occupational PA could be important for the prevention of MetS. Leisure-time and occupational PA should be considered an essential part of workplace health promotion for reducing risk of MetS.

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## What Makes Your Bones Brittle? Beverages For Bone Health—A Systematic Review

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 2Department of Family Medicine, National Cheng Kung University Hospital, No. 138, Shengli Road, Tainan, Taiwan

### INTRODUCTION:

Osteoporosis is an asymptomatic disease that causes bone loss, which leads to fractures and further public health burden. Coffee, tea and carbonated beverages are the most popular non-alcoholic beverage in the world. The association of these beverages with bone health has been investigated for more than two decades but yielded inconsistent conclusions. In the presentation, we performed updated systematic review with stricter clinical and methodological considerations to explore the efficacies of coffee, tea and carbonated beverages on bone health.

### MATERIALS & METHODS:

We searched MEDLINE-ORIGINAL, Cochrane CENTRAL databases and Chinese Electronic Periodical Services, aiming specifically at randomized controlled trials (RCT) and people with the diagnosis of osteoporosis or osteopenia. Search terms included both scientific and common names of coffee, tea, caffeinated beverages and carbonated beverages. The screening of the included studies, data extraction, critical appraisal and software followed the standards of Cochrane Collaborations, and was performed by two independent reviewers.

The primary outcomes were any effects of bone health, including bone mineral density and bone turnover markers, with secondary outcomes of any other benefits, in order to examine the effect as broad as possible. Other outcomes included adverse events of beverages. We would not perform meta-analysis if the outcomes were not reported or inappropriate to be analysed.

### RESULTS:

We initially identified a total of 25 reports addressing the topic. After full-text inspection and de-duplication, only one study met the criteria for analysis. Because it is meaningless to perform meta-analysis, we performed critical appraisal and narrative review of other related studies.

### DISCUSSIONS:

We found only one RCT evaluating the effects of green tea on bone health in postmenopausal osteoporotic women, and this made the planned meta-analysis not possible. The same author had published a detailed design prior to this RCT, demonstrating low risk of bias after our critical appraisal. This four-arm study, comparing the effect of 6-month of green tea polyphenols and/or Tai-Chi exercise, found that green tea supplement positively improved bone turnover markers and muscle strength. This finding was consistent with most observational studies, revealing that tea consumption might be beneficial for bone mineral density.

On the other hand, several longitudinal studies have found an inverse correlation between caffeine intake and bone density, especially for women. In the Framingham Osteoporosis Study, consumption of over 2.5 units of caffeine per day increased the hip fracture risk. It also showed that cola was associated with lower hip BMD in women.

For researchers, we urge more randomized controlled studies in the future. For clinicians, giving advices to people concerning about this issue should still be in a more conservative manner, especially among female group.

### CONCLUSION:

The evidence supporting tea, coffee and carbonated beverages on bone health is still very limited. We need more meticulously performed studies with robust methodology to define its true efficacy.

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## Marantodes Pumilum Leaves Increased Callus Volume And Connective Density Of Osteotomized Tibia In Osteoporosis Rat Model

Tijjani RG1, Ahmad NS1, Ima Nirwana S1, Norliza M1, Jamia Azdina J2, Norazlina M1\*  
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### INTRODUCTION:

Due to increasing life expectancy, osteoporotic fractures now accounts for higher incidence of morbidity and mortality that poses enormous economic challenge. One out of every 3 women above age 50 was reported to experience osteoporotic fracture [1]. Hip fractures, which account for 17% osteoporotic fractures, are the most fatal complication of osteoporosis with a mortality rate of 37.5% [2,3]. *Marantodes pumilum* var *alata*, MPVa (syn. *Labisia pumila*), the queen of Malaysia herbs, has been reported to protect bone of laboratory animals against osteoporosis, and its being used as a supplement in managing postmenopausal symptoms such as hot flashes [4]. Despite the enormous data on osteoprotective properties of MPVa in postmenopausal state, no previous study has investigated its effects on the most important complication of osteoporosis, fracture. In current study, the influence of aqueous leaf extract of MPVa on repair rate of fractured tibia of ovariectomized Sprague-Dawley rats was investigated.

