

EANM Congress 2014

Clinical Impact of BSI

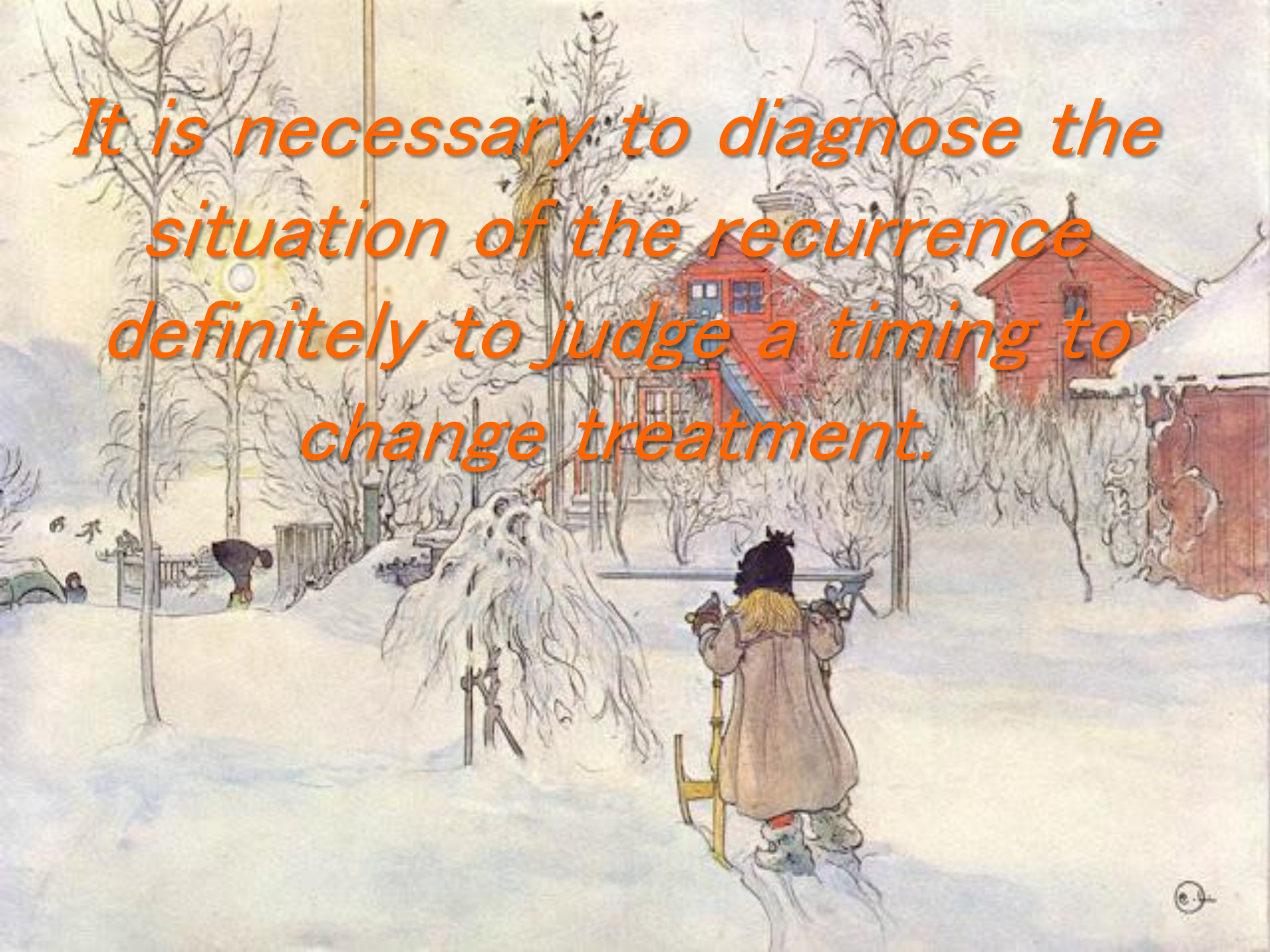
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2014. 10. 20

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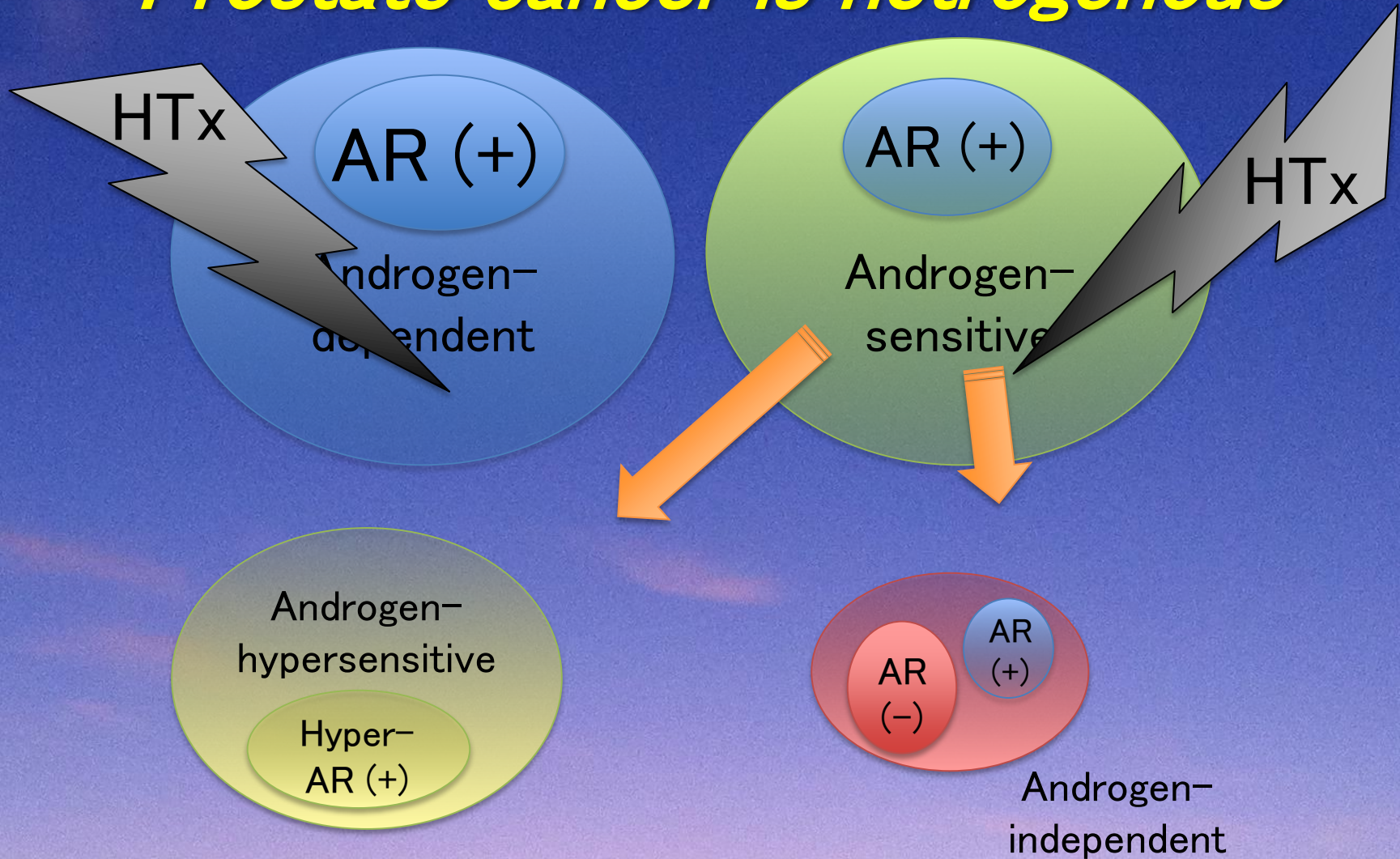
It is necessary to diagnose the situation of the recurrence definitely to judge a timing to change treatment.



What is the index to use for follow-up of the PCa with bone metastasis?

- We monitor a proliferation of the PCa with **PSA**.
- A proliferation of the PCa usually correlates with PSA
- However, **PSA** value and a **proliferation** may sometimes dissociate in CRPC
- May we really monitor a proliferation of PCa with PSA?

