Requirements, by any of the following methods:

• *Regulations.gov: http://www.regulations.gov.* Submit comments via the Federal eRulemaking portal by inputting the OMB Control number. Select the link ''Submit a Comment'' that corresponds with ''Information Collection 9000–0061, Transportation Requirements''. Follow the instructions provided at the ''Submit a Comment'' screen. Please include your name, company name (if any), and ''Information Collection 9000–0061, Transportation Requirements'' on your attached document.

• *Mail:* General Services Administration, Regulatory Secretariat Division (MVCB), 1800 F Street NW, Washington, DC 20405. ATTN: Ms. Mandell/IC 9000–0061, Transportation Requirements.

Instructions: Please submit comments only and cite Information Collection 9000–0061, Transportation Requirements, in all correspondence related to this collection. Comments received generally will be posted without change to *regulations.gov,* including any personal and/or business confidential information provided. To confirm receipt of your comment(s), please check *regulations.gov,* approximately two-to-three business days after submission to verify posting (except allow 30 days for posting of comments submitted by mail).

FOR FURTHER INFORMATION CONTACT: Mr. Curtis E. Glover, Sr., Procurement Analyst, Office of Governmentwide Acquisition Policy, GSA 202–501–1448 or via email at *curtis.glover@gsa.gov.*

SUPPLEMENTARY INFORMATION:

A. Purpose

FAR Part 47 contains policies and procedures for applying transportation and traffic management considerations in the acquisition of supplies. The FAR part also contains policies and procedures when acquiring transportation or transportation-related services. Generally, contracts involving transportation require information regarding the nature of the supplies, method of shipment, place and time of shipment, applicable charges, marking of shipments, shipping documents and other related items.

Contractors are required to provide the information in accordance with the following FAR Part 47 clauses: 52.247–29 through 52.247–44, 52.247–48, 52.247–52, and 52.247–64. The information is used to ensure that: (1) Acquisitions are made on the basis most advantageous to the Government and; (2) supplies arrive in good order and condition, and on time at the required place.

B. Annual Reporting Burden

Respondents: 65,000.
Responses per Respondent: 22.
Annual Responses: 1,430,000.
Hours Per Response: .05.
Total Burden Hours: 71,500.

C. Public Comments

Public comments are particularly invited on: Whether this collection of information is necessary; whether it will have practical utility; whether our estimate of the public burden of this collection of information is accurate, and based on valid assumptions and methodology; ways to enhance the quality, utility, and clarity of the information to be collected; and ways in which we can minimize the burden of the collection of information on those who are to respond, through the use of appropriate technological collection techniques or other forms of information technology.

Obtaining Copies of Proposals: Requesters may obtain a copy of the information collection documents from the General Services Administration, Regulatory Secretariat Division (MVCB), 1800 F Street NW, Washington, DC 20405, telephone 202–501–4755. Please cite OMB Control No. 9000–0061, Transportation Requirements, in all correspondence.

Dated: April 4, 2018.

Lorin S. Curit,

Director, Federal Acquisition Policy Division, Office of Governmentwide Acquisition Policy, Office of Acquisition Policy, Office of Governmentwide Policy.

[FR Doc. 2018–07371 Filed 4–10–18; 8:45 am]

BILLING CODE 6820–EP–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Centers for Medicare & Medicaid Services

[CMS–3353–N]

Medicare Program; Reconciling National Coverage Determinations on Positron Emission Tomography (PET) Neuroimaging for Dementia

AGENCY: Centers for Medicare & Medicaid Services (CMS), HHS.

ACTION: Notice.

SUMMARY: In accordance with the court order on July 19, 2016 (Kort v. Burwell), this notice provides further explanation on the National Coverage Determinations for positron emission tomography (PET) neuroimaging for dementia.

FOR FURTHER INFORMATION CONTACT: Linda Gousis, (410) 786–8616.