### MATERIALS & METHODS:

Thirty healthy female Sprague-Dawley rats (4 months) were put into 5 groups (n=6): sham-operated (Sham); ovariectomized control (OVXC); estrogen treatment (ERT) and 2 leaf extract treatments (MPV20 and MPV100). All animals, except sham-operated group, were ovariectomized. After ovariectomy, right tibiae of rats in all groups were osteotomized using pulse ultrasound and fixed with titanium plates. After 2 weeks healing period, the ERT group were treated with 64.5µg/kg/d p.o. estrogen (Premarin®) while the MPV20 and MPV100 groups received 20mg and 100 mg/kg/d p.o. doses of aqueous leaf extract of MPVa, respectively, for 8 weeks. All animals were then sacrificed and osteotomized tibia were carefully harvested and investigated for morphometry and mineralization of calluses using micro-computed tomography (Skyscan 1076).

### RESULTS:

The Sham, ERT as well as MPV20 and MPV100 groups showed better ossification and restoration of Frac- tured site when compared with the OVXC (Figure 1). To similar extent as ERT and Sham groups, significantly higher bone volume (BV) of callus was seen in MPV20 group when compared with the OVXC. However, also similar to ERT and Sham groups, significantly higher bone volume fraction (BV/TV) of callus was seen in both MPV20 and MPV100 groups when compared with the OVXC. Connective density (Conn. D.) of callus, when compared with the OVXC, was significantly higher in MPV20 and group only.

Parameters	Sham	OVXC	ERT	MPV20	MPV100
BV (mm <sup>3</sup> )	12.33 ±0.5*	4.42 ±1.1	7.81 ±2.2	11.30 ±2.2*	9.35 ±1.6
BV/TV (%)	11.01 ±0.9*	4.72 ±1.0	11.72 ±3.04*	14.28 ±2.9*	13.45 ±1.5*
Conn.D (1/mm <sup>3</sup> )	163.20 ±12.5#	68.49 ±17.12	80.20 ±17.41	106.63 ±19.6#	57.45 ±12.7

### DISCUSSIONS:

The cycle of primary and secondary fracture healing is usually completed 6-8 weeks after initial injury [5]. Osteoporotic fractures are often refractory to healing. MPVa leaves contain flavonoids and phenolic acids such as quercetin, myricetin, kaempferol, syringic acid, vanillic acid and gallic acid with estrogen-like activities (phytoestrogens) [6]. Increased fracture healing properties of MPVa seen in this study could be attributed to estrogen replacement due to its phytoestrogenic content.

### CONCLUSION:

Similar to healthy controls, MPVa leaf extract restored ovariectomized rat's fractured tibia. Thus, MPVa is a useful supplement in repair of fracture in postmenopausal ovary.

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## Surgical Outcomes In Nonagenarians Following Hip Fracture

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### INTRODUCTION:

Studies of surgical intervention for hip fractures among nonagenarians are limited, and most of them were performed in western countries. Therefore, the object of this study is to investigate the short-term outcomes of nonagenarians undergoing surgery for hip fracture in Taiwan.

### MATERIALS & METHODS:

Patients who were older than 90 years and who had undergone surgery for hip fracture during the period from 2012 to 2015 were identified from the hospital's computerized database. The medical records of all of the identified patients were retrospectively reviewed. Information regarding age, gender, type of fracture, underlying diseases, and the timing and types of surgery were recorded. The data was collected on a routine basis and the analysis was carried out retrospectively

### RESULTS:

During the study period, a total of 101 patients underwent surgery for management of hip fractures. The age of patients ranged from 90 to 96 years (mean, 91.9 years) and women comprised most of the patients. The sites of hip fractures were intertrochanteric (n = 57, 56.4%) and the neck of the femur (n = 44, 43.6%). Hypertension was the most common underlying disease, followed by diabetes mellitus. DHS was the most common type of device (n = 53, 52.5%) followed by Austin Moore cemented hemiarthroplasty (n = 38, 37.6%), cannulated screws (n = 5, 5.0%), and reconstruction nails (n = 5, 5.0%). General anesthesia was the most common type of anesthesia in 87 patients, followed by spinal anesthesia.