Prostate cancer is heterogeneous

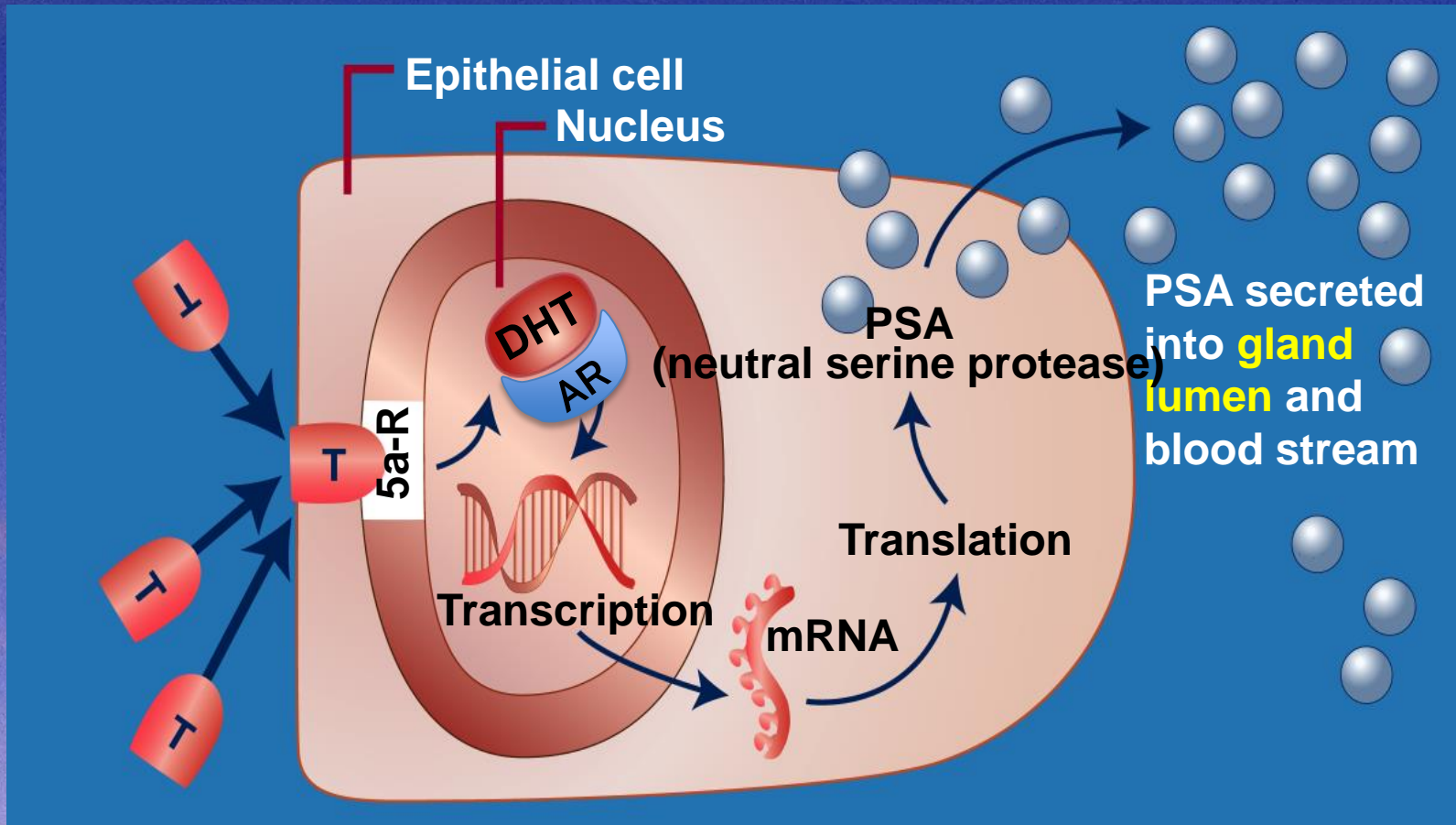


A ratio differs in each patient

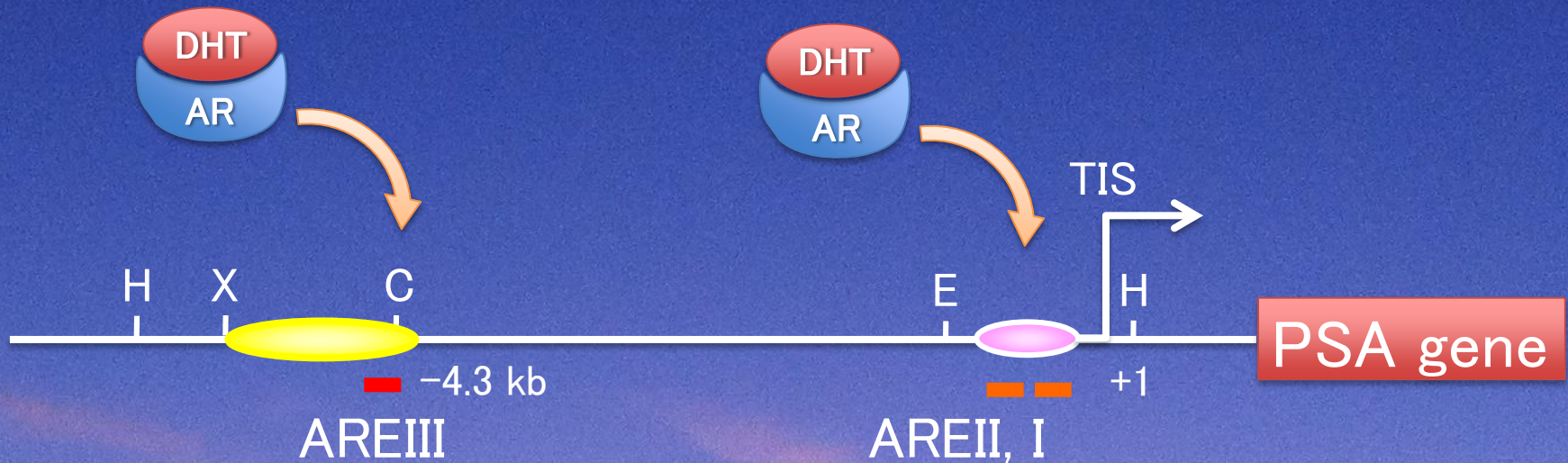
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Production of PSA



