HHS Public Access

Author manuscript

J Clin Densitom. Author manuscript; available in PMC 2018 October 01.

Published in final edited form as:

J Clin Densitom. 2017 ; 20(4): 464–471. doi:10.1016/j.jocd.2016.08.012.

Activating Patients with a Tailored Bone Density Test Results Letter and Educational Brochure: The PAADRN Randomized Controlled Trial

Fredric D. Wolinsky, PhD[1,2,3], **Yiyue Lou, MS**[4], **Stephanie W. Edmonds, BSN MPH**[2,3], **Sylvie F. Hall, BA**[2], **Michael P. Jones, PhD**[4], **Nicole C. Wright, PhD**[5,6], **Kenneth G. Saag, MD MS**[6], **Peter Cram, MD MBA**[2,7,8], and **Douglas W. Roblin, PhD**[9,10] **on behalf of the PAADRN Investigators***

[1] Department of Health Management and Policy, University of Iowa College of Public Health, Iowa City, IA

[2] Division of General Internal Medicine, Department of Internal Medicine, University of Iowa Carver College of Medicine, Iowa City, IA

[3] University of Iowa College of Nursing, Iowa City, IA

[4] Department of Biostatistics, University of Iowa College of Public Health, Iowa City, IA

[5] Department of Epidemiology, University of Alabama at Birmingham, Birmingham, AL

[6] Division of Clinical Immunology and Rheumatology, Department of Medicine, University of Alabama at Birmingham, Birmingham, AL

[7] Department of Medicine, University of Toronto, Toronto, Canada

[8] Division of General Internal Medicine, Mt. Sinai/UHN Hospitals, Toronto, Canada

[9] Kaiser Permanente, Atlanta, GA

[10] School of Public Health, Georgia State University, Atlanta, GA

Corresponding Author: Fredric D. Wolinsky, PhD, The University of Iowa, 145 North Riverside Drive, CPHB N211, Iowa City, Iowa 52242, fredric-wolinsky@uiowa.edu, 319.384.3821.

*Jeffrey R. Curtis, MD, MPH, Sarah L. Morgan, RD, MD, CCD, FACP, Janet A. Schlechte, MD, Jessica H. Williams, MPH, PhD, David J. Zelman, MD

Disclosures: F. D. Wolinsky, Y. Lou, S. W. Edmonds, S. F. Hall, M. P. Jones, P. Cram, and D. W. Roblin have no conflicts of interest. N. C. Wright has received unrestricted grant support from Amgen for work unrelated to this project. K. G. Saag has received grants from Amgen, Eli Lilly and Merck and has served as a paid consultant to Amgen, Eli Lilly, and Merck unrelated to this project.

Additional Contributions: We thank Rebecca Burmeister, MPH (UI), Mollie Giller, MPH (UI), April Miller RT(UI), CBDT, Amna Rizvi-Toner, BA, BS (UI), Kara Wessels, BA (UI), Brandi Robinson (KP), Akeba Mitchell (KP), Aimee Khamar (KP), and Roslin Nelson (KP) and all of the staff at the Iowa Social Science Research Center for recruiting and interviewing all study participants. All except Ms. Miller were compensated from grant funds for their time. We also thank Ryan Outman, MS (UAB), for coordinating and facilitating recruitment of study participants. We also thank Thuy Nguyen, MS (UI), for managing trial data. Finally, we thank the 7749 patients who participated in PAADRN.

Trial Registration: clinicaltrials.gov identifier NCT01507662

Abstract

Background—In cross-sectional studies, patient activation has been associated with better health behaviors, health outcomes, and health care experiences. Moreover, tailored interventions have led to clinically meaningful improvements in patient activation, as well as health outcomes over time. We tested whether a tailored patient-activation letter communicating bone mineral density (BMD) test results plus an educational brochure improved patient activation scores and levels at 12- and 52-weeks post-baseline as the mechanism leading to enhanced bone healthcare.

Methodology—In a randomized, controlled, double-blinded, multi-center pragmatic clinical trial we randomized 7,749 patients ≥ 50 years old and presenting for BMD testing at three medical centers in the United States between February 2012 and August 2014. The outcome measures were patient activation scores and levels based on six-items taken from the Patient Activation Measure (PAM) that were administered at the baseline, 12-week, and 52-week follow-up interviews.

Results—Mean age was 66.6 years, 83.8% were women, and 75.3% were Non-Hispanic-Whites. Overall, PAM activation scores improved from 58.1 at baseline to 76.4 by 12-weeks ($p < 0.001$) and to 77.2 ($p = 0.002$) by 52-weeks post-baseline. These improvements, however, were not significantly different between the intervention and usual care groups (18.7 vs. 18.1, $p = 0.176$, at 12-weeks) in intention-to-treat analyses.

