INSTRUCTIONS FOR USE
This Medical Policy provides assistance in interpreting UnitedHealthcare benefit plans. When deciding coverage, the enrollee specific document must be referenced. The terms of an enrollee’s document (e.g., Certificate of Coverage (COC) or Summary Plan Description (SPD) and Medicaid State Contracts) may differ greatly from the standard benefit plans upon which this Medical Policy is based. In the event of a conflict, the enrollee’s specific benefit document supersedes this Medical Policy. All reviewers must first identify enrollee eligibility, any federal or state regulatory requirements and the enrollee specific plan benefit coverage prior to use of this Medical Policy. Other Policies and Coverage Determination Guidelines may apply. UnitedHealthcare reserves the right, in its sole discretion, to modify its Policies and Guidelines as necessary. This Medical Policy is provided for informational purposes. It does not constitute medical advice.

UnitedHealthcare may also use tools developed by third parties, such as the MCG™ Care Guidelines, to assist us in administering health benefits. The MCG™ Care Guidelines are intended to be used in connection with the independent professional medical judgment of a qualified health care provider and do not constitute the practice of medicine or medical advice.

BENEFIT CONSIDERATIONS

Essential Health Benefits for Individual and Small Group:
For plan years beginning on or after January 1, 2014, the Affordable Care Act of 2010 (ACA) requires fully insured non-grandfathered individual and small group plans (inside and outside of Exchanges) to provide coverage for ten categories of Essential Health Benefits (“EHBs”). Large group plans (both self-funded and fully insured), and small group ASO plans, are not subject to the requirement to offer coverage for EHBs. However, if such plans choose to provide coverage for benefits which are deemed EHBs (such as maternity benefits), the ACA requires all dollar limits on those benefits to be removed on all Grandfathered and Non-Grandfathered plans. The determination of which benefits constitute EHBs is made on a state by state basis. As such, when using this guideline, it is important to refer to the enrollee’s specific plan document to determine benefit coverage.
COVERAGE RATIONALE

Serum or urine collagen crosslinks or biochemical markers are unproven and not medically necessary to assess risk of fracture, predict bone loss or assess response to antiresorptive therapy.

There is insufficient evidence in the clinical literature demonstrating the clinical utility of bone turnover markers and the impact on patient management.

APPLICABLE CODES

The Current Procedural Terminology (CPT®) codes and Healthcare Common Procedure Coding System (HCPCS) codes listed in this policy are for reference purposes only. Listing of a service code in this policy does not imply that the service described by this code is a covered or non-covered health service. Coverage is determined by the enrollee specific benefit document and applicable laws that may require coverage for a specific service. The inclusion of a code does not imply any right to reimbursement or guarantee claims payment. Other policies and coverage determination guidelines may apply. This list of codes may not be all inclusive.

<table>
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<tr>
<th>CPT® Code</th>
<th>Description</th>
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<tr>
<td>82523</td>
<td>Collagen cross links, any method</td>
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DESCRIPTION OF SERVICES

Collagen crosslinks, part of the matrix of bone upon which bone mineral is deposited, are biochemical markers the excretion of which provide a quantitative measurement of bone resorption. Elevated levels of urinary collagen crosslinks indicate elevated bone resorption. Elevated bone resorption contributes to age-related and postmenopausal loss of bone leading to osteoporosis and increased risk of fracture. The collagen crosslinks assay can be performed by immunoassay or by high performance liquid chromatography (HPLC).

Biochemical markers of bone turnover in the serum or urine are sometimes used to assess risk of fracture, predict bone loss or assess response to antiresorptive therapy. Biochemical markers such as pyridinoline, telopeptides and urinary cross-linked N-telopeptide of type I collagen (NTx) (which measure bone resorption) and osteocalcin and bone alkaline phosphatase (which measure bone formation) are obtained through minimally invasive tests involving serum and urine, making biochemical markers an attractive method for determining risk of fracture and for osteoporosis management. Specifically, the information obtained could potentially be used to measure the rate of bone loss, assist in determining osteoporosis management, monitor changes in bone metabolism and density resulting from therapy, and manage osteoporosis therapy as needed for the individual patient. Biochemical markers are controversial because of the complexity of interpreting the values for individual patients related to the intricacies inherent in bone metabolism, and the lack of standardization, which has led to unacceptable levels of variation between processing laboratories.