PSA gene structure

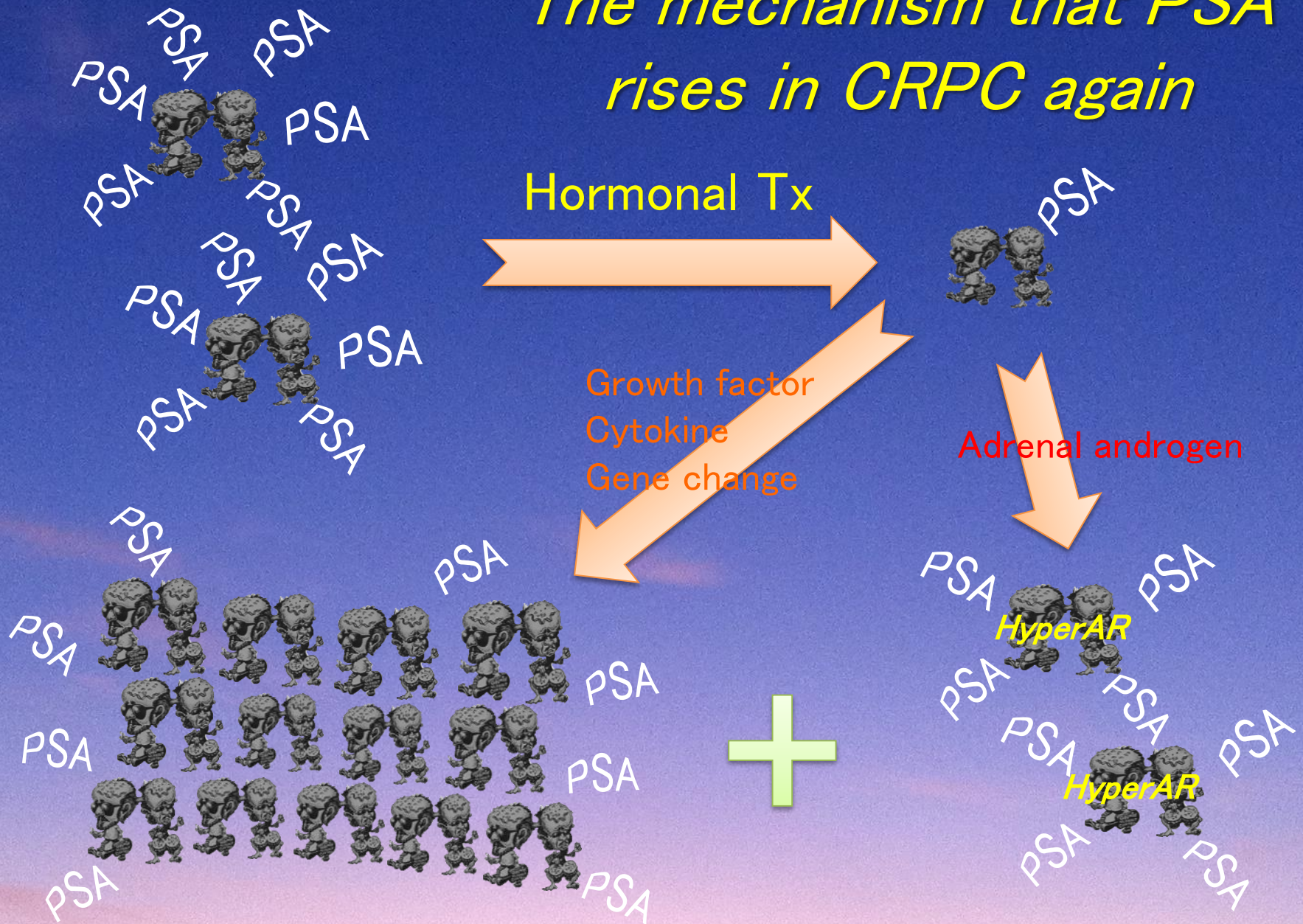


PSA is androgen-responsive gene

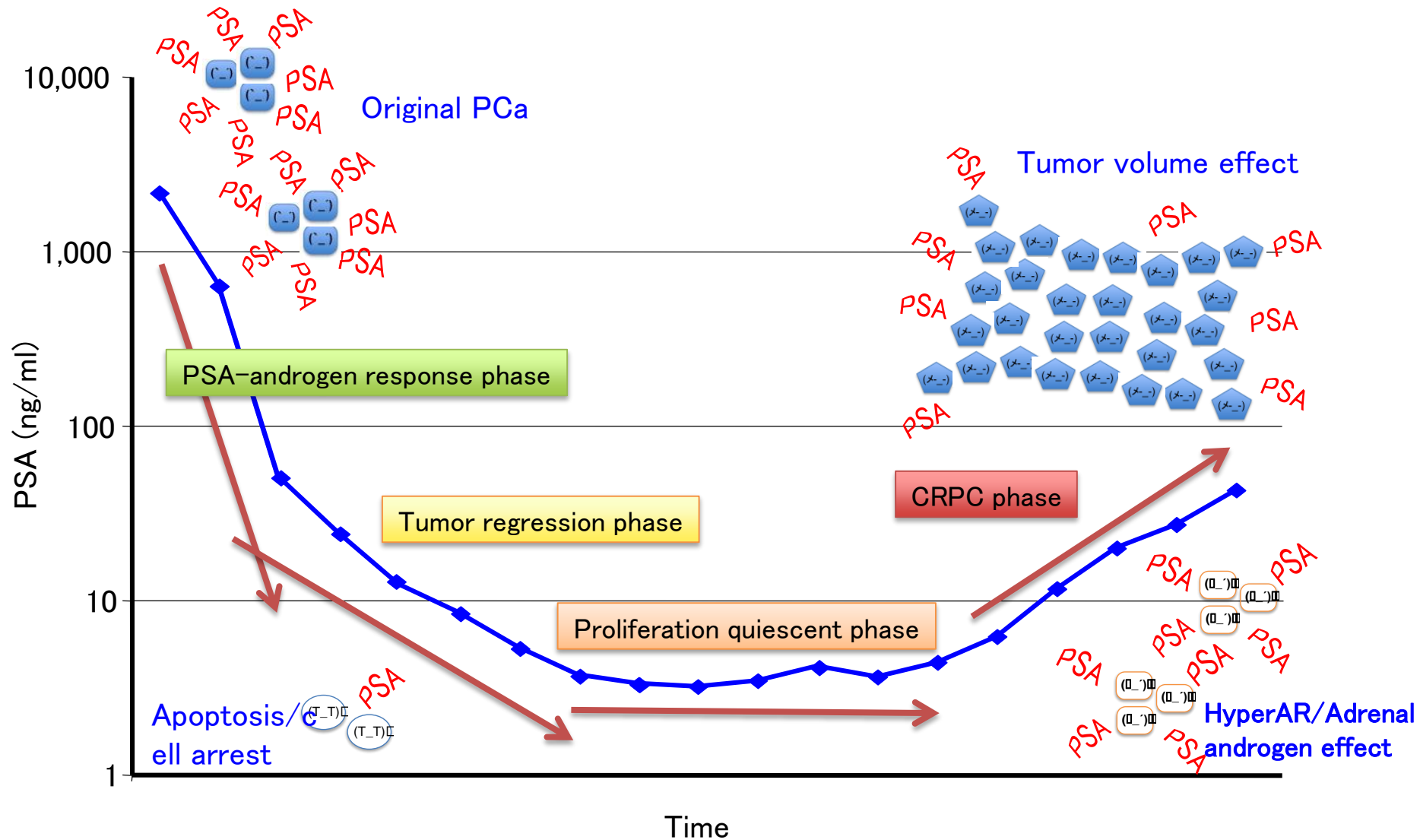
Androgen deletion decreases PSA expression