### DISCUSSIONS:

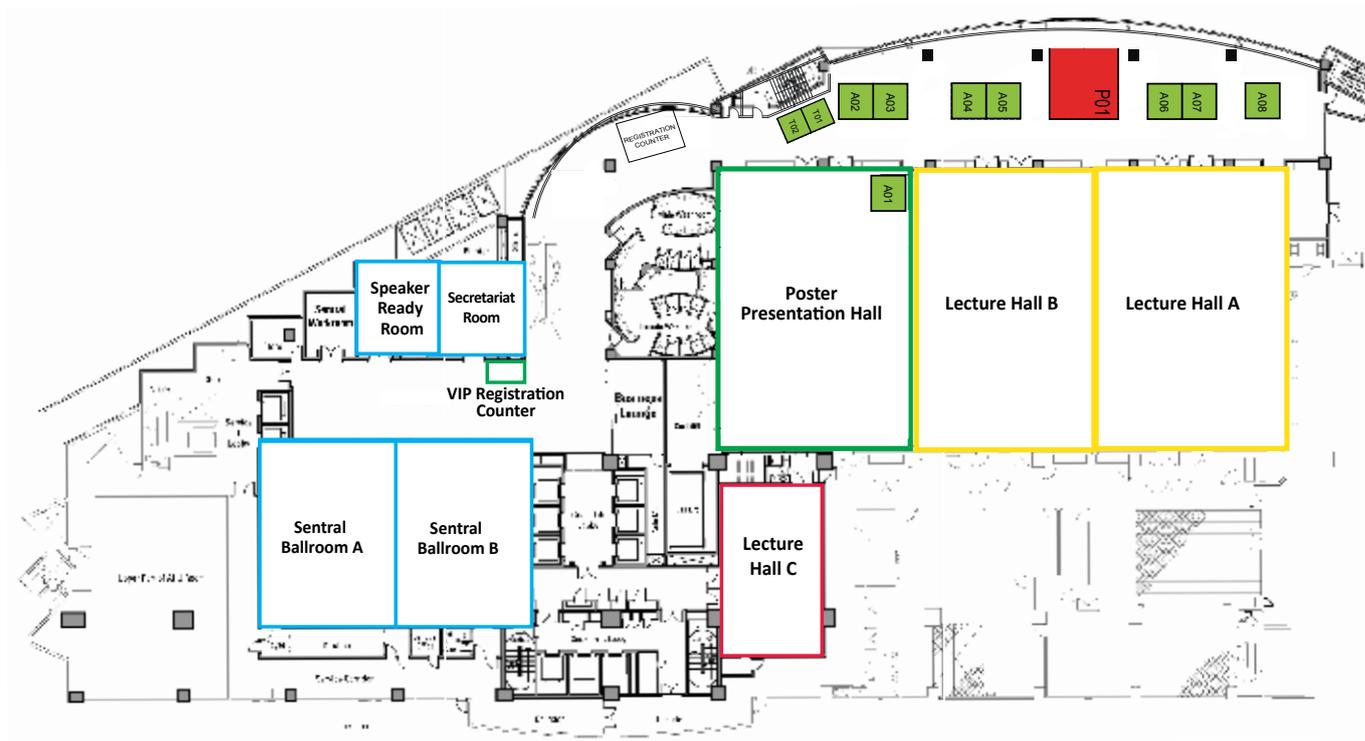
We investigated the short-term surgical outcomes of nonagenarians with hip fractures in Taiwan due to the lack of published information on this age group. Although our study may be different from other studies in design, population, and the type of management, the low mortality rate in the present work is similar as previous studies in western countries. Overall, it suggests that surgery may be a safe procedure for nonagenarians with hip fractures.

### CONCLUSION:

In conclusion, the short-term results for Asian nonagenarians with hip fractures showed that surgical management is a safe intervention in selected patients.

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 **ACKNOWLEDGEMENT**

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Booth	Company
P01	Amgen Asia Holding Ltd
A02	Eli Lilly (Malaysia) Sdn Bhd
A03	GE Healthcare Sdn Bhd
A04	Fracture Liaison Services Booth, sponsored by Amgen
A05	Fracture Liaison Services Booth, sponsored by Amgen
A06	A. Menarini Singapore Pte Ltd
A07	A. Menarini Singapore Pte Ltd
A08	Mylan Malaysia / Meda Healthcare Sdn Bhd
T01	Abbott Laboratories (M) Sdn Bhd
T02	Abbott Laboratories (M) Sdn Bhd
	Merck Sharp & Dohme (Malaysia) Sdn Bhd



# EXPERIENCE

# MALAYSIA

## TROPICAL RAINFORESTS AND GORGEOUS BEACHES



### OVERVIEW

Populated by a blend of Malays, Chinese, Indians and indigenous groups, Malaysia boasts a rich cultural heritage, from a huge variety of annual festivals and wonderful cuisines, to traditional architecture and rural crafts. There's astonishing natural beauty to take in too, including gorgeous

beaches and some of the world's oldest tropical rainforest, much of which is surprisingly accessible. Malaysia's national parks are superb for trekking and wildlife-watching, and sometimes for cave exploration and river rafting.