CLINICAL EVIDENCE

A Hayes report concluded that overall, evidence of low to moderate quality suggests that urinary cross-linked N-telopeptide of type I collagen (NTx) has limited clinical utility for the management of treatment for osteoporosis. Several studies of fair to good quality consistently reported that bisphosphonate or HRT treatment significantly reduced (i.e., had a positive effect) NTx levels relative to baseline. However, the minimum threshold for therapeutic responsiveness has not yet been established for resorptive biomarkers, so it is not clear to what extent the significant reductions in NTx translate into meaningful therapeutic responses in individual osteoporosis patients. Several studies of poor to good quality with generally consistent results suggest a weak (negative) correlation between change in bone mineral density and change in NTx levels.
following treatment with bisphosphonates. There is insufficient evidence regarding the usefulness of NTx for selecting appropriate patients for specific osteoporosis treatment. Similarly, there is insufficient evidence regarding the ability of NTx to guide clinicians in making appropriate decisions during the course of treatment in patients with osteoporosis. Additional well-designed clinical trials are necessary to establish the clinical utility of NTx for patient selection, clinical decision-making during treatment, and to more fully establish the role of NTx as a means of influencing patients to adhere, persist, and/or comply with treatment. In addition, well-designed diagnostic studies using bone mineral density (BMD) as a gold standard reference sample are necessary to establish the capability of NTx to accurately quantify the magnitude of patient response to osteoporosis treatment, and to accurately classify low, moderate, and high responders to treatment (Hayes, 2010a; updated 2012).

A separate Hayes report concluded that overall evidence suggests that the urinary cross-linked N-telopeptide of type I collagen (NTx) has very limited clinical utility for the diagnosis of osteoporosis, assessment of the rate of bone loss and assessment of the risk of fracture in postmenopausal women. Specifically, evidence was scarce and of low quality regarding the diagnostic accuracy of urinary NTx to correctly identify osteoporotic postmenopausal women. There was no evidence regarding the use of urinary NTx as an adjunct to the conventional diagnostic tool of bone mineral density (BMD). Evidence pertaining to the role of urinary NTx to assess the rate of bone loss in postmenopausal women was limited and of low quality. Only indirect evidence was available to draw conclusions regarding the ability of NTx to predict the rate of bone loss. These studies reported data stratified by quartiles and correlation analyses, showing a very weak to weak (negative or inverse) correlation between NTx and BMD values. Stronger correlation coefficients between changes in NTx and BMD were reported for older postmenopausal women. Diagnostic accuracy of NTx to determine the rate of bone loss was relatively low for sensitivity, specificity, and positive predictive value, and somewhat higher for negative predictive value. The body of evidence regarding the role of urinary NTx to predict the risk of fracture was of low to moderate quality and results of the individual studies were conflicting. Given the low-quality evidence, no conclusions can be drawn regarding the clinical utility of urinary NTx alone or in conjunction with BMD to accurately assess the rate of bone loss or to assess the risk of fracture in treated or untreated postmenopausal women, or in women not yet diagnosed with the disease. The measurement of urinary NTx is minimally invasive, and no important safety issues were reported in any of the available evidence. Additional well-designed clinical trials are necessary to establish the clinical utility of NTx to diagnose osteoporosis, to predict the rate of bone loss, to assess the risk of fracture, and to determine the role of NTx as a potential adjunct to BMD assessment in the overall management of osteoporosis in men and postmenopausal women (Hayes, 2010b; updated 2012).

A systematic review published in 2012 by Biver and colleagues reviewed the literature on bone turnover markers for diagnosing osteoporosis and predicting fracture risk. To be included in the review, studies needed to report at least one bone turnover marker and report either BMD or fracture assessment. In post-menopausal women, the markers that have been studied the most and also have the strongest negative correlations with BMD are alkaline phosphatase (ALP), osteocalcin (OC), type 1 cross-linked C-telopeptide (CTx), and type 1 cross-linked N-telopeptide (NTx). The investigators addressed the issue of the potential association between bone turnover markers and prevalent asymptomatic vertebral fractures. A pooled analysis was conducted only for the marker osteocalcin (OC). When findings from 3 studies were pooled, there was not a statistically significant mean difference in OC levels in patients with and without vertebral fractures. The authors also reported that bone turnover markers were not able to reliably distinguish primary osteoporosis from secondary causes. There was a high degree of heterogeneity among the published studies included in this review. According to these data, the clinical usefulness of bone turnover markers for diagnosing osteoporosis is low due to patient variability and other factors that can influence bone turnover marker levels.
An Agency for Healthcare Research and Quality (AHRQ) report addressed the role of markers of bone turnover for identifying women at risk of bone loss, guiding initial treatment decisions, or monitoring response to therapy. The report found the following:

1. No single marker or cluster of markers accurately predicted the results of densitometry in individuals. Densitometry measures current bone status, whereas markers measure the process of turnover.