PSA does not regulate cell proliferation

The mechanism that PSA rises in CRPC again

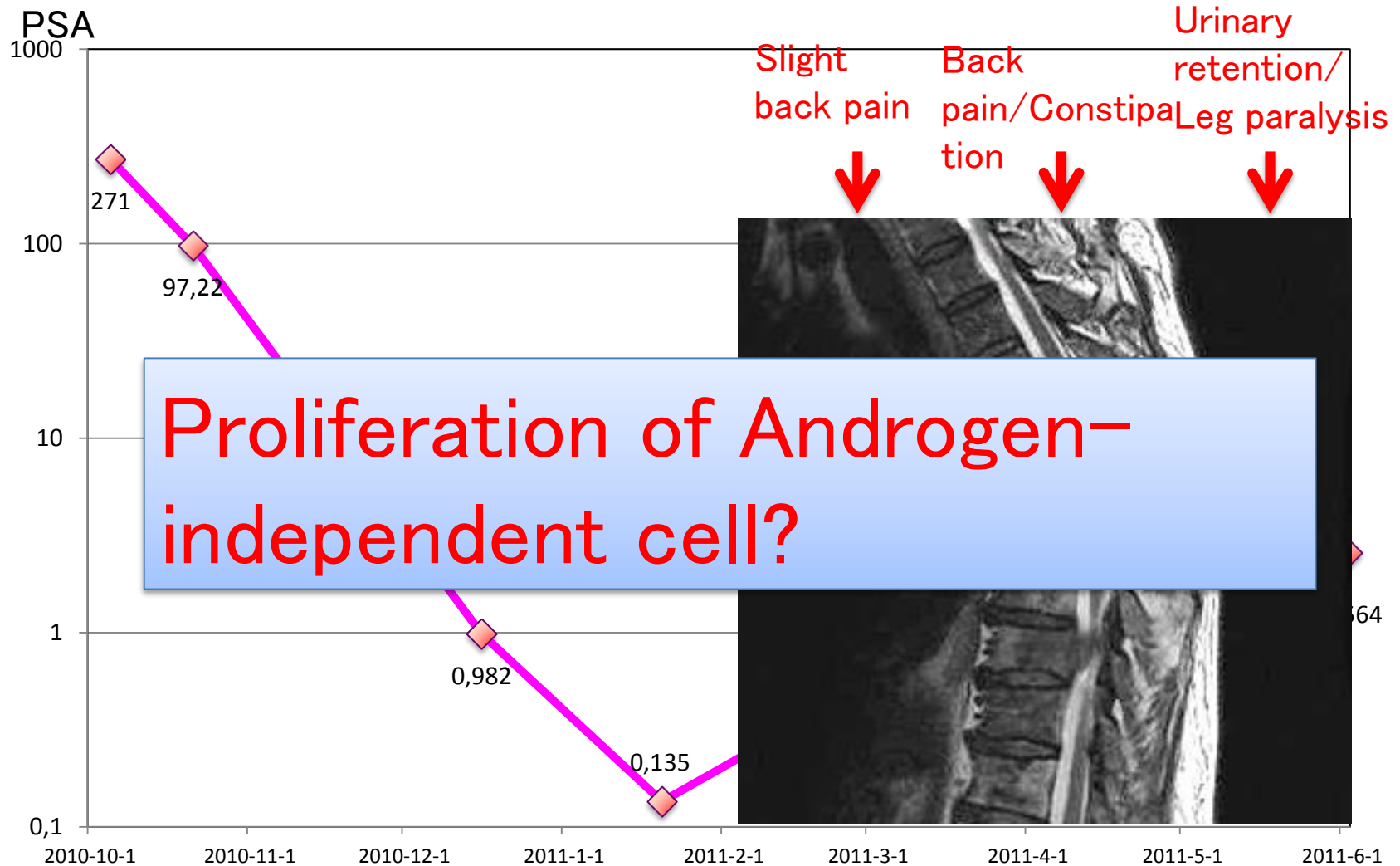


Typical change of PSA

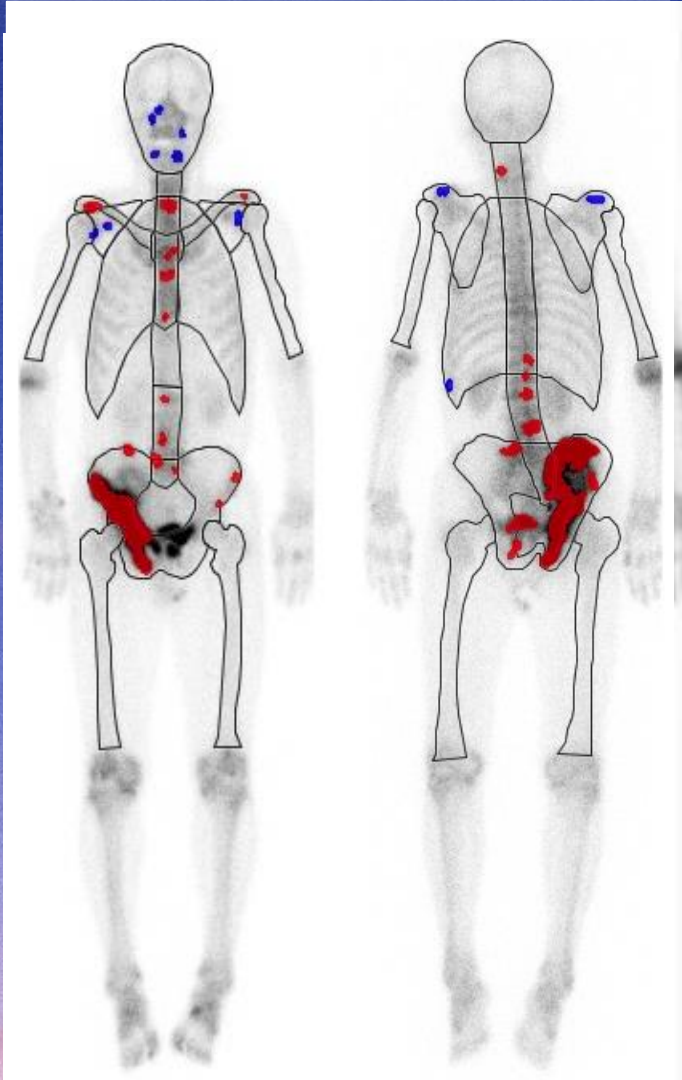


