EPA SCIENTIFIC ADVISORY COMMITTEE ON CHEMICALS
CHARGE TO THE PANEL – 1-BROMOPROPANE (1-BP)
CASRN: 106-94-5

As amended by the Frank R. Lautenberg Chemical Safety for the 21st Century Act on June 22, 2016, the Toxic Substances Control Act (TSCA), requires the U.S. Environmental Protection Agency (EPA) to conduct risk evaluations on existing chemicals. In December of 2016, EPA published a list of the initial ten chemical substances that are the subject of the Agency's chemical risk evaluation process (81 FR 91927), as required by TSCA. 1-BP is one of the first ten chemical substances to undergo a peer review by the Science Advisory Committee on Chemicals (SACC). In response to this requirement, EPA has prepared and published a draft risk evaluation for 1-BP. EPA has solicited comments from the public on the draft and will incorporate them as appropriate, along with comments from peer reviewers, into the final risk evaluation.

The draft risk evaluation contains the following components:

Presentation of chemistry and physical-chemical properties
Characterization of uses/sources
Systematic review
Environmental fate and transport assessment
Occupational exposure assessment
Environmental, and consumer exposure assessment
Environmental hazard assessment
Human health hazard assessment
Risk characterization
Risk determination

The focus of this meeting is to conduct the peer review of the Agency's draft risk evaluation of 1-BP. At the conclusion of the peer review process, EPA will use the reviewers' comments/recommendations, as well as public comment, to finalize the risk evaluation.

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CHARGE QUESTIONS:

EPA is seeking SACC advice on the clarity and scientific underpinnings of the overall assessment. The peer review should consider whether the conclusions presented in the draft risk evaluation are clearly presented, scientifically supported and based on the best available scientific information. The SACC should also consider whether the methods employed to generate the information are reasonable for and consistent with the intended use of the information. As per TSCA, where unreasonable risks are identified, once finalized the risk evaluation will be used to support rulemaking to mitigate identified risks.

Throughout the peer review, the SACC should be mindful that TSCA now requires that EPA use data and/or information in a manner consistent with the "best available science" and that EPA base decisions on the "weight of the scientific evidence". EPA's Final Rule, *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* (82 FR 33726), defines ''best available science'' as science that is reliable and unbiased. This involves the use of supporting studies conducted in accordance with sound and objective science practices, including, when available, peer reviewed science and supporting studies and data collected by accepted methods or best available methods (if the reliability of the method and the nature of the decision justifies use of the data). The Final Rule also defines the "weight of the scientific evidence" as a systematic review method, applied in a manner suited to the nature of the evidence or decision, that uses a pre-established protocol to comprehensively, objectively, transparently, and consistently identify and evaluate each stream of evidence, including strengths, limitations, and relevance of each study and to integrate evidence as necessary and appropriate based upon strengths, limitations, and relevance.

Below, are a set of charge questions for each major element of the risk evaluation. The SACC is expected to consider questions and issues raised during public comment as part of its deliberations.

1. Content and Organization

EPA's Final Rule, *Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act* (82 FR 33726) stipulates the process by which EPA is to complete risk evaluations under the Frank R. Lautenberg Chemical Safety for the 21st Century Act. To that end, EPA has completed a draft risk evaluation for 1-BP.

As part of this risk evaluation for 1-BP, EPA conducted an assessment of potential environmental, occupational and consumer exposures. The evaluation considered reasonably available information, including manufacturing, import, processing, distribution in commerce, use, and disposal information. It is important that the information presented in the risk evaluation and accompanying documents are clear and concise and describe the process in a scientifically credible manner.

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Q 1.1	*Please comment on the overall content, organization, and presentation of the draft risk evaluation of 1-BP.*
Q 1.2	*Please provide suggestions for improving the clarity of the information presented in the documents.*

2. *Systematic Review* (Draft Risk Evaluation and Supplemental Files)

To meet the TSCA scientific standards, EPA applied systematic review approaches and methods to support the draft risk evaluation of 1-BP. Information on the approaches and/or methods is described in the draft risk evaluation as well as the following documents:

- Application of Systematic Review in TSCA Risk Evaluations (U.S. EPA, 2018a)
- Strategy for Conducting Literature Searches for 1-BP: Supplemental Document for the TSCA Scope Document (U.S. EPA, 2017b)
- 1-BP (CASRN: 106-94-5) Bibliography: Supplemental File for the TSCA Scope Document {EPA-HQ-OPPT-2016-0741-0047)}
- Scope of the Risk Evaluation for 1-BP (U.S. EPA, 2017a)
- Problem Formulation for 1-Bromopropane (U.S. EPA, 2018b)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Quality Evaluation of Environmental Fate and Transport Studies (EPA, 2019e)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Extraction Tables for Environmental Fate and Transport Studies (EPA, 2019b)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Quality Evaluation of Environmental Release and Occupational Exposure Data (EPA, 2019f)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Quality Evaluation of Environmental Release and Occupational Exposure