2. No marker was associated with increased fracture risk consistently across all studies. One study provides evidence that using markers in conjunction with densitometry may increase predictability, but this result has not been otherwise confirmed.

3. As to whether markers can help select patients for treatment, studies correlating marker results and bone loss indicated no clear trend. Furthermore, sensitivity and specificity of markers were too low to be useful for the purpose of selecting patients for treatment.

4. Some studies found better test accuracy when a combination of two or more markers and/or other risk factors was used to predict bone loss.

5. There is a small correlation between response to therapy as measured by densitometry and marker results, but no marker is accurate enough to reliably identify those individuals who will fail to respond to treatment (Nelson et al., 2001).

Guidelines on osteoporosis prevention and treatment from the University of Michigan Health System (2011) make no recommendation for the use of biochemical markers in osteoporosis. The guidelines state: “Biochemical markers of bone resorption are used in research and may be used clinically to assess the effectiveness of antiresorptive therapy. In the latter setting, a decrease in these markers to premenopausal levels usually occurs after two to three months of therapy. Some data suggest that elevated levels of bone resorption markers in older women are an independent risk factor for fractures. However, bone markers are not a reliable predictor of BMD, and are not a substitute for DXA in women at risk. Generally, their use in the diagnosis of osteoporosis is not recommended.”

Gertz et al. (1994) evaluated an immunoassay using an ELISA approach for measuring urinary excretion of cross-linked N-telopeptides of type 1 collagen as a specific measure of bone resorption. The assay was applied to 65 early postmenopausal women who participated in a placebo-controlled trial of the aminobisphosphonate, alendronate sodium. Baseline cross-linked peptide excretion correlated significantly (p < 0.001) with baseline total urine lysylpyridinoline and serum osteocalcin, but not with the other biochemical markers. The investigators concluded that measurement of the urinary cross-linked N-telopeptides of type I collagen by this particular ELISA approach appears promising as a simple and reliable method to assess overall bone resorption.

Parviainen et al. (1999) studied the clinical usefulness of urinary bone resorption markers in postmenopausal women with symptomatic osteoporosis in a randomized double-blind placebo controlled study in which patients were daily treated for 24 months either with a hormone analogue plus 800 mg calcium (n = 14), or with placebo plus 800 mg calcium (n = 19). All resorption markers decreased for both groups during the 2 years the study was conducted. After 2 years there was, however, a significant increase in bone density both in the spine and in the femoral neck in the women with hormone treatment. In the control group a significant increase (P = 0.0012) in the spine, whereas a non-significant decrease in the femoral neck was observed. The investigators concluded that measurement of urinary cross-linked peptides derived from Type I collagen (NTx and DPD) might be a useful biochemical method of observing the positive clinical effect (i.e. reduction in bone resorption) following hormone replacement therapy in postmenopausal fracture patients.

Marcus et al. (1999) assessed the associations of eight bone turnover markers (BTMs) with baseline and 1-year percentage changes in lumbar spine and hip bone mineral density (BMD) of 293 postmenopausal women undergoing treatment with hormone replacement therapy (HRT) (n=293) or placebo (n=54). In 239 women assigned to treatment with estrogen alone or with estrogen plus progestins (active treatment), mean percentage changes for all markers decreased significantly and remained below baseline values through 3 years of study, whereas mean
percentage changes for 54 women assigned to the placebo group showed no significant change from baseline in any marker. The investigators concluded that BTMs are not a surrogate for BMD to identify women with low bone mass and that they offer little useful information for predicting BMD changes for individual untreated or HRT-treated postmenopausal women.