GS:3+5, Stage: T3, N0, M1, EOD: 4

LH-RH agonist + Bicalutamide → Flutamide



Bone scintigraphy

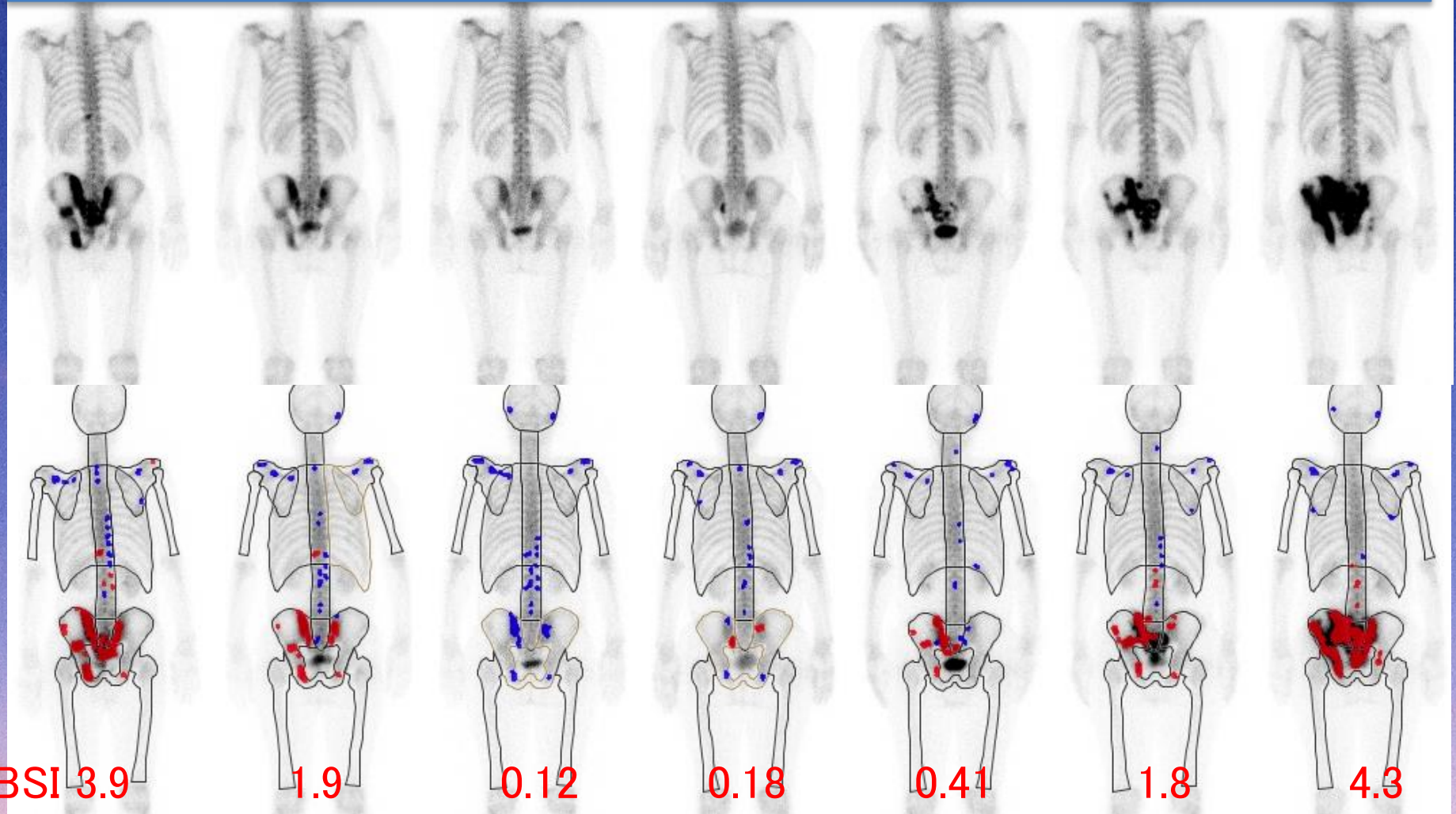


BONENAVI

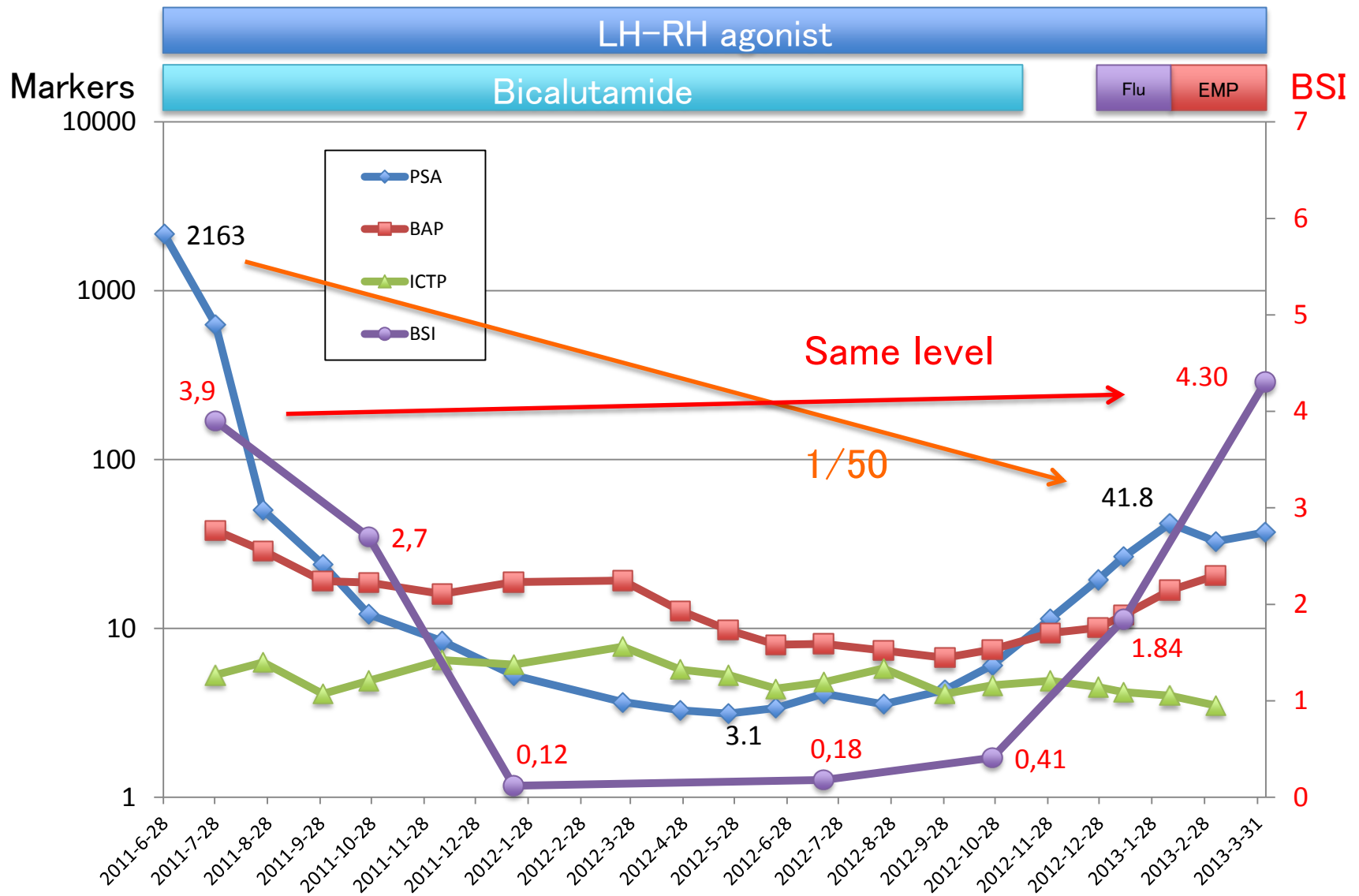
BSI (Bone Scan Index): 4.9