### LANGUAGE

The conference will be conducted in the English language. Externally, most of the locals are fluent in English and easily converse in the language.

### CURRENCY

There are many currency exchange outlets available in the airport, major shopping malls, and bank offices, where you may change your currency with the Malaysian currency Ringgit Malaysia (RM).

### CLIMATE

High temperatures and humid climate. Heavy rain, mostly in the afternoons. We advise attendees to keep hydrated by drinking lots of water.

### DINING

In Malaysia, we have wide ranges of food from Indian, Chinese to Malay cuisine.



#### Roti Canai

A classic Malaysian breakfast of Indian derivation, though this flaky finger food is good any time of day.



#### Nasi Lemak

Nasi lemak could be considered Malaysia's national dish—a little banana leaf parcel that cradles a bed of coconut rice with spicy sambal, roasted peanuts, egg.



#### Char Kuey Teow

Rice noodles stir-fried in seconds over a smoking, sparking charcoal fire.



#### Seafood

Seafood is a must to try in Malaysia! Crabs are good to go with various choices of gravy from Sweet & Sour, salt and pepper, Kam Heong or Butter.



# PLACES, VIBRANT CITIES AND OUTSTANDING FOOD

## SIGHTSEEING IN KUALA LUMPUR



### PETRONAS Twin Tower

At 451.9 meters, the Petronas Twin Towers is the world's tallest twin structures. The Twin Towers are the centrepiece of the Kuala Lumpur City Centre which is also home to the Kuala Lumpur Convention Centre, Suria KLCC shopping mall, star-rated hotels, a mosque and a beautifully landscaped KLCC Park



### Central Market

Once a wholesale and retail wet market, Central Market is among Kuala Lumpur's most prominent heritage buildings. It is now a hub for all things to do with crafts and ornaments.



### Sunway Lagoon

With over 90 attractions spread across 88 acres, Sunway Lagoon provides the ultimate theme park experience in 6 adventure zones — Water Park, Amusement Park, Wildlife Park, Extreme Park, Scream Park & Nickelodeon Lost Lagoon— Asia's 1st Nickelodeon Themed Land. Book your tickets at [sunwaylagoon.com](http://sunwaylagoon.com).



### Batu Caves

Batu Caves is a an iconic and popular tourist attraction in Selangor. Site of a Hindu temple and shrine, Batu Caves attracts thousands of worshippers and tourists, especially during the annual Hindu festival, Thaipusam. A limestone outcrop located just north of Kuala Lumpur, Batu Caves has three main caves featuring temples and Hindu shrines.

Its main attraction is the large statue of the Hindu God at the entrance, besides a steep 272 climb up its steps to finally view the stunning skyline of the city centre.





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**40%**  
*P* = 0.04  
ARR: 0.3%

New vertebral fracture

RRR  
**68%**  
*P* < 0.001  
ARR: 4.8%

Non-vertebral fracture

RRR  
**20%**  
*P* = 0.01  
ARR: 1.5%

**1** injection once every **6** months  
Be confident that your patient is receiving 6 months therapy with each injection<sup>1</sup>

**PROLIA<sup>®</sup>** is indicated for the treatment of osteoporosis in postmenopausal women at increased risk of fracture. In postmenopausal women with osteoporosis, PROLIA<sup>®</sup> increases bone mineral density (BMD) and reduces the incidence of hip, vertebral and non-vertebral fractures.

References: 1. Prolia<sup>®</sup> full prescribing information for Malaysia. 2. Cummings SR, et al. N Engl J Med. 2009; 361(8):756-65.

RRR: Relative risk reduction; ARR: Actual risk reduction.

#### PROLIA<sup>®</sup> Abbreviated PI

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