

# Bone Density Screening Belongs in Primary Care

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SAN FRANCISCO — Measuring bone mineral density in older patients is as justifiable as measuring lipids, Dennis M. Black, Ph.D., said at a meeting on osteoporosis sponsored by the University of California, San Francisco.

Lipid testing and treatment for high cholesterol is accepted as an integral part of primary care, but bone densitometry

and treatment for low bone density isn't as readily accepted, said Dr. Black, professor of epidemiology and biostatistics at the university.

That's partly because measurements and treatments for osteoporosis came along well after tests and treatments for heart disease and its risk factors, he explained. The ready acceptance of lipid screening compared with bone density screening bothers some osteoporosis experts. "It might be called lipid envy," he joked.

The value of bone density testing stacks up nicely against the value of lipid testing. Studies have shown that people with cholesterol measurements in the highest quartile have four times the risk for heart disease compared with people whose cholesterol measurements are in the lowest quartile, Dr. Black said. Stratifying hip bone density by quartile, the risk for hip fracture increases 10-fold in people whose bone density is in the lowest quartile compared with those in the highest quartile.

Heart disease risk increases from about 0.5% in the lowest low-density lipoprotein (LDL) quartile to about 4% in the highest lipid quartile. Hip fracture risk increases from about 0.5% in the highest quartile of hip bone density to about 10% in the quartile with the least hip bone density.

Cost-effectiveness compares well, too, he added. Screening lipid levels in a 52-year-old woman and treating her for an LDL level greater than 160 mg/dL costs about \$400,000 per quality-adjusted life-year. Screening bone density in a 65-year-old woman and treating her with bisphosphonates for a T score of -2.5 (suggesting osteoporosis) costs about \$30,000 per quality-adjusted life-year, "which is considered cost effective," Dr. Black said.

The National Osteoporosis Foundation recommends bone mineral density testing for all women aged 65 years and older, and for postmenopausal women with a risk factor for osteoporosis.

The definition of risk factors for osteoporosis is a bit murky. Dr. Black includes postmenopausal women who have a history of fracture after menopause, whose mothers have a history of fracture (especially hip fracture), who take steroids, or who have very low body weight. Very low body weight commonly is considered being below 125 pounds, but that depends somewhat on height, he added.

The U.S. Preventive Services Task Force recommends bone mineral density measurements for all women above age 60. Medicare covers bone density tests for women over age 65.

Dr. Black recently analyzed 16 years of follow-up data on women in the Study of Osteoporotic Fractures and found that a single measurement of hip bone density is a good predictor of fracture risk. In these white women with a mean age of 71 years, 5% of those in the highest quartile of hip bone density developed a hip fracture over the 16-year period, compared with 32% of women in the lowest quartile of hip bone density.