Change of BSI by hormonal therapy

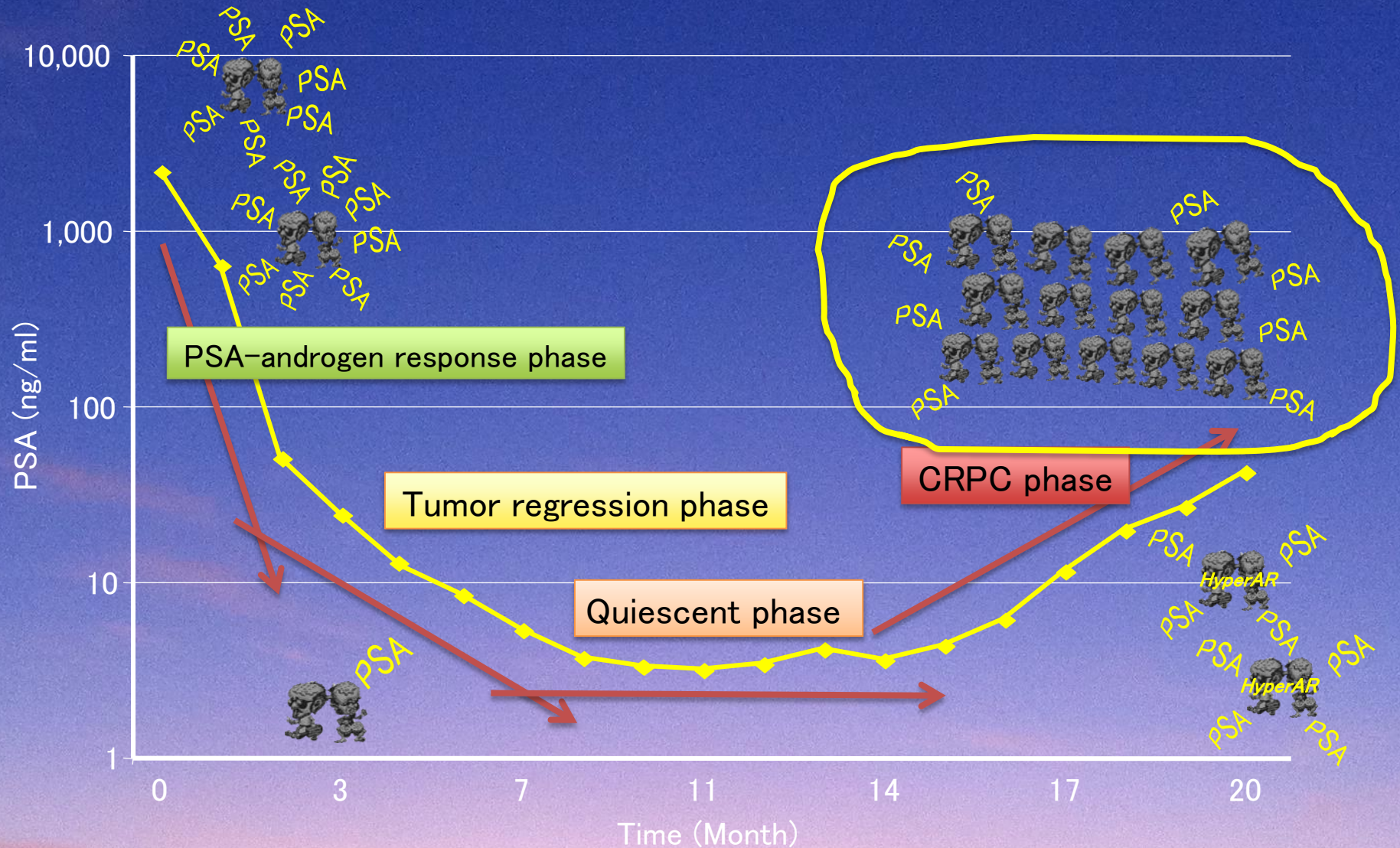
Hormonal therapy



A chronologic change of PSA and BSI

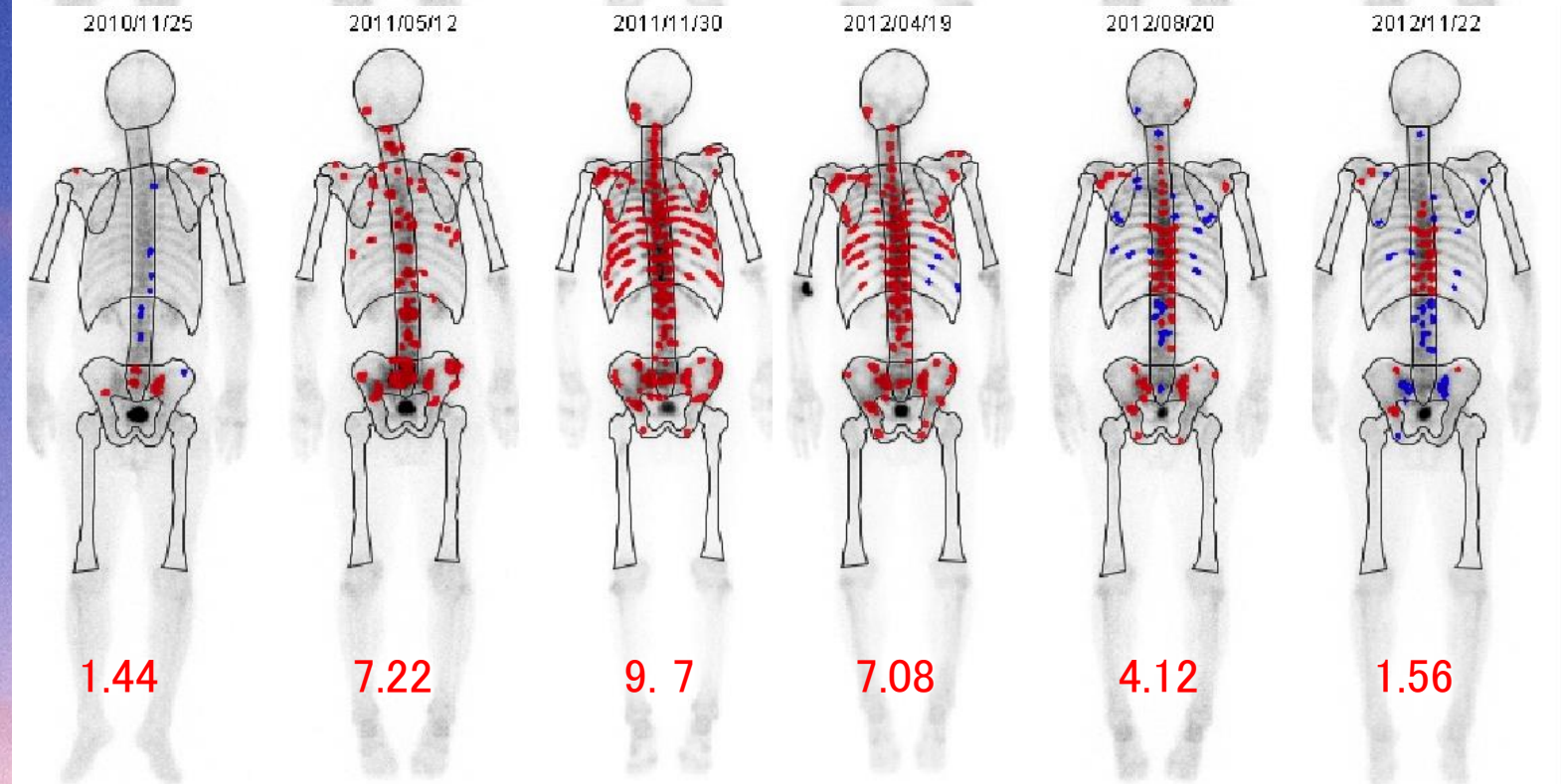
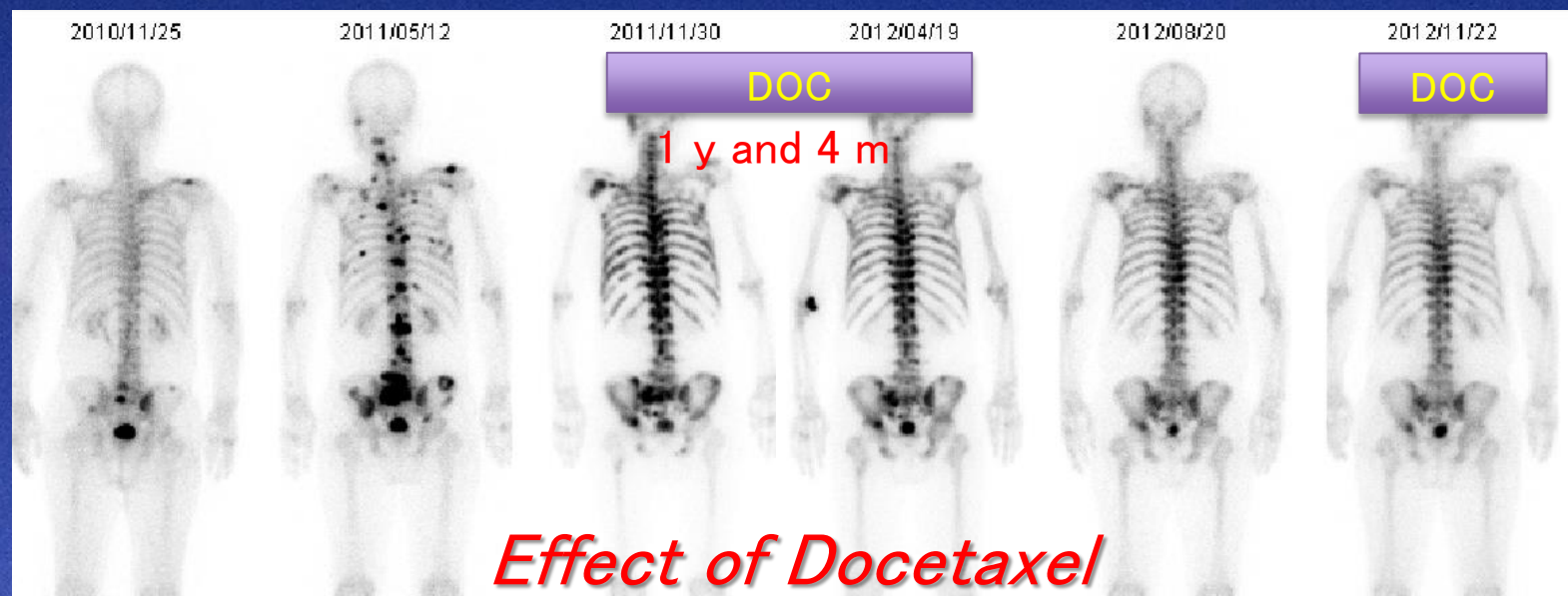