SUPPLEMENTARY INFORMATION:

I. Background

On July 19, 2016, the United States District Court for the District of Columbia issued an order requiring the Secretary of Health and Human Services (HHS) to further explain one aspect of a National Coverage Determination (NCD) decision memorandum issued by the Centers for Medicare & Medicaid Services (CMS). *Kort* v. *Burwell,* 209 F.Supp.3d 98 (D.D.C. 2016). In particular, the court called for CMS to explain how its 2013 NCD denying coverage for a beta amyloid positron emission tomography scan (amyloid PET) [1] could be reconciled with an earlier 2004 NCD relating to fluorodeoxyglucose (FDG) positron emission tomography (PET) (FDG PET).[2] We issued the NCDs under our authority to interpret the ''reasonable and necessary'' statutory standard in section 1862(a)(1)(A) of the Social Security Act (the Act) as it applies to coverage of items and services in the Medicare program. In this notice, we describe the key differences between the two NCDs. We relied on the existing record in preparing this document.

II. Provisions of the Notice

In accordance with the Court's order, we explain why CMS covers one diagnostic test for specific patients, while covering the other only in the context of a clinical study (*Kort,* 115). Briefly, the differences arose from the type of assessment the test provided; predictive value of the test; and consensus panels' conclusions about the use of the tests.

A. Summary of the NCDs

The 2004 NCD for FDG PET resulted in narrow coverage of the diagnostic test for specific subpopulations of patients meeting narrowly defined criteria (CMS

[1] CMS, *Decision Memo for Beta Amyloid Positron Emission Tomography in Dementia and Neurodegenerative Disease (CAG–00431N);* 2013 September 27. Available from: *https://www.cms.gov/medicare-coverage-database/details/nca-decision-memo.aspx?NCAId=265* (accessed on June 22, 2017). Note that amyloid PET is referred to in the 2013 NCD as ''βA PET'' or ''amyloid PET'' interchangeably. In this document, we are using ''amyloid PET''; however, quotes may refer to it by the similar terms.

[2] CMS, *Decision Memo for Positron Emission Tomography (FDG) and Other Neuroimaging Devices for Suspected Dementia (CAG–00088R);* 2014 September 15. Available from: *https://www.cms.gov/medicare-coverage-database/details/nca-decision-memo.aspx?NCAId=104* (accessed on June 22, 2017).

2004, 32).[3] We determined that the ''scan is reasonable and necessary in patients with documented cognitive decline of at least six months and a recently established diagnosis of dementia who meet diagnostic criteria for both Alzheimer's disease (AD) and fronto-temporal dementia (FTD), who have been evaluated for specific alternate neurodegenerative diseases or causative factors, and for whom the cause of the clinical symptoms remains uncertain'' (CMS 2004, 3).

The 2013 amyloid PET NCD resulted in non-coverage of amyloid PET for dementia and neurodegenerative disease; however, coverage was made available in the context of a clinical study. There, one amyloid PET scan per patient would be covered through coverage with evidence development (CED) pursuant to section 1862(a)(1)(E) of the Act (CMS 2013, 4). The diagnostic test is covered under certain research parameters ''in two scenarios: (1) To exclude Alzheimer's disease (AD) in narrowly defined and clinically difficult differential diagnoses, such as AD versus frontotemporal dementia (FTD); and (2) to enrich clinical trials seeking better treatments or prevention strategies, by allowing for selection of patients on the basis of biological as well as clinical and epidemiological factors'' (CMS 2013, 4).

B. Kort v. Burwell Summary

The plaintiffs in *Kort* were beneficiaries who exhibited symptoms of cognitive impairment but did not have a diagnosis for their illness. They wanted amyloid PET scans because they thought the scans would help their doctors make a differential diagnosis. The court determined that the amyloid PET NCD failed to adequately explain how the decision denying coverage for amyloid PET could be reconciled with the earlier decision approving coverage of FDG PET in certain contexts. The court noted, ''[t]he similarities between FDG PET and BA scans are manifest. Both are diagnostic tests that involve the use of a PET scan and a radiopharmaceutical tracer. Both are indicated for use on overlapping patient populations exhibiting symptoms of cognitive impairment. And, although neither test can affirmatively diagnose a disease, both have diagnostic value as a tool for differentially diagnosing patients who exhibit symptoms associated with several different diseases'' (*Kort,* 114–115). Without vacating the 2013 NCD, the Court remanded ''the Decision Memo so that the agency can evaluate in the first instance whether its coverage decisions can be reconciled'' (*Kort,* 115).