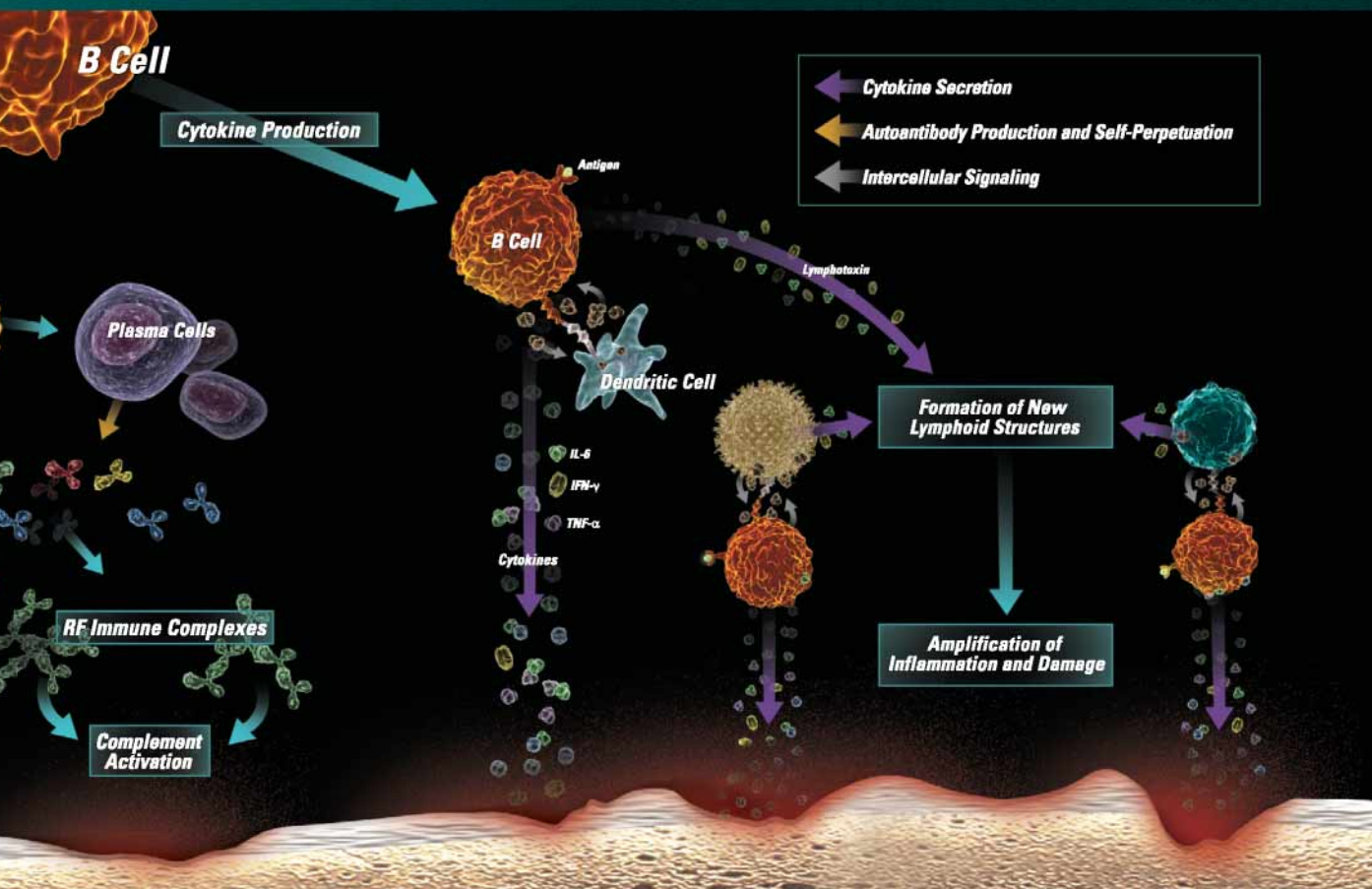
The difference was "fairly dramatic" he said. Women in the lowest quartile of hip bone density on a single measurement at the start of the study had an immediate increase in risk for hip fracture that continued as far out as 16 years.

"If it's not possible to repeat bone density measurements in 2, 3, or 4 years, the (one) value that you have is still going to be predictive long term," he said.

There is a growing recognition that T scores shouldn't be used for peripheral measurements. If a patient brings you a printout from a wrist bone density measurement that she got in a pharmacy, use that as an opportunity to talk about bone health and maybe get a more central bone density measurement, he suggested. ■

CHANGING THE WAY WE SEE RA

## IN RHEUMATOID ARTHRITIS



### AUTOREACTIVE B CELLS PRODUCE AUTOANTIBODIES THAT MAY HELP DRIVE THE DISEASE PROCESS IN RA<sup>3,5,10,11</sup>

B cells produce autoantibodies such as RF, anti-CCP, anti-GPI, and anti-RA33. RF immune complexes within the synovium may

- activate the complement system and stimulate an immune response<sup>3,10</sup>
- bind to, and activate, macrophages in the synovium<sup>11</sup>

Macrophages activated by immune complexes produce proinflammatory cytokines that perpetuate inflammation and joint destruction.<sup>11</sup>

### ACTIVATED B CELLS MAY PRODUCE CYTOKINES KNOWN TO PROMOTE INFLAMMATION AND JOINT DAMAGE IN RA<sup>3,4,6,12</sup>

B cells may be activated to produce cytokines such as TNF- $\alpha$ , IL-6, and lymphotoxin in a variety of ways:

- antigen binding to the B-cell receptor<sup>4,6</sup>
- binding of the costimulatory ligand found on activated T cells, macrophages, and dendritic cells to the costimulatory receptor on B cells<sup>4,5,12</sup>
- exposure of B cells to cytokines produced by other cells<sup>4</sup>

B-cell-produced lymphotoxin may also indirectly perpetuate RA by promoting the formation of new tertiary lymphoid structures in the synovium.<sup>9</sup>

The increased understanding of the potential roles of B cells may provide further insight into the pathogenesis of this systemic autoimmune disease and ultimately change the way we see RA.