Trento et al. (2009) investigated the clinical role of the bone turnover markers type I collagen C telopeptide (CTX), osteocalcin (OC) and bone-specific alkaline phosphatase (BAP) in the assessment of bone status in 200 women with postmenopausal osteoporosis. Serum bone turnover markers were measured at the initial visit and correlated with spine and femur bone mineral density (BMD), determined on dual-energy X-ray absorptiometry. No correlation was found between serum levels of OC and BAP and vertebral or femur BMD when analyzed against biochemical markers of bone turnover and age, age at menopause, body mass index (BMI) and BMD. S-CTX levels were higher in women with osteoporosis than in women with normal or moderately low (osteopenic) values of BMD. The sensitivity and specificity versus spine BMD were 73.9% and 41.6% for s-CTX, 40.4% and 80.6% for BAP, and 68.3% and 39% for OC, respectively. The sensitivity and specificity versus femur BMD were 76.9% and 40.4% for s-CTX, 23.8% and 88.3% for BAP, and 80.4% and 53.3% for OC, respectively. The authors concluded that determination of s-CTX, BAP and OC is of limited clinical value in the initial evaluation of bone status in menopausal women.

Lukaszkiewicz et al. (2008) evaluated the correlation between bone resorption and bone formation markers to assess bone turnover rate and qualify an individual postmenopausal woman as a possible elevated bone turnover (EBT) subject. A total of 320 postmenopausal women were enrolled at seven clinical sites in this cross-sectional observational study. The group was a random sample of the population. BMD measurements of the lumbar spine, total hip, trochanter, and femoral neck regions were performed. Bone resorption and formation rates were evaluated by serum levels of C-terminal telopeptide of type I collagen (CTX) and osteocalcin (OC), respectively. Using logistic regression to correlate the concentrations of CTX and OC it was possible not only to distinguish the EBT subgroup, but also to construct a simple nomogram for easy classification of individual patients as possible EBT subjects. EBT patients showed generally decreased BMD values and increased bone formation and resorption rates. The investigators concluded that evaluation of both CTX and OC levels enables a more proper indication for EBT.

An observational study that included 432 Japanese elderly women who were not receiving any drug treatment for osteoporosis were followed for 5.2 +/- 3.3 years. Vertebral fractures and bone mineral density were assessed at baseline and then at 1- to 2-year intervals or at indication of any symptom. Two types of collagen metabolites were measured at baseline: urinary N-terminal telopeptide of type I collagen (NTX), a marker of pyridinium cross-link, and urinary pentosidine, a nonenzymatic collagen cross-link produced by AGEs. A total of 97 incident vertebral fractures on 72 subjects were observed. Simple regression analysis using Cox’s hazards model showed that log-transformed urinary NTX and pentosidine are significant risk factors for time-dependent incidence of vertebral fractures, in addition to the traditional risk factors (age, lumbar bone mineral density, and number of prevalent vertebral fractures). However, urinary excretion of pentosidine was a significant predictor of incident vertebral fracture after adjustment for other traditional risk factors. The present data suggest that AGE-related collagen cross-link is a novel risk for vertebral fracture (Shiraki et al., 2008).

Perier and colleagues (2007) noted that Homocysteine (Hcy) has recently been described as an independent risk factor for osteoporotic fractures in the elderly. These investigators prospectively followed women belonging to the OFELY study during a mean follow-up of 10 years. Homocysteine was measured at baseline in 671 post-menopausal women from the OFELY cohort (mean age of 62.2 +/- 9 years). Incident clinical fractures were recorded during annual follow-up and vertebral fractures were evaluated with radiographs every 4 years. A Cox proportional hazards model based on time to first fracture was used to calculate hazard ratios for quartiles of Hcy values. Mean Hcy was 10.6 +/- 3.4 mumol/L, increasing with age. After adjustment for age, Hcy was significantly associated with physical activity, calcium intake, serum
albumin and serum creatinine; but not with bone turnover markers and bone mineral density (BMD). During a mean follow-up of 10 years, 183 fractures occurred among 134 women. After adjustment for age, the overall relative risk of fracture for each one SD increment of Hcy was 1.03 (95% CI 0.87 - 1.31). Fracture risk was higher in women with Hcy in the highest quartile without adjustment but no longer after adjustment for age. The authors concluded that Hcy is not an independent risk factor of osteoporotic fractures in healthy post-menopausal women from the OFELY cohort with a broad age range.