C. Analytic Framework for Reviewing Clinical Evidence

We evaluated the relevant clinical evidence to determine whether or not the evidence is sufficient to support a finding that an item or service is reasonable and necessary for the Medicare population, which consists largely of adults 65 years of age and older (CMS 2004, 13 and CMS 2013, 13). This process was discussed in the methodological principles for both NCDs. The critical appraisal of the evidence enables CMS to determine to what degree the agency is confident that the intervention will improve health outcomes for beneficiaries (CMS 2004, 13 and CMS 2013, 13).

Specifically for diagnostic imaging tests, the overall assessment focuses on whether use of the test to guide patient management and treatment improves health outcomes (also referred to as clinical utility). Before appropriately reaching a consideration of outcomes, two fundamental properties of diagnostic tests need to be established: (1) the test accurately and reliably measures the intended analyte, factor, or component (also referred to as analytic validity); and (2) the test accurately and reliably identifies the condition or disorder of interest (also referred to as clinical validity). Outcomes such as change in patient management due to diagnostic tests and accuracy, sensitivity, and specificity are also of interest to CMS (CMS 2004, 14 and CMS 2013, 30).

D. Review of the Clinical Evidence for FDG and Amyloid PET

While both diagnostic tests use a PET scan, there is a distinction in the tracers used for the scans: FDG provides a physiologic (functional) assessment of the brain since it highlights glucose metabolism; meanwhile, beta amyloid tracers such as florbetapir (Amyvid®) and flutemetamol (Vizamyl®) provide a molecular (anatomic) assessment since they bind to amyloid β plaques (CMS 2004, 5 and CMS 2013, 11). In both coverage analysis, we focused on whether the PET scans can accurately and reliably identify dementias, including AD, and whether use of the scans to guide management and treatment improves meaningful health outcomes (CMS 2004, 14 and CMS 2013, 14). We focused on these because numerous mechanism of action studies have shown that PET scans can accurately and reliably detect radionuclide tracers that tag nitrogen, oxygen, glucose, and amyloid.[4]

Ultimately, we determined that evidence for FDG PET for differential diagnosis of dementias was more compelling and substantiated than for amyloid PET when the same analytic framework was applied to these diagnostic imaging tests. There were several reasons for CMS finding FDG PET more compelling. The ability of the FDG PET test to accurately and reliably identify the disorder of interest is better established and accepted than for molecular PET scans, such as beta amyloid (CMS 2004, 8). Since the 1980s, functional assessment of the brain using one of a number of tracers, such as ones for blood flow, oxygen utilization, and glucose metabolism, has been used to diagnose dementia. Among these, FDG is a glucose analog and behaves similar to glucose in the cell. Glucose metabolism may be viewed as an indicator of cell activity. Used as a PET tracer, FDG will indicate the cell activity. In the brain, function as shown by cell activity (glucose metabolism or FDG tagging) may be used to differentiate causes of dementia (CMS 2004, 7). For example, in frontal lobe dementia, imaging tests have shown marked hypometabolism (darker areas) of the frontal or temporal lobes with sparing of parietal lobes. In patients with Alzheimer's disease, there is typically hypometabolism bilaterally in the temporal and parietal lobes (CMS 2004, 5, 7, and 33). Additionally, ''the presumed higher specificity of FDG PET for detecting metabolic patterns correlated with FTD could decrease the number of false positive results for AD and consequently increase the number of true positives for FTD to inform

[3] In this document, page numbers for the decision memorandum citations are based off of the page number at the bottom of the page on the PDF version which is available for download from web page provided in the previous footnotes for this document. Click on the ''Need a PDF?'' icon on the right side of the screen to obtain a PDF.