Conclusion—PAM activation scores and levels substantially improved at 12-weeks and 52-weeks, but no differences were observed in these improvements between the intervention and usual care groups. These null findings may have occurred because the tailoring focused on the patient's BMD and fracture risk results, rather than on the patient's BMD and fracture risk results as well as the patient's baseline PAM activation scores or levels.

Keywords

Patient activation; bone density testing; osteoporosis; clinical trial

Introduction

Patient engagement, also known as patient activation, became a key component in the reformation of health care delivery in the United States (U.S.) with the passage of the Patient Protection and Affordable Care Act of 2010. Patient activation "emphasizes patients' willingness and ability to take independent actions" (1) by understanding their "role in the care process and having the knowledge, skill, and confidence to manage one's health and health care" (2). Across various diseases and conditions other than osteoporosis, patient activation scores and levels predict health behaviors, with higher patient activation scores and levels being associated with better health outcomes and care experiences, and lower health care costs (1, 3, 4). Furthermore, a growing body of evidence shows that well-crafted, patient-tailored interventions lead to statistically and clinically meaningful improvements in patient activation scores and levels and improved health outcomes (3,4). Therefore, if the triple aim of improving the patient care experience, raising the health of populations, and lowering per capita health care costs is to be achieved, then interventions to achieve patient activation should routinely be embedded in health care management programs.

Patient activation may be especially important for conditions like osteoporosis, which is “a progressive systemic skeletal disease characterized by low bone mass and microarchitectural deterioration of bone tissue” (5) that increases fracture risk, but is largely silent until a fracture occurs. Prevalence rates among those ≥ 50 years old are 10.3% for osteoporosis and 43.9% for osteopenia (low bone mass) (6). Osteoporosis-related fractures usually occur at the spine, hip, or wrist, and frequently occur incidental to a fall (7). It is estimated that by 2025 nearly three million osteoporosis-related fractures will occur every year, resulting in associated health care costs of $25.3 billion (8) as well as “premature mortality, loss of independence and function, [and] reduced quality of life” (9).

Accordingly, health care foundations, quality assurance organizations, and federal agencies (7, 9-11) have focused on policies for decreasing the prevalence of osteoporosis and osteoporosis-related fractures. The three main strategies encourage healthy behaviors (adequate calcium and vitamin D intake, weight-bearing and muscle-strengthening exercise, fall prevention, smoking cessation, and avoidance of excessive alcohol intake) (7), bone mineral density (BMD) testing using dual energy X-ray absorptiometry (DXA) (12), and when appropriate, guideline-concordant pharmacological treatment. Nonetheless, older adults generally do not engage in healthy bone behaviors (13) despite aggressive federal campaigns like *Senior Health* (http://nihseniorhealth.gov/), *Move!* (http://www.move.va.gov/), and *Let’s Move* (http://www.letsmove.gov/). Screening rates are also low with 40% of all women on Medicare reporting that they have never had a DXA (14), even though Medicare covers and encourages such testing every two years (15). Pharmacological treatment rates are even lower, with recent estimates reporting that only 23.3% received pharmacological treatment within two years of an osteoporosis diagnosis or any fragility fracture (16), and that only 28.5% received pharmacological treatment after discharge from a hip fracture (17), with both studies showing that treatment rates declined over time.

Because most prior efforts to improve osteoporosis healthcare have targeted providers with complicated interventions that have little if any patient involvement (18-25), we designed a pragmatic randomized controlled trial (RCT) known as the Patient Activation after DXA Result Notification (PAADRN) study (NCT-01507662). PAADRN’s primary focus was to evaluate whether a simple, scalable patient activation intervention improved guideline-concordant pharmacological treatment as the clinical endpoint. Based on patient activation theory (1-4), we assumed that timely, direct-to-patient communication of DXA results and fracture risk would inform patients about osteoporosis, activate them to be more pro-active in their interactions with health care providers, and improve their adherence with guideline-concordant pharmacological treatment. In this article we test whether our intervention improved patient activation scores and levels.

Materials and Methods

Design

PAADRN was a pragmatic, double-blinded RCT, in which patients either received a postal mailed tailored-letter with their DXA results accompanied by an educational brochure plus usual care, or usual care alone (26). Patients 50 years old or older and presenting for DXA

testing between February 2012 and August 2014 at the University of Iowa (UI), the University of Alabama at Birmingham (UAB), and Kaiser Permanente of Georgia (KPGA) were eligible to enroll. Age-eligible patients were excluded if they were prisoners, had overt cognitive limitations, did not speak or read English, or were deaf or without telephone access. The study protocol and consent procedures were reviewed and approved by each site's Institutional Review Board (IRB).