Rhew et al. (2008) examined the relationship of baseline Hcy levels with BMD and incidence of fractures over 2 years in women with and without systemic lupus erythematosus (SLE). Women with SLE (n = 100) and without SLE (n = 100) were matched according to age (+/- 5 years), race, and menopausal status. Data were collected from 1997 to 2004, including hip, lumbar spine (L-spine), and distal forearm BMD, serum Hcy levels, and a self-administered questionnaire on osteoporosis risk factors, medications and symptomatic fractures at baseline and 2-year follow-up. Analyses were performed to compare Hcy levels, BMD, and incident fractures and to evaluate the relationship of Hcy with BMD and incident fractures in both groups. Mean Hcy +/- SD was higher (p < 0.001) in women with SLE (9.88 +/- 3.8 micromol/L) than in women without SLE (7.98 +/- 2.6 micromol/L). Women with SLE had significantly lower L-spine BMD Z-scores, while hip BMD Z-scores and distal forearm BMD T-scores were non-significantly lower than in women without SLE. No significant correlations were observed between Hcy and BMD in either group. A total of 13 women with SLE experienced new fractures, while four women without SLE had new fractures over 2 years (p = 0.035); however, there was no association between Hcy levels and incident fractures in either group. The authors concluded that women with SLE had significantly greater baseline Hcy, lower L-spine BMD, and more new fractures over 2 years, compared with women without SLE. However, Hcy levels were not significantly associated with BMD and did not predict new fractures in women with or without SLE over 2 years.

Loddenkemper et al. (2006) concluded there was a correlation between ostease/crosslinks with BMD, but the correlation coefficients were low. A general recommendation for the routine use of a specific bone marker in patients with rheumatic diseases on glucocorticoid therapy cannot be made from a cost-benefit point of view mainly because of limited predictive power (low correlation coefficients, incomplete correlation with different sites of BMD measurement).

It has been hypothesized that the ability to assess an early decrease in bone turnover that predicts increases in spine and hip BMD might provide the motivation some patients need to remain compliant. Future studies are needed to determine if early assessment improves long-term patient compliance or uncovers poor compliance, thereby aiding the physician in maximizing the benefits of therapy. (Greenspan et al., 1998)

Several nonrandomized controlled trials also discussed the potential value of bone turnover markers (Meier, 2005; Worsfold, 2004; Garnero, 2000; Iki, 2006). However, no outcomes studies were found in which patient management was changed by the results of bone turnover markers. Proponents of collagen crosslinks point out that even though results of BMD testing are the single best predictor of fracture risk, determinations of bone turnover may be an independent predictor of fracture risk. However, it is unclear how that knowledge would change patient management and whether such treatment decisions would ultimately result in a reduction in the fracture risk in individual patients. Collagen crosslinks have also been studied in diseases associated with high bone turnover rates, such as glucocorticoid-induced osteoporosis, hyperparathyroidism or renal osteodystrophy. Similar to age-related osteoporosis, it is unclear how levels of collagen crosslinks as a marker of bone turnover might be used in the management of the patient.

Bergmann et al. (2009) published evidence-based guidelines for the Belgian Bone Club on the use of biochemical markers for osteoporosis. The guidelines state that although the correlation between bone mineral density (BMD) and bone turnover markers (BTM is statistically significant), BTM cannot be used as predictive markers of BMD in an individual patient. Both are independent predictors of fracture risk, but BTM can only be used as an additional risk factor in the decision to
Current data do not support the use of BTM to select the optimal treatment. However, they can be used to monitor treatment efficacy.

A National Institutes of Health (NIH) statement indicates that bone remodeling can be assessed by the measurement of surrogate markers of bone turnover in the blood or urine. These markers include bone-specific alkaline phosphatase and osteocalcin, which are indices of bone formation, and the urinary levels of pyridinolines and deoxypyridinolines and serum and urine levels of type I collagen telopeptides (CTX and NTX), which are indices of bone resorption. The level of these markers may identify changes in bone remodeling within a relatively short time interval (several days to months) before changes in BMD can be detected. However, according to available data, marker levels do not predict bone mass or fracture risk and are only weakly associated with changes in bone mass. Therefore, they are of limited utility in the clinical evaluation of individual patients. Despite these limitations, markers have been shown in research studies to correlate with changes in indices of bone remodeling and may provide insights into mechanisms of bone loss (NIH, 2000).

**Professional Societies**

**American Academy of Family Physicians (AAP)**
AAP recommends against the use of biochemical markers for the diagnosis of osteoporosis (Sweet et al. 2009).

**American Association of Clinical Endocrinologists (AACE)**
In 2010, the American Association of Clinical Endocrinologists (AACE) considered biochemical markers in its guidelines for preventing and treating postmenopausal osteoporosis. AACE noted that biochemical markers of bone turnover cannot be used to diagnose osteoporosis but may be useful for assessing fracture risk in elderly patients and for assessing therapeutic responses to antiresorptive agents. Their use in clinical practice, however, is limited by high in vivo and assay variability (resorption markers), poor predictive ability in individual patients and lack of evidence-based thresholds for clinical decision making (Watts et al., 2010).