[4] Petersen et al. Practice parameter: Early detection of dementia: Mild cognitive impairment (an evidence-based review), Report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology. May 2001; Neuroimaging in the Diagnosis of Alzheimer's Disease and Dementia. Expert panel convened by the Neuroscience and Neuropsychology of Aging Program, National Institute on Aging (NIA), HHS. April 5, 2004. *https://www.cms.gov/Medicare/Coverage/DeterminationProcess/downloads/id104d.pdf* (accessed on August 9, 2017); and D Matchar, S Kulasingam, B Huntington, M Patwardhan, L Mann. Technology Assessment: Positron emission tomography, single photon emission computed tomography, computed tomography, functional magnetic resonance imaging, and magnetic resonance spectroscopy for the diagnosis and management of Alzheimer's disease. Duke Center for Clinical Health Policy Research and Evidence Practice Center. December 2001. *https://www.cms.gov/Medicare/Coverage/DeterminationProcess/downloads/id9TA.pdf* (accessed August 9, 2017). (CMS 2004, 43 and 46)

patient management and caregiver counseling'' (CMS 2004, 35).

In contrast to the evidence supporting use of FDG PET, there were uncertainties regarding the use of amyloid PET. The presence or absence of amyloid in the brain has been considered in diagnosis of AD, but it is not diagnostic because some normal individuals also have amyloid plaques (CMS 2013, 10). Amyloid tracers bind to and statically mark amyloid plaque providing an anatomic or structural assessment (location and concentration) but do not provide information on cell activity or brain function. This is an inherent limitation of anatomic assessments compared to functional assessments because the hallmark of dementia is an abnormal decline in cognitive function (CMS 2013, 7). Thus, the premise that the test accurately and reliably identifies the disorder is reduced in amyloid imaging compared to functional imaging, such as FDG, due to the different mechanisms of action. Additionally, the ability of amyloid PET scans to diagnose AD is inherently reduced by the pathophysiologic characteristics of AD since the presence extracellular amyloid β is only one of two specific findings required for the diagnosis of AD. The second key factor is the presence of intracellular neurofibrillary tangles (NFTs) consisting of abnormal tau proteins. Amyloid tracers do not show the presence of NFTs or abnormal tau proteins, which are not detected by any commercially available radionuclide tracer (CMS 2013, 10). In addition, findings based on postmortem investigation and studies (pathophysiologic alternations in brain biopsies) may not directly translate to factors that may be used to make a clinical diagnosis of patients with dementia.

The FDG PET NCD acknowledged that AD-type physiology may be present in normal individuals with normal cognitive function; therefore, a positive amyloid PET scan does not necessarily mean the individual has AD (CMS 2004, 5). As subsequently noted in the amyloid PET decision memo nine years later, ''[A]myloid plaques are seen in other diseases, such as dementia with Lewy bodies, cerebral amyloid angiopathy, Parkinson's disease, Huntington's disease, and inclusion body myositis. Amyloid plaques can also be detected in cognitively normal older adults. Autopsy studies demonstrate that approximately 33% of older individuals (20–65% depending on age) who are cognitively normal have amyloid accumulation at levels consistent with AD pathology (Hulette 1998, Price 1999, Knopman 2003, Rowe 2010)'' (CMS 2013, 10).

The reliability of test is a necessary component for determining health outcomes or clinical utility. The foundation of clinical utility for functional PET scans, like FDG PET, is better established than anatomic PET scans, like amyloid PET. While direct, high quality evidence on clinical utility of FDG PET for dementia was not found in published literature at the time of the 2004 decision, there were related studies that showed clinical utility of FDG PET for other treatable causes of cognitive impairment or dementia such as cerebrovascular disease, certain inherited diseases, and metabolic conditions that could possibly be diagnosed with FDG PET, and then treated with proven therapies to improve health outcomes (CMS 2004, 32, 37). At the time of the amyloid PET NCD, there was no published evidence of clinical utility similar to what was reviewed for FDG PET, and there were no related studies suggesting that amyloid PET would be helpful in the differential diagnosis of AD and FTD (CMS 2013, 14). Further, because amyloid PET does not specifically diagnose other conditions, the clinical utility or improved health outcomes associated with other diseases is not applicable.