Randomization and Intervention

Detailed information about the randomization process is available elsewhere (26). Simply put, we rank-ordered providers at each site based on their DXA volume during 2010-2011, and randomly assigned (1:1:1) one provider within each sequential block of three at each site to each of three groups (A, B, or C). All providers in group A's patients were assigned to the intervention, all providers in group B's patients were assigned to usual care, with the providers in group C's patients randomly allocated (1:1) to either the intervention or usual care. Providers without historical DXA ordering volume data were randomized to one of the three groups when their first patient entered the study.

All patients, providers, baseline interviewers, other project staff, and investigators were initially blinded to treatment assignment because randomization only occurred after the study DXA and baseline interviews were completed. Because all follow-up data were collected by interviewers from the Iowa Social Science Research Center (ISRC) who were not PAADRN project staff and were blinded to patient assignment, concealment for all investigators and other project staff was maintained. Patients in the intervention group could have become unblinded as they received their DXA results letter and educational brochure, but to minimize this possibility and with IRB-approval we did not inform patients that they would be randomly assigned to receive the intervention letter. Similarly, providers could have become unblinded as one of their patients brought in to show or discuss with them their intervention letter and/or educational brochure.

We notified intervention patients of their DXA results using a tailored letter and an educational brochure sent by postal mail. The reliance on postal mail was based on patient preferences expressed in our pilot study (27). Intervention materials were sent from the central coordinating center (UI) about four weeks after the baseline DXA. Described elsewhere (28-30), these materials were developed using best practices in health education. The intervention letters reported the clinical impression (normal, osteopenia, or osteoporosis) and 10-year major osteoporotic fracture risk, and encouraged the patient to bring the letter to their next provider visit for discussion. The brochure explained osteoporosis, defined T-scores, and laid out five steps to better bone health, including discussing results with the patient's provider, achieving and maintaining proper calcium and vitamin D intake, the benefits of exercise, and the dangers of smoking and excessive alcohol intake. Patients in the usual care only group received information about their DXA results consistent with the normal practices of their providers and health centers.

Setting

As reported elsewhere (26, 31), the three health centers were diverse in the way that patients were notified of their DXA results. Two sites (UI and KPGA) used the *Epic* electronic health record (Verona, Wisconsin). Patients at UI had to take the initiative to sign-up to participate in the *Epic MyChart* patient portal, with 29.5% using that portal to view their DXA results (T-scores and clinical impression [normal, osteopenia, or osteoporosis]). Patients at KPGA were routinely registered for the *MyChart* patient portal (locally branded as *KP.Org*), but could not access any radiology test results, including DXA. DXA results were provided to KPGA patients via postal mailing using generic, non-tailored template letters that only included the clinical impression. Because UAB had no patient portal at the time of this intervention, its patients were notified of their DXA results at the discretion of their ordering provider.

Baseline Data Collection

Baseline interviews were conducted by PAADRN project staff using the *REDCap* computer-assisted-interviewing system (32). These telephone or in-person interviews took place up to 28-days before or 3-days after the baseline DXA. T-scores, femoral neck BMDs, and clinical impressions were taken from the baseline DXA tests, and the FRAX tool was used to calculate 10-year hip and major osteoporotic fracture risks (8). The covariates from the baseline interviews that are used in the multivariable analyses reported here have been described in detail elsewhere (26, 31) and included study site, patient age, sex, race, education, self-rated health, history of chronic obstructive pulmonary disease and/or depression, smoking status, alcohol use, engagement in weight-bearing exercise, fractures after age 40, parental hip fractures after age 50, patient prior DXA testing, fracture risk, prior diagnoses for osteopenia or osteoporosis, and current or former osteoporosis medication use.

Patient Activation

Patient activation was measured at each interview using six items from the patient activation measure (PAM) (2). The original PAM included 22 items for generically assessing patient knowledge and skill levels as well as the confidence for self-management based on well-established, modern psychometric test (item response) theory (2, 3). PAM activation scores theoretically range from the lowest (0) to the highest (100), and can be used to categorize patients into four activation levels: may not yet believe that the patient role is important; lacks confidence and knowledge to take action; beginning to take action; and, has difficulty maintaining behaviors over time. A shorter version of the PAM uses only 13 items but has similar psychometric properties and is scored by taking the sum of the ordinal responses (Strongly Disagree = 1, Disagree = 2, Agree = 3, Strongly Agree = 4) and translating those raw scores to the original 22-item PAM activation scores and levels (2) using the published scoring conversion table (33). If no items are missing this is straightforward. But if one or more items are missing, then the average of the non-missing items is multiplied by 13, and that product is converted to the 0-100 activation score using the published scoring conversion table.