PSA kinetics



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Zoledronic acid

Bicalutamide

Flutamide

UFT

EMP

E2

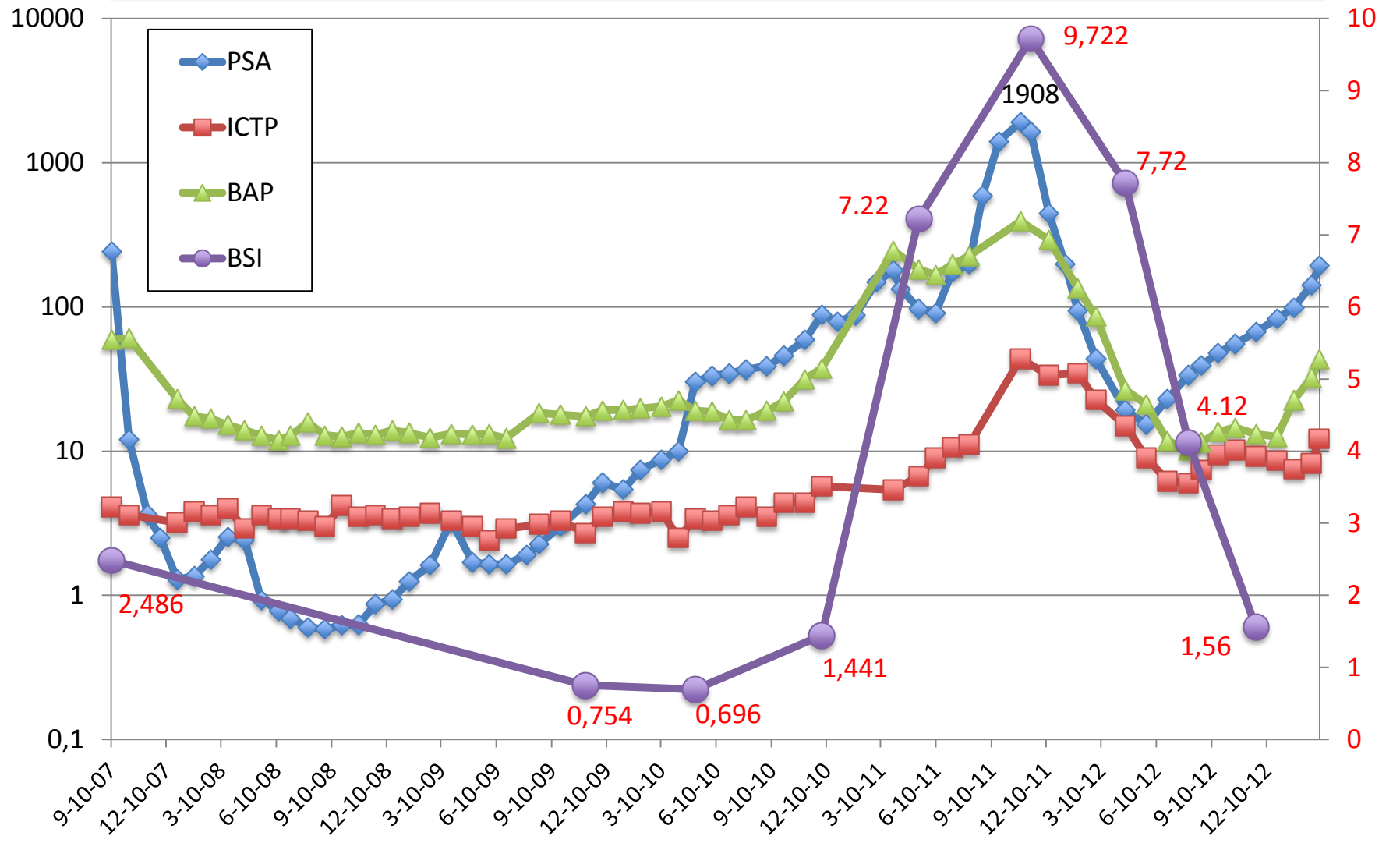
R
T

DOC

Marker

LH-RH agonist

BSI

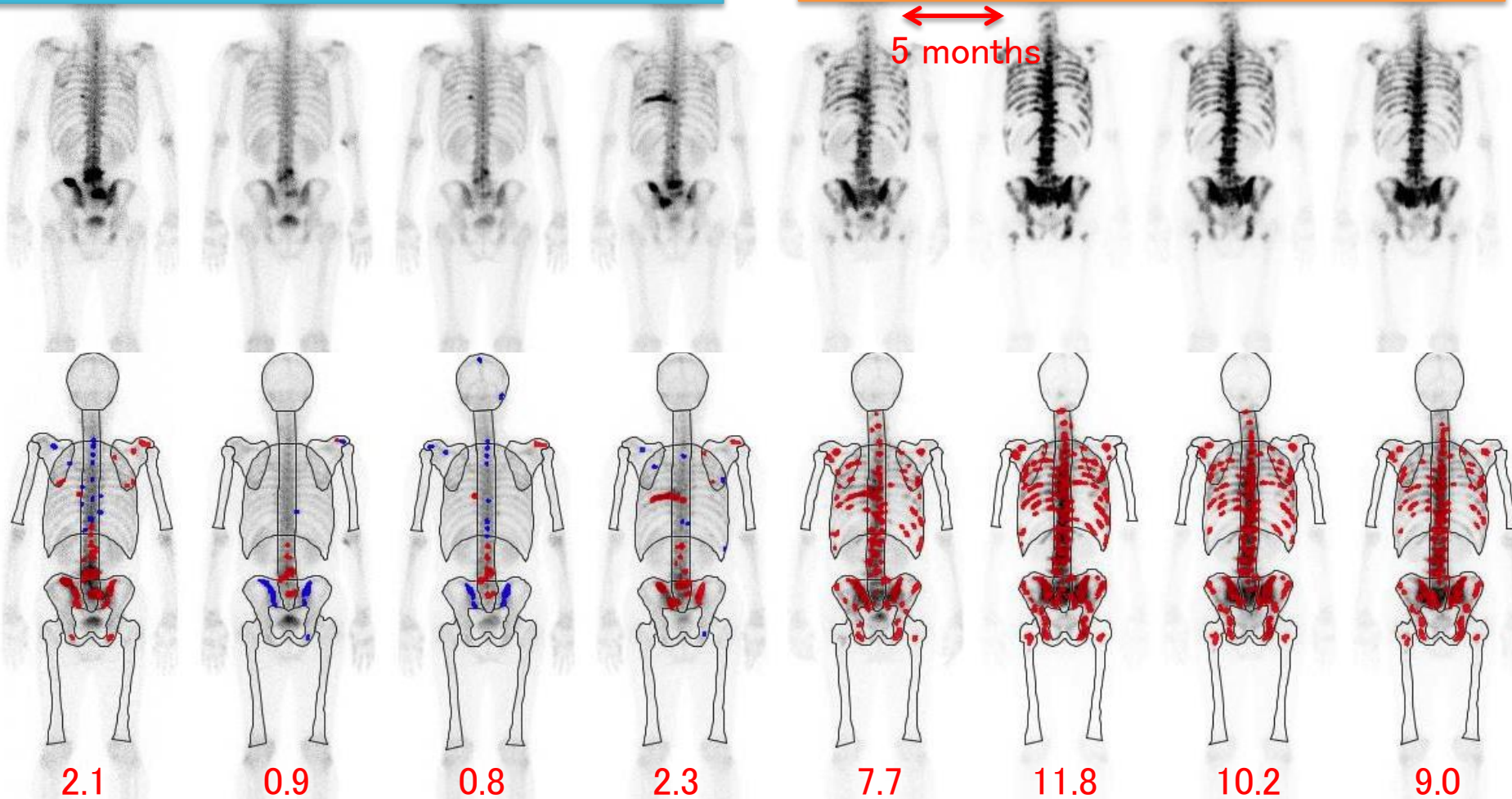


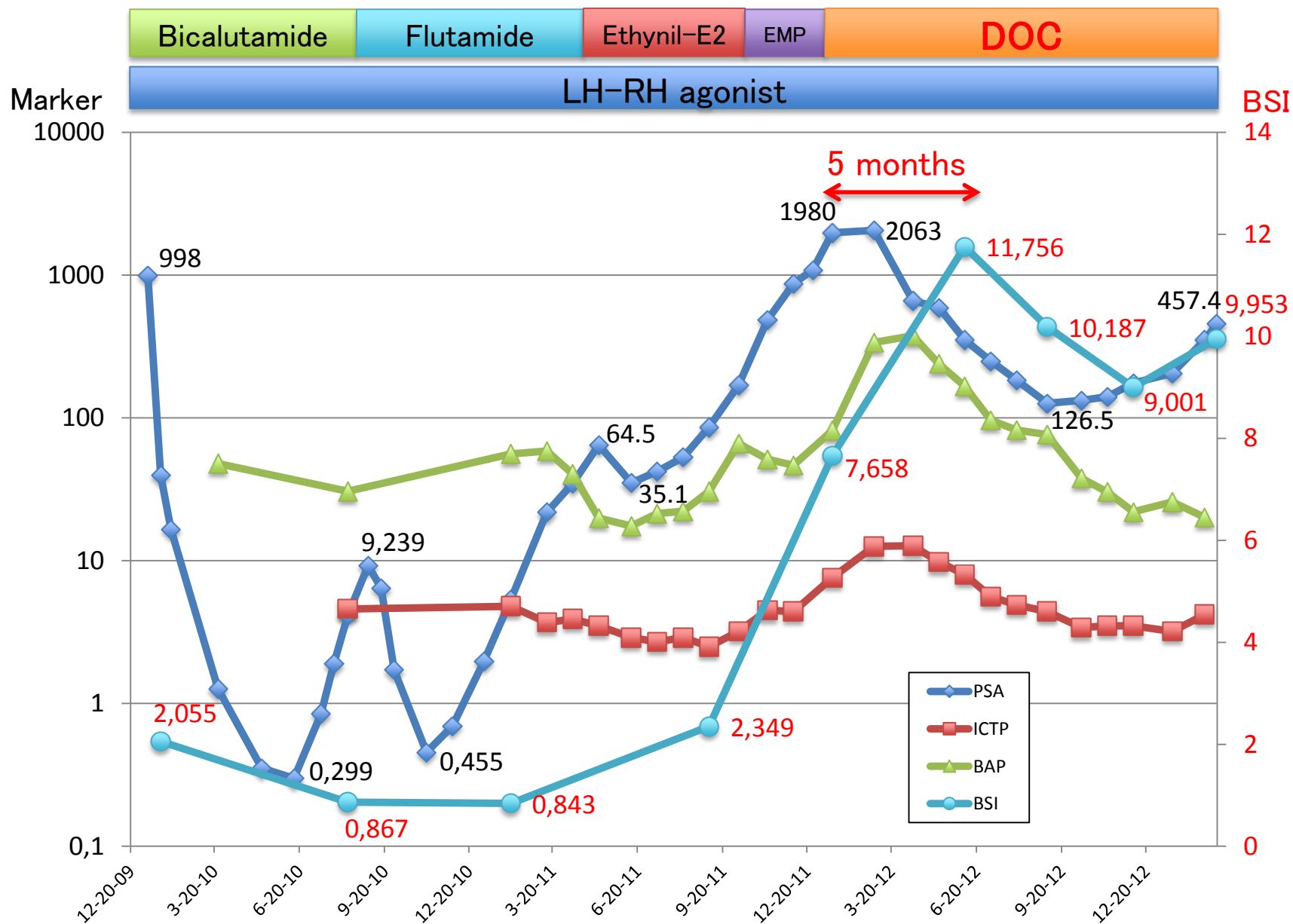
Flare phenomenon

Hormonal therapy

DOC