Since the mid-2000s, a number of clinical trials of different therapies that target amyloid have failed to produce results of improvement in health outcomes (CMS 2013, 61).[5] FDG PET did not have the same negative trials at the time of our 2004 decision.

E. Determining the Predictive Value of Amyloid PET Compared to FDG PET

We did not have the same concerns regarding false positives using FDG PET to differentially diagnose AD as we did with amyloid PET. The predictive value of the amyloid PET scan cannot be based solely on its capability to ''rule out'' AD, because there is also the risk of positively diagnosing patients with Alzheimer's when they do not have it. Conversely, for a patient faced with the possibility of having Alzheimer's, a negative amyloid PET result could be reassuring (CMS 2013, 52–53). However, such reassurance would not change clinical management because the patient may still have AD. If a clinician did not have ''a convincing clinical picture [of AD], work up to exclude other diagnosable and potentially treatable diseases should proceed anyway (as it would if an amyloid scan were negative). The unavailability of an amyloid scan does not change that logic'' (CMS 2013, 52).

At the same time, the amyloid PET scan portends great risk because there is no evidence for what a positive scan means in specific patients since they can have amyloid plaques but not have AD. At the Medicare Evidence Development & Coverage Advisory Committee (MEDCAC) meeting held specifically on amyloid PET on January 30, 2013, one expert speaker mentioned that he believed that a patient with mild cognitive impairment (MCI) and a positive amyloid PET scan had Alzheimer's disease and that many other experts agreed with him (MEDCAC 2013, 31, 53).[6] However, no published clinical trials, studies, consensus publications, or further MEDCAC discussions identified whether, for amyloid PET, ''objectively-defined subpopulations of patients with cognitive impairment for which the scan (alone or combined with other tests) may be more or less appropriate. Yet there are many subtypes of MCI, and some (*e.g.,* amnestic MCI) may be more relevant than others. Furthermore, there is evidence that the same level of amyloid burden detected by a scan may mean something very different in say, a 66 year-old compared to an 86 year-old (*e.g.,* Le Couteur 2013, Laforce 2011). Yet the [Amyloid Imaging Task Force] AIT is silent about such potentially important distinctions'' (CMS 2013, 33). (The AIT was a consensus panel that developed recommendations for use of amyloid PET.)

We concluded in the amyloid PET NCD that ''widespread clinical use of the scan both in many types of patients with unexplained MCI, and to make a positive diagnosis of Alzheimer's disease (despite insufficient evidence on

[5] See also Peterson RC. Early Diagnosis of Alzheimer's Disease: Is MCI Too Late? Current Alzheimer Research. 2009; 6(4):329; Petersen RC, Smith G, Waring S, et al. Mild cognitive impairment: clinical characterization and outcome. Archives of Neurology. 1999;56:303–8; National Institute on Aging (NIA) and the Reagan Institute. Consensus recommendations for the postmortem diagnosis of Alzheimer's disease. The National Institute on Aging, and Reagan Institute Working Group on Diagnostic Criteria for the Neuropathological Assessment of Alzheimer's Disease. Neurobiology of Aging. 1997 Jul-Aug;18(4 Suppl):S1–2; and Technology Evaluation Center (TEC), Blue Cross Blue Shield. Beta Amyloid Imaging with Positron Emission Tomography (PET) for Evaluation of Suspected Alzheimer's Disease or Other Causes of Cognitive Decline. 2013 February;27(5).

[6] Medicare Evidence Development & Coverage Advisory Committee (MEDCAC), Meeting: Beta Amyloid Positron Emission Tomography (PET) in Dementia and Neurodegenerative Disease, Meeting Transcript; 2013 January 30. Available from: *https://www.cms.gov/Regulations-and-Guidance/Guidance/FACA/downloads/id66d.pdf* (accessed on June 22, 2017). (CMS 2013, 79, 80, and 82)

the clinical meaning of a positive scan) has great potential to lead to over-diagnosis of Alzheimer's disease. Such misdiagnosis of Alzheimer's disease portends real harm to our beneficiaries (La Couteur 2013), and this must be considered in our coverage decision'' (CMS 2013, 33).