We used six of the 13 items from the shorter version of the PAM because of concerns about respondent burden arising from the detailed data that needed to be collected at every interview to address the primary clinical endpoint and numerous secondary and tertiary outcomes (26). The six selected items are shown in Table 1 and include at least one question representing each of the four activation levels. Because we only used six of the PAM items, we took the average of those items, multiplied by 13, transformed that raw score to the 0-100 activation scores using the published scoring conversion table (33), and then applied the designated cut-points to derive the four activation levels.

Sample Size & Power

PAADRN was designed to provide 89% power to detect an 8% absolute difference for its primary clinical endpoint—guideline-concordant pharmacological treatment. Although PAADRN was not powered for the six items taken from the PAM, we used baseline PAM levels to estimate the minimally detectable difference. Those calculations indicated that we would have 80% power at $p < 0.025$ (based on Bonferroni adjustments, see below) to detect a difference as small as 0.8 patient activation points.

Data Analyses

Intention-to-treat analyses were conducted with multiple imputation for participants without 12- and/or 52-week follow-up interviews using the baseline covariates for missing data at 12-weeks, and the baseline covariates and 12-week outcomes for missing data at 52-weeks. Standard graphical and statistical techniques were used to evaluate each variable using bivariable methods. We compared unadjusted PAM activation scores and levels between the intervention and usual care groups. Random effects linear (for PAM activation scores) and logistic (for PAM activation levels) regression were used to adjust for patient clustering within providers and all covariates. The primary independent variable was random assignment to the intervention vs. usual care groups. Because the outcomes occur at 12- and 52-weeks and we model them independently (baseline to 12-weeks, and baseline to 52-weeks), we use Bonferroni adjustments to correct for multiple comparisons. All p-values are 2-tailed, with those ≤ 0.025 deemed statistically significant, except at baseline where no adjustments were necessary. All statistical analyses were performed using *SAS 9.4* (SAS Institute Inc., Cary, NC).

Results

The CONSORT patient flow chart and descriptive baseline data for the covariates have been published elsewhere (31). To summarize, about 54% (7,749) of the 14,280 patients known to be eligible for the study consented to participate and were enrolled. Follow-up interviews were completed by 86.8% at 12-weeks and 77.7% at 52-weeks. In general, there were no meaningful differences between patients assigned to the intervention and usual care groups on the baseline covariates (31). Mean age was 66.6, 83.8% were women, and 75.3% were Non-Hispanic-Whites. The prevalence of osteoporosis (19.5%) and osteopenia (53.1%) were higher than recent national estimates (10.3% and 43.9%) because PAADRN patients were recruited as they presented for DXA tests rather than from the general population (6).

Table 2 shows the results from the intention-to-treat unadjusted bivariable comparisons including mean PAM activation scores and the percentages for the PAM activation levels at baseline, 12-weeks and 52-weeks post-baseline for the intervention and usual care groups. Also shown are the changes in activation scores and the percentages of patients whose activation levels increased over time. At baseline, the distribution of PAM activation levels was similar to published normative data (11.8%, 29.3%, 36.5%, and 22.3% for levels 1-4) (33). Overall, PAM activation scores improved from a baseline mean of 58.1 to 76.4 at 12-weeks post-baseline ($p < 0.001$), with a further improvement to 77.2 ($p = 0.002$) by 52-weeks post-baseline. Similarly, 65.1% and 66.7% improved by one or move activation levels by 12-weeks and 52-weeks post-baseline (both $p < 0.001$). Comparing the intervention and usual care groups, however, no significant ($p > 0.025$) or meaningful differences in either PAM activation score or level improvements were observed.

Table 3 contains the regression coefficients and adjusted odds ratios (AORs) for the intervention group vs. the usual care group obtained from the intention-to-treat random effects linear and logistic regressions, respectively, that adjusted only for patient clustering within providers (crude column) and for patient clustering within providers and differences in the covariates listed above (adjusted column). Once again, no significant ($p > 0.025$) or meaningful differences were observed between the intervention and control groups on changes in PAM scores or levels by either 12-weeks or 52-weeks post-baseline.