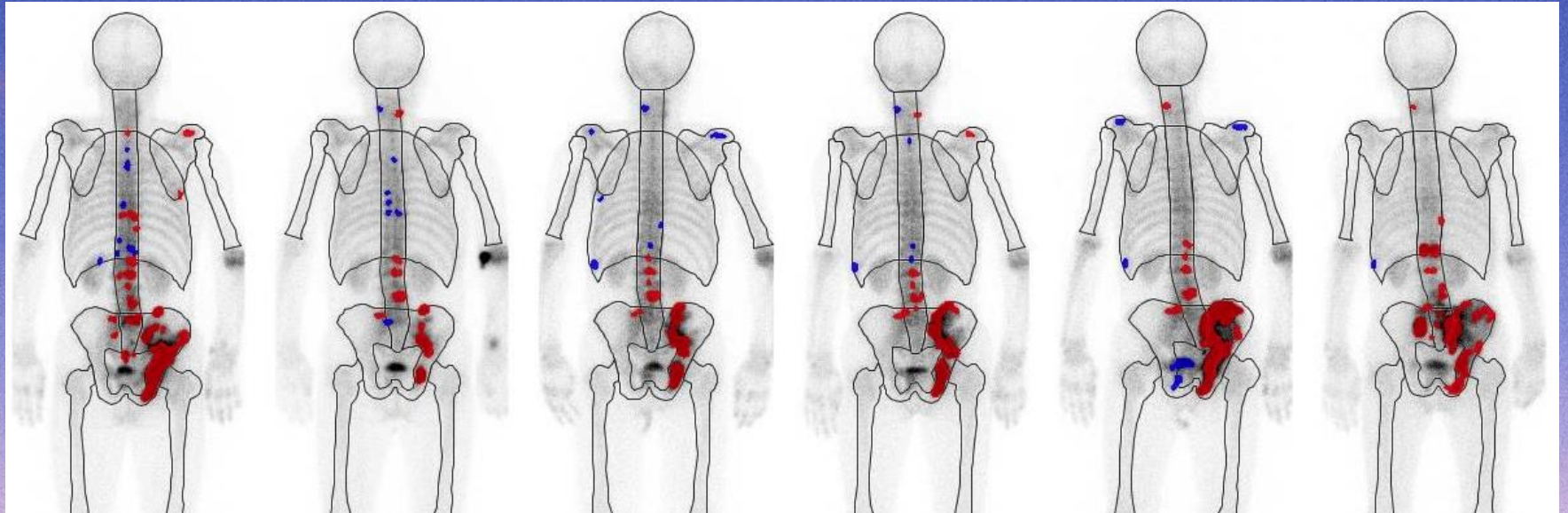
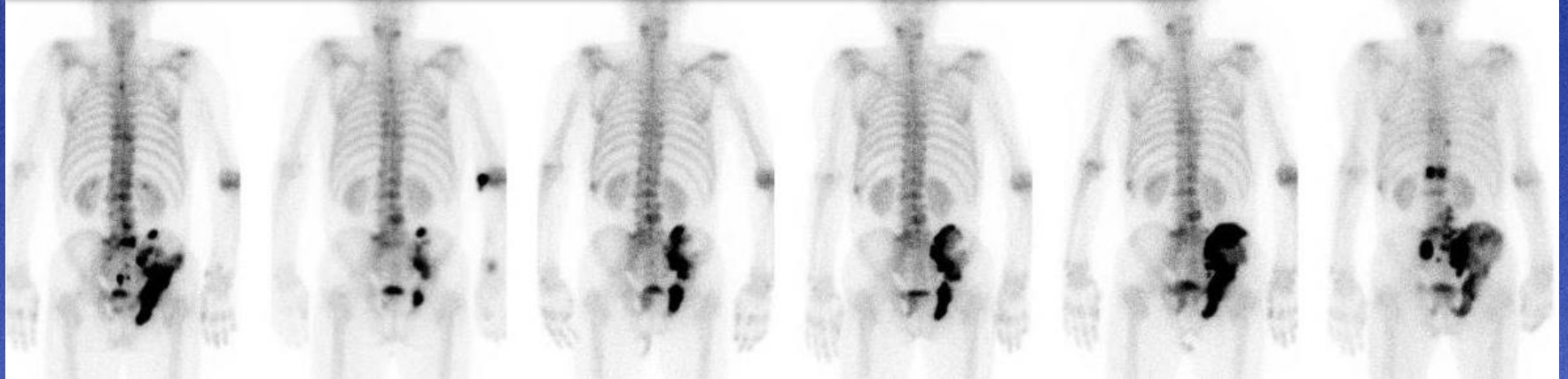
5 months





PSA does not always correlate with BSI

Hormonal therapy



BSI 4.59

1.80

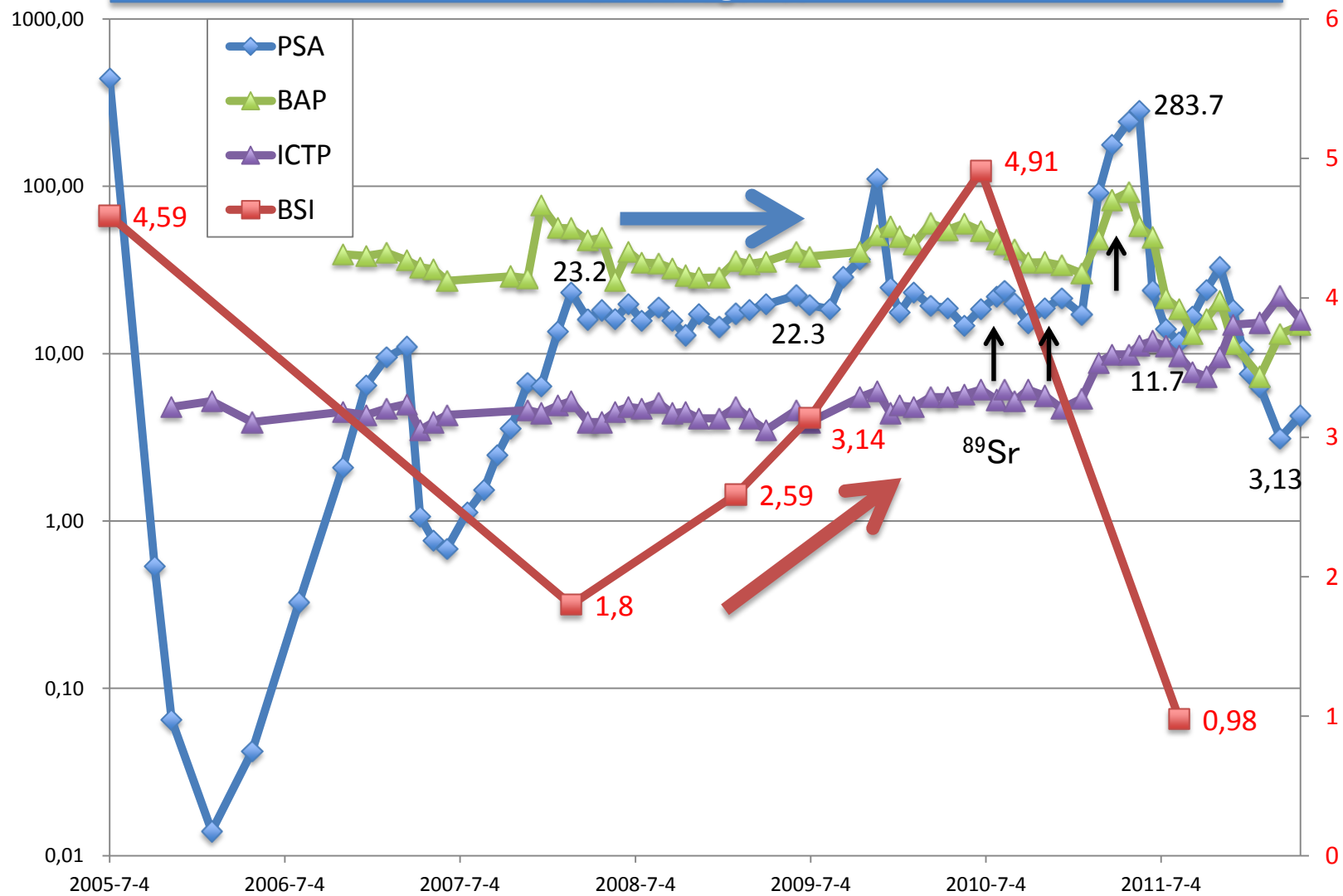
2.59

3.14

4.91

0.98

RT



*It is extremely important to evaluate
bone scintigraphy and **BSI**
as well as **PSA!***