''False positive'' test results, widely considered by radiologists as the bane of diagnostic imaging, are of special concern for amyloid PET. The following are scenarios that contrast the impacts of negative, positive, and false positive test results. For example, if a patient were to get a computed tomography (CT) study of the chest, abdomen, and pelvis to ''rule out'' cancer, and if the CT study were negative, that indeed would be reassuring to the patient. However, if the study were positive for an enlarged lymph node, liver lesion, or some questionable pulmonary nodule, these findings could be followed up by biopsy, surgical resection, or assessing for progression of disease on a close follow-up CT. In contrast, a completely different clinical scenario follows amyloid PET. Those options to further explore findings common for other ''positive'' diagnostic tests do not exist. Providers cannot do a biopsy, resection, or close follow up of amyloid imaging after a positive amyloid scan.

Concern about false positive test results was not a major factor in the 2004 decision memorandum on FDG PET. Based off of an external technical assessment that helped inform the 2004 decision memorandum, we concluded that ''FDG–PET testing would reduce the number of false positive results'' (CMS 2004, 16). FDG PET has the ability to diagnose patients with disease (dementias, not only Alzheimer's) since it is a functional test and measures glucose metabolism (activity) as noted earlier. Based on the patterns of uptake (cellular function indicating activity), a differential diagnosis between FTD (characteristic hypometabolism in the frontal lobe of the brain) versus AD (characteristic hypometabolism in temporal and parietal lobes of the brain) versus normal patterns (no hypometabolism) may be made. In our FDG PET decision, we noted, ''Patients with FTD generally tend to show bifrontal and bitemporal hypoperfusion in single photon emission computerized tomography (SPECT) or glucose hypometabolism in FDG PET scans. In contrast, temporoparietal defects are predominant in AD'' (CMS 2004, 7).

In contrast, the false positive results were a greater concern with amyloid PET (CMS 2013, 48–50), since amyloid plaques may be present in many individuals with normal cognitive function. As noted earlier, the presence of amyloid (positive test) by itself does not diagnose AD since the diagnosis of AD is based on the presence of both amyloid and tau proteins on autopsy. A positive amyloid PET does not allow a differential diagnosis between FTD versus AD versus an individual with normal cognitive function since amyloid is a structural component and does not indicate function.

F. Expert Consensus in Making Evidence-based NCDs

Two expert panels, in 2002, the Medicare Coverage Advisory Committee (MCAC) [7] Diagnostic Imaging Panel,[8] and, in 2004, the National Institute on Aging (NIA) agreed on a narrow conditioned clinical use for the FDG PET scan (MCAC–DIP 2002, 122, 196–197 and CMS 2004, 35). The expert panel convened by NIA believed the existing evidence warranted use of FDG–PET for a limited number of cases including differential diagnosis of AD and FTD (NIA 2004, 32, 35, 45, 48, and 51–52). For these reasons, in 2004 we had confidence in the plausibility of downstream health outcomes for a narrow indication for FDG PET for differential diagnosis of AD and FTD.

In contrast to the uniform consensus for FDG PET, in 2013, two expert panels, the AIT [9] and MEDCAC,[10] manifestly disagreed about the clinical use of the amyloid PET scan (CMS 2013, 33 and MEDCAC 2013, 55). While the AIT noted amyloid imaging may be appropriate in progressive unexplained or unclear clinical presentations (Johnson 2013, e6), the MEDCAC did not find sufficient evidence for CMS to support outright coverage of amyloid PET (MEDCAC 2013, 248, 250). This different degree of consensus between 2004 and 2013 was a contributing factor in our decisions. However, our evidence-based approach to coverage determinations does not rely on consensus alone. As explained in the 2013 NCD, ''two credible expert panels—the AIT and the MEDCAC—produced differing consensuses. That's why, in the well-established process of scientific evaluation, evidence must be evaluated to determine the strength of the consensus opinion'' (CMS 2013, 33). At the time the amyloid PET NCD was finalized, there was no evidence to support or refute the consensus opinions. CED for amyloid PET supported the needed development of evidence for future evaluation. Therefore, based on the evidence reviewed as described above and the conclusions of the expert panels, we came to differing conclusions because the evidence for FDG PET for a narrowly defined patient population was better established than for amyloid PET.

G. Summary

As required by the court order that accompanied the *Kort* opinion, this document further explains why we reached different conclusions with respect to section 1862(a)(1)(A) of the Act in the NCDs for FDG PET and amyloid PET. Both decisions were based on the available evidence according to our analytic framework described herein. Based on that evidence, we created narrow coverage for a small patient population with extensive patient eligibility criteria and provider requirements for FDG PET. For amyloid PET, the totality of the evidence available was not sufficient to demonstrate that the test produced diagnostic value as a tool for differentially diagnosing patients who exhibit symptoms associated with AD or FTD. Therefore, we established coverage for amyloid PET in the context of a clinical study setting with patient and provider eligibility criteria under the authority of section 1862(a)(1)(E) of the Act.

III. Collection of Information Requirements

This document does not impose information collection requirements, that is, reporting, recordkeeping or third-party disclosure requirements. Consequently, there is no need for review by the Office of Management and Budget under the authority of the Paperwork Reduction Act of 1995 (44 U.S.C. 3501 *et seq.*).

[7] The MCAC was the predecessor to the MEDCAC.

[8] MEDCAC, Meeting: Positron Emission Tomography (FDG) for Alzheimer's Disease/Dementia (Diagnostic Imaging Panel), Meeting Transcript; 2002 January 10. *https://www.cms.gov/Regulations-and-Guidance/Guidance/FACA/downloads/id2a.pdf* (accessed June 22, 2017).

[9] Johnson et al., *Appropriate use criteria for amyloid PET: A report of the Amyloid Imaging Task Force, the Society of Nuclear Medicine and Molecular Imaging, and the Alzheimer's Association,* Alzheimer's and Dementia; 2013 January. *http://www.alz.org/research/downloads/appropriate_use_criteria_for_amyloid_PET_Alz_and_Dem_January_2013.pdf* (accessed June 22, 2017).

[10] MEDCAC, Meeting: Beta Amyloid Positron Emission Tomography (PET) in Dementia and Neurodegenerative Disease; 2013 January 30. *https://www.cms.gov/medicare-coverage-database/details/medcac-meeting-details.aspx?MEDCACId=66&year=2013&bc=AAAIAAAAAAAAAA%3d%3d&* (accessed June 22, 2017).

Dated: March 16, 2018.

Seema Verma,

Administrator, Centers for Medicare & Medicaid Services.

Dated: April 5, 2018.

Alex M. Azar II,

Secretary, Department of Health and Human Services.

[FR Doc. 2018–07410 Filed 4–10–18; 8:45 am]

BILLING CODE 4120–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. FDA–2017–N–4561]

Advisory Committee; Bone, Reproductive and Urologic Drugs Advisory Committee, Renewal

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice; renewal of advisory committee.

SUMMARY: The Food and Drug Administration (FDA) is announcing the renewal of the Bone, Reproductive and Urologic Drugs Advisory Committee by the Commissioner of Food and Drugs (the Commissioner). The Commissioner has determined that it is in the public interest to renew the Bone, Reproductive and Urologic Drugs Advisory Committee for an additional 2 years beyond the charter expiration date. The new charter will be in effect until March 23, 2020.

DATES: Authority for the Bone, Reproductive and Urologic Drugs Advisory Committee will expire on March 23, 2020, unless the Commissioner formally determines that renewal is in the public interest.

FOR FURTHER INFORMATION CONTACT: Kalyani Bhatt, Division of Advisory Committee and Consultant Management, Center for Drug Evaluation and Research, Food and Drug Administration, 10903 New Hampshire Ave., Bldg. 31, Rm. 2417, Silver Spring, MD 20993–0002, 301–796–9001, email: *BRUDAC@fda.hhs.gov.*

SUPPLEMENTARY INFORMATION: Pursuant to 41 CFR 102–3.65 and approval by the Department of Health and Human Services pursuant to 45 CFR part 11 and by the General Services Administration, FDA is announcing the renewal of the Bone, Reproductive and Urologic Drugs Advisory Committee (the Committee). The Committee is a discretionary Federal advisory committee established to provide advice to the Commissioner.