Discussion

PAADRN was a pragmatic RCT whose primary clinical endpoint was to improve guideline-concordant pharmacological treatment, along with several secondary and tertiary outcomes among patients presenting for DXA testing at three clinical sites (26, 31). Based on patient activation theory (1-4), we assumed that timely, direct-to-patient communication of DXA results and fracture risk would inform patients about osteoporosis, activate them to be more pro-active in their interactions with health care providers, and improve their adherence with guideline-concordant pharmacological treatment. To encourage patient activation, we postal mailed intervention patients a tailored letter containing their DXA results and fracture risks along with an educational brochure about osteoporosis, while the control group only received usual care. Elsewhere we have shown that compared to the usual care group, patients in the intervention group had statistically and clinically meaningful improvements in first receiving and understanding their DXA results, and then having subsequent contact with their providers to discuss their results and treatment options, although no effect was observed on guideline-concordant pharmacological treatment (31). The purpose of this article was to evaluate whether these improvements resulted from improved patient activation. To do so, we included six items from the PAM (2, 33) at every baseline, 12-week, and 52-week interview. Although PAM activation scores and levels substantially improved by 12-weeks and improved a modest additional amount by 52-weeks post-baseline, the tailored DXA test result letter and educational brochure failed to differentiate the magnitude of improvements between the intervention and usual care groups.

There are at least three plausible reasons why the PAADRN intervention did not improve patient activation compared to the usual care group—measurement limitations, practice

effects, and the tailoring focus. We only used six items from the PAM, and therefore may have had insufficient sensitivity to detect small differences. The six items that we selected (Table 1), however, included at least one statement from within each activation level, and the range of activation scores was the same as it was for the 13-item PAM. Furthermore, the baseline distribution on activation levels was very similar to normative data on the PAM, and the overall improvements in activation scores and levels were substantial. Thus, it is unlikely that measurement limitations explain our null findings.

Practice (or learning) effects occur when participants are asked the same questions at several different times (i.e., at subsequent follow-up interviews) over the course of a study. This generally leads to improved performance reflected in higher scores after the baseline interview. Practice effects are known to occur frequently, may be as large as 0.25 SD, but generally decline with age and the length of time in-between administrations (34). As such, practice effects could also explain our null findings, especially for the substantial improvement in activation scores and levels between baseline and 12-weeks post-baseline. This seems unlikely, however, for two reasons. First, the item response theory based reliability for the PAM is reported to be excellent (0.85) as is coefficient alpha (0.87), and test-retest reliability for the PAM has been shown to be moderate to very good (2, 33). Second, previous reports have shown virtually no change in control group activation scores over six weeks, and only a modest improvement of 3.3 activation points over six months (35). Nonetheless, it is possible that the magnitude of the overall improvements in patient activation scores and levels shown here may have diminished the opportunity to differentiate between intervention and practice effects, especially given the reduced sensitivity of our six-item measure.

The third explanation for our null findings involves the focus of our intervention tailoring. While the activation intervention letter was tailored to each patient's study DXA and fracture risk, the educational brochure was not, although it was developed specifically for this study using best practices in health education. Perhaps we should have focused the intervention letter and the educational brochure not only on the patient's study DXA and fracture risk, but also on their baseline PAM activation score and level. This would be consistent with recent cutting-edge patient activation interventions that are tailored in all respects to the individual patient (1, 3), although that would have been far more complex in such a large clinical trial, and generally not consistent with the logic of pragmatic RCTs, unless a computer-assisted infrastructure to do so had been developed and put in place.

Conclusions

We found that directly communicating patients' DXA results and fracture risk to them via a tailored DXA result letter accompanied by an educational bone health brochure did not lead to greater improvements in patient activation scores or levels. Thus, PAADRN was a negative trial with respect to patient activation as the mechanism through which statistically and clinically meaningful improvements in patients receiving and understanding their DXA results, and having subsequent contact with their providers to discuss their results and treatment options were observed (31). Future patient activation studies should consider tailoring all aspects and materials of the intervention to the patient's study DXA and fracture

risk and their baseline PAM activation score and level based on either the 13- or 22-item PAM measures.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding/Support: This work was supported by R01 AG033035 to Dr. Cram and Dr. Wolinsky from the NIA at the NIH. Dr. Cram is also supported by a K24 AR062133 award from the NIAMS at the NIH. Dr. Saag is also supported by a K24 AR052361 award from the NIAMS at the NIH.

Role of the Sponsor: The NIA, NIAMS, and NIH had no role in the (a) design and conduct of the study, (b) collection, management, analysis, and interpretation of the data, (c) preparation, review, or approval of the manuscript, or (d) decision to submit the manuscript for publication.

References

1. Hibbard JH, Greene J. What the evidence shows about patient activation: better health outcomes and care experiences; fewer data on costs. Health Aff. 2013; 32:207–214.
2. Hibbard JH, Stockard J, Mahoney ER, et al. Development of the Patient Activation Measure (PAM): conceptualizing and measuring activation in patients and consumers. Health Serv Res. 2004; 39:1005–1026. [PubMed: 15230939]
3. Hibbard J, Gilburt H. Supporting people to manage their health: an introduction to patient activation. The King's Fund, United Kingdom. 2014
4. Greene J, Hibbard JH. Why does patient activation matter? An examination of the relationships between patient activation and health-related outcomes. J Gen Intern Med. 2012; 27:520–526. [PubMed: 22127797]
5. Kanis JA, McCloskey EV, Johansson H, et al. European guidance for the diagnosis and management of osteoporosis in postmenopausal women. Osteoporos Int. 2013; 24:23–57. [PubMed: 23079689]
6. Wright NC, Looker AC, Saag KG, et al. The recent prevalence of osteoporosis and low bone mass in the United States based on bone mineral density at the femoral neck or lumbar spine. J Bone Miner Res. 2014; 29:2520–2526. [PubMed: 24771492]
7. National Osteoporosis Foundation. Clinician's guide to prevention and treatment of osteoporosis. National Osteoporosis Foundation; Washington, DC: 2014.
8. Kanis JA, McCloskey EV, Johansson H, et al. Case finding for the management of osteoporosis with FRAX--assessment and intervention thresholds for the UK. Osteoporos Int. 2008; 19:1395–1408. [PubMed: 18751937]
9. Nelson, HD., Haney, EM., Chou, R., et al. Screening for osteoporosis: systematic review to update the 2002 US Preventive Services Task Force recommendation. Agency for Healthcare Research and Quality; Maryland: 2010. U.S. Preventive Services Task Force evidence syntheses.
10. Healthy People 2020. Arthritis, osteoporosis, and chronic back conditions. Available at: https://www.healthypeople.gov/2020/topics-objectives/topic/Arthritis-Osteoporosis-and-Chronic-Back-Conditions. Accessed March 17, 2015.
11. National Committee for Quality Assurance. HEDIS 2016. Available at: http://www.ncqa.org/hedis-quality-measurement/hedis-measures/hedis-2016. Accessed April 13, 2016.
12. Kanis JA. Diagnosis of osteoporosis and assessment of fracture risk. Lancet. 2002; 359:1929–1936. [PubMed: 12057569]
13. Centers for Disease Control and Prevention. Centers for Disease Control and Prevention. US Dept of Health and Human Services; Georgia: 2013. The state of aging and health in America 2013.
14. Amarnath AL, Franks P, Robbins JA, et al. Underuse and overuse of osteoporosis screening in a regional health system: a retrospective cohort study. J Gen Intern Med. 2015; 30:1733–1740. [PubMed: 25986135]