**American College of Obstetricians and Gynecologists (ACOG)**
An ACOG practice bulletin addresses the use of biochemical markers to predict bone turnover in osteoporosis. The guideline states that bone turnover markers cannot be used to diagnose osteoporosis, and the usefulness of markers as an incentive for adherence has been questioned (ACOG, 2012).

**International Osteoporosis Foundation (IOF)/ International Federation of Clinical Chemistry and Laboratory Medicine (IFCC)**
The IOF and the IFCC evaluated the clinical potential of bone turnover markers (BTMs) in the prediction of fracture risk and for monitoring treatment. Research evidence suggests that BTMs may provide information on fracture risk independently from BMD, so that fracture risk prediction might be enhanced by their inclusion in assessment algorithms. The potential use of BTMs to predict the response to treatments for osteoporosis in the individual patient is also of great interest. Treatment-induced changes in specific markers account for a substantial proportion of fracture risk reduction. However, there is still a need for stronger evidence on which to base practice in both situations. IOF/IFCC recommends one bone formation marker (serum procollagen type I N propeptide, s-PINP) and one bone resorption marker (serum C-terminal cross-linking telopeptide of type I collagen, s-CTX) to be used as reference markers and measured by standardized assays in observational and intervention studies in order to enlarge the international experience of the application of markers to clinical medicine and to help resolve uncertainties over their clinical use (Vasikaran et al., 2011).

**National Osteoporosis Foundation (NOF)**
In a general guidance statement, the NOF states that biochemical markers of bone turnover may:

- Predict risk of fracture independently of bone density
• Predict extent of fracture risk reduction when repeated after 3-6 months of treatment with FDA-approved therapies
• Predict magnitude of BMD increases with FDA-approved therapies
• Predict rapidity of bone loss
• Help determine adequacy of patient compliance and persistence with osteoporosis therapy
• Help determine duration of 'drug holiday' and when and if medication should be restarted (Data are quite limited to support this use but studies are underway) (NOF, 2013).

North American Menopause Society (NAMS)
A NAMS guideline on managing osteoporosis in postmenopausal women states that the routine use of biochemical markers of bone turnover is not generally recommended (NAMS, 2010).

U.S. FOOD AND DRUG ADMINISTRATION (FDA)
The FDA regulates commercially marketed tests and test systems such as bone markers and categorizes these test systems to one of three Clinical Laboratory Improvement Act (CLIA) of 1988 regulatory categories (i.e., waived, moderate, or high) based on their potential risk to public health. Commercially marketed tests that have received 510(k) marketing clearance can be accessed through the 510(k) database (search by manufacturer or test system name) or through the CLIA database search by manufacturer, test system, or analyte name). Laboratories that use their own tests but do not market the kits to others are subject to the standards of the Clinical Laboratory Improvement Act (CLIA), but not to FDA marketing regulations.

Additional Products
OsteoMark® NTx (N-telopeptides of bone collagen); Metra DPD; Serum CrossLaps® in vitro assay; VITROS® Immunodiagnostics System; MicroVue™ DPD

CENTERS FOR MEDICARE AND MEDICAID SERVICES (CMS)
Medicare does cover collagen crosslinks testing when criteria are met. Refer to the National Coverage Determination (NCD) for Collagen Crosslinks, any Method (190.19). Local Coverage Determinations (LCDs) do not exist at this time.

(Accessed January 10, 2014)

REFERENCES


Gertz BJ, Shao P, Hanson DA, et al. Monitoring bone resorption in early postmenopausal women...


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**POLICY HISTORY/REVISION INFORMATION**

<table>
<thead>
<tr>
<th>Date</th>
<th>Action/Description</th>
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<tr>
<td>04/01/2014</td>
<td>• Reorganized policy content</td>
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<tr>
<td></td>
<td>• Added benefit considerations language for <em>Essential Health Benefits for Individual and Small Group</em> plans to indicate:</td>
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on a state by state basis; as such, when using this guideline, it is important to refer to the enrollee's specific plan document to determine benefit coverage

- Updated coverage rationale; added language to indicate the unproven services are “not medically necessary”
- Updated supporting information to reflect the most current description of services, clinical evidence, CMS information and references
- Archived previous policy version 2013T0419I