The Committee advises the Commissioner or designee in discharging responsibilities as they relate to helping to ensure safe and effective drugs for human use and, as required, any other product for which FDA has regulatory responsibility.

The Committee reviews and evaluates data on the safety and effectiveness of marketed and investigational human drug products for use in the practice of osteoporosis and metabolic bone disease, obstetrics, gynecology, urology, and related specialties, and makes appropriate recommendations to the Commissioner.

The Committee shall consist of a core of 11 voting members including the Chair. Members and the Chair are selected by the Commissioner or designee from among authorities knowledgeable in the fields of osteoporosis and metabolic bone disease, obstetrics, gynecology, urology, pediatrics, epidemiology, or statistics and related specialties. Members will be invited to serve for overlapping terms of up to 4 years. Almost all non-Federal members of this committee serve as Special Government Employees. The core of voting members may include one technically qualified member, selected by the Commissioner or designee, who is identified with consumer interests and is recommended by either a consortium of consumer-oriented organizations or other interested persons. In addition to the voting members, the Committee may include one non-voting member who is identified with industry interests.

Further information regarding the most recent charter and other information can be found at *https://www.fda.gov/AdvisoryCommittees/CommitteesMeetingMaterials/Drugs/ReproductiveHealthDrugsAdvisoryCommittee/ucm107572.htm* or by contacting the Designated Federal Officer (see **FOR FURTHER INFORMATION CONTACT**). In light of the fact that no change has been made to the committee name or description of duties, no amendment will be made to 21 CFR 14.100.

This document is issued under the Federal Advisory Committee Act (5 U.S.C. app.). For general information related to FDA advisory committees, please check *https://www.fda.gov/AdvisoryCommittees/default.htm.*

Dated: April 5, 2018.

Leslie Kux,

Associate Commissioner for Policy.

[FR Doc. 2018–07437 Filed 4–10–18; 8:45 am]

BILLING CODE 4164–01–P

DEPARTMENT OF HEALTH AND HUMAN SERVICES

Food and Drug Administration

[Docket No. FDA–2018–N–0981]

Preparation for International Cooperation on Cosmetics Regulation Twelfth Annual Meeting; Public Meeting

AGENCY: Food and Drug Administration, HHS.

ACTION: Notice of public meeting.

SUMMARY: The Food and Drug Administration (FDA or we) is announcing the following public meeting entitled ''International Cooperation on Cosmetics Regulation (ICCR)—Preparation for ICCR–12 Meeting.'' The purpose of the public meeting is to invite public input on various topics pertaining to the regulation of cosmetics. We may use this input to help us prepare for the ICCR–12 meeting that will be held July 10 to 12, 2018, in Tokyo, Japan.

DATES: The public meeting will be held on June 7, 2018, from 2 p.m. to 4 p.m. See the **SUPPLEMENTARY INFORMATION** section for registration date and information.

ADDRESSES: The public meeting will be held at the Food and Drug Administration, Center for Food Safety and Applied Nutrition, 5001 Campus Dr., Wiley Auditorium (first floor), College Park, MD 20740.

FOR FURTHER INFORMATION CONTACT: Jonathan Hicks, Office of Cosmetics and Colors, Food and Drug Administration, 5001 Campus Dr. (HFS–125), College Park, MD 20740, *jonathan.hicks@fda.hhs.gov,* 240–402–1375.

SUPPLEMENTARY INFORMATION:

I. Background

The intention of the ICCR multilateral framework is to pave the way for the removal of regulatory obstacles to international trade while maintaining global consumer protection. The purpose of the meeting is to invite public input on various topics pertaining to the regulation of cosmetics. We may use this input to help us prepare for the ICCR–12 meeting that will be held July 10 to 12, 2018, in Tokyo, Japan.

ICCR is a voluntary international group of cosmetics regulatory authorities from Brazil, Canada, the European Union, Japan, and the United States of America. These regulatory authority members will engage in constructive dialogue with their relevant cosmetics industry trade