15. U.S. Department of Health and Human Services, Centers for Medicare & Medicaid Services. Your guide to Medicare's preventive services. U.S. Department of Health and Human Services Centers for Medicare & Medicaid Services; Maryland: 2015.
16. Liu Z, Weaver J, de Papp A, et al. Disparities in osteoporosis treatments. Osteoporos Int. 2016; 27:509–519. [PubMed: 26216226]
17. Solomon DH, Johnston SS, Boytsov NN, McMorrow D, Lane JM, Krohn KD. Osteoporosis medication use after hip fracture in U.S. patients between 2002 and 2011. J Bone Miner Res. 2014; 29:1929–1937. [PubMed: 24535775]
18. Majumdar SR, Johnson JA, McAlister FA, et al. Multifaceted intervention to improve diagnosis and treatment of osteoporosis in patients with recent wrist fracture: a randomized controlled trial. CMAJ. 2008; 178:569–575. [PubMed: 18299546]
19. Majumdar SR, McAlister FA, Johnson JA, et al. Interventions to increase osteoporosis treatment in patients with 'incidentally' detected vertebral fractures. Am J Med. 2012; 125:929–936. [PubMed: 22938928]
20. Ciaschini PM, Straus SE, Dolovich LR, et al. Community based intervention to optimize osteoporosis management: randomized controlled trial. BMC Geriatr. 2010; 10:60. [PubMed: 20799973]
21. Majumdar SR, Johnson JA, Lier DA, et al. Persistence, reproducibility, and cost-effectiveness of an intervention to improve the quality of osteoporosis care after a fracture of the wrist: results of a controlled trial. Osteoporos Int. 2007; 18:261–270. [PubMed: 17086470]
22. Majumdar SR, Rowe BH, Folk D, et al. A controlled trial to increase detection and treatment of osteoporosis in older patients with a wrist fracture. Ann Intern Med. 2004; 141:366–373. [PubMed: 15353428]
23. Leslie WD, LaBine L, Klassen P, et al. Closing the gap in postfracture care at the population level: a randomized controlled trial. CMAJ. 2012; 184:290–296. [PubMed: 22184366]
24. Outman RC, Curtis JR, Locher JL, et al. Improving osteoporosis care in high-risk home health patients through a high-intensity intervention. Contemp Clin Trials. 2012; 33:206–212. [PubMed: 22005175]
25. Cranney A, Lam M, Ruhland L, et al. A multifaceted intervention to improve treatment of osteoporosis in postmenopausal women with wrist fractures: a cluster randomized trial. Osteoporos Int. 2008; 19:1733–1740. [PubMed: 18629567]
26. Edmonds SW, Wolinsky FD, Christensen AJ, et al. The PAADRN Study: A design for a randomized controlled practical clinical trial to improve bone health. Contemp Clin Trials. 2013; 34:90–100. [PubMed: 23085132]
27. Cram P, Rosenthal GE, Ohsfeldt R, et al. Failure to recognize and act on abnormal test results: the case of screening bone densitometry. Jt Comm J Qual Patient Saf. 2005; 31:90–97. [PubMed: 15791768]
28. Edmonds SW, Cram P, Lu X, et al. Improving bone mineral density reporting to patients with an illustration of personal fracture risk. BMC Med Inform Decis Making. 2014; 14:101.
29. Edmonds SW, Solimeo SL, Lu X, et al. Developing a bone mineral density test result letter to send to patients: a mixed-methods study. Patient Prefer Adherence. 2014; 8:827–841. [PubMed: 24940049]
30. Edmonds SW, Solimeo SL, Nguyen VT, et al. Understanding preferences for osteoporosis information to develop an osteoporosis-patient education brochure. Perm J. 2016; 20 In Press.
31. Cram P, Wolinsky FD, Lou Y, et al. Patient-activation and gguideline-concordant pharmacological treatment after bone density testing: the PAADRN randomized controlled trial. Osteoporos Int. 2016; 27 In Press.
32. Harris PA, Taylor R, Thielke R, et al. Research electronic data capture (REDCap)--a metadata-driven methodology and workflow process for providing translational research informatics support. J Biomed Inform. 2009; 42:377–381. [PubMed: 18929686]
33. Hibbard JH, Mahoney ER, Stockard J, et al. Development and testing of a short form of the patient activation measure. Health Serv Res. 2005; 40:1918–1930. [PubMed: 16336556]

34. Calamia M, Markon K, Tranel D. Scoring higher the second time around: meta-analyses of practice effects in neuropsychological assessment. Clin Neuropsychol. 2012; 26:543–570. [PubMed: 22540222]

35. Hibbard JH, Mahoney ER, Stock R, et al. Do increases in patient activation result in improved self-management behaviors? Health Serv Res. 2007; 42:1443–1463. [PubMed: 17610432]

Table 1

The six items from the Patient Activation Measure (PAM) (33) that were used at each interview.

Items	Activation Level	Raw Activation Score
1. When all is said and done, you are the person who is responsible for managing your health condition.	1	38.6
2. Taking an active role in your own health care is the most important factor in determining your health and ability to function.	1	41.1
5. You are confident that you can tell when you need to go get medical care and when you can handle a health problem yourself.	2	43.7
9. You know the different medical treatment options available for your health condition.	3	49.8
10. You have been able to maintain the lifestyle changes for your health that you have made.	3	50.5
13. You are confident that you can maintain lifestyle changes like diet and exercise even during times of stress.	4	53.0

Note: The raw scores of the 13 items in the short form of the PAM range from a low of 38.6 to a high of 53.0. These raw scores are then converted to the 0-100 PAM activation scores using the published scoring conversion table.

Table 2

Patient Activation Measure (PAM) activation scores and levels at baseline, and at 12-weeks and 52-weeks post-baseline by intervention group using unadjusted, intention-to-treat analyses.

	Intervention Group N = 3,898	**Usual Care Group N = 3,851**	**P-value**
PAM activation scores and changes			
PAM activation score at baseline, mean (SD)	57.8 (11.4)	58.3 (11.8)	0.056
PAM activation score at 12-weeks, mean (SD)	76.5 (16.2)	76.4 (16.4)	0.877
PAM activation score at 52-weeks, mean (SD)	76.9 (16.1)	77.5 (15.8)	0.169
PAM activation score change from baseline to 12-weeks), mean (SD)	18.7 (17.3)	18.1 (17.5)	0.176
PAM activation score change from baseline to 52-weeks), mean (SD)	19.1 (17.1)	19.2 (17.0)	0.875
PAM activation levels at baseline, number (%)			0.262
Level 1: May not yet believe that the patient role is important	495 (12.7)	484 (12.6)	
Level 2: Lacks confidence and knowledge to take action	834 (21.4)	825 (21.4)	
Level 3: Beginning to take action	1849 (47.4)	1757 (45.6)	
Level 4: Has difficulty maintaining behaviors over time	720 (18.5)	785 (20.4)	
PAM activation levels at 12-weeks, number (%)			0.600
Level 1: May not yet believe that the patient role is important	58 (1.5)	71 (1.9)	
Level 2: Lacks confidence and knowledge to take action	130 (3.3)	133 (3.4)	
Level 3: Beginning to take action	983 (25.2)	971 (25.2)	
Level 4: Has difficulty maintaining behaviors over time	2727 (70.0)	2676 (69.5)	
PAM activation levels at 52-weeks, number (%)			0.114
Level 1: May not yet believe that the patient role is important	55 (1.4)	57 (1.5)	
Level 2: Lacks confidence and knowledge to take action	128 (3.3)	108 (2.8)	
Level 3: Beginning to take action	900 (23.1)	837 (21.7)	
Level 4: Has difficulty maintaining behaviors over time	2815 (72.2)	2850 (74.0)	
Improvements in PAM activation scores and levels			
Any PAM score improvement baseline to 12-weeks, number (%)	3114 (79.9)	3034 (78.8)	0.239
Any PAM score improvement baseline to 52-weeks, number (%)	3133 (80.4)	3128 (81.2)	0.385

	Intervention Group N = 3,898	**Usual Care Group N = 3,851**	**P-value**
Any PAM level improvement baseline to 12-weeks, number (%)	2567 (65.9)	2477 (64.3)	0.171
Any PAM level improvement baseline to 52-weeks, number (%)	2607 (66.9)	2559 (66.5)	0.698

Table 3

Crude and adjusted effects of the intervention group (vs. the usual care group) from intention-to-treat linear and logistic random effect models on changes in Patient Activation Measure (PAM) activation scores and levels at 12-weeks and 52-weeks post-baseline.

		Crude	Adjusted
PAM activation score at 12-weeks[1]	Estimate	0.28	0.26
	p-value	0.503	0.517
	95% CI	(−0.55, 1.11)	(−0.53, 1.05)
PAM activation score at 52-weeks[1]	Estimate	−0.28	−0.36
	p-value	0.51	0.377
	95% CI	(−1.12, 0.56)	(−1.17, 0.45)
PAM activation score change from baseline to 12-weeks[2]	Estimate	0.57	0.48
	p-value	0.226	0.283
	95% CI	(−0.36, 1.5)	(−0.39, 1.35)
PAM activation score change from baseline to 52-weeks[2]	Estimate	0.04	−0.16
	p-value	0.927	0.723
	95% CI	(−0.88, 0.97)	(−1.03, 0.72)
PAM activation scores increased from baseline to 12-weeks[3]	AOR	1.03	1.02
	p-value	0.682	0.729
	95% CI	(0.90, 1.17)	(0.90, 1.16)
PAM activation scores increased from baseline to 52-weeks[3]	AOR	0.89	0.89
	p-value	0.116	0.106
	95% CI	(0.77, 1.03)	(0.77, 1.03)
PAM activation level increased from baseline to 12-weeks[4]	AOR	1.00	1.00
	p-value	0.992	0.958
	95% CI	(0.88, 1.14)	(0.88, 1.15)
PAM activation level increased from baseline to 52-weeks[4]	AOR	0.91	0.91
	p-value	0.215	0.190
	95% CI	(0.79, 1.05)	(0.79, 1.05)

Note: The covariates included study site, patient age, sex, race, education, self-rated health, history of chronic obstructive pulmonary disease, history of depression, smoking status, alcohol use, engagement in weight-bearing exercise, fractures after age 40, parental hip fractures after age 50, patient prior DXA testing, fracture risk, prior diagnoses for osteopenia or osteoporosis, and current or former osteoporosis medication use.

[1]These are regression coefficients from linear random effect models on increases in PAM activation scores at 12- and 52-weeks without adjustment to baseline PAM score.

[2]These are regression coefficients from linear random effect models on PAM activation scores at 12- and 52 weeks with adjustment for the baseline activation score.

[3]These are adjusted odds ratios from logistic random effect regression models on any increase in PAM activation scores at 12- and 52-weeks with adjustment for PAM activation scores at baseline.

[4]These are adjusted odds ratios from logistic random effect models on any increase in PAM levels at 12- and 52-weeks with adjustment for PAM activation scores at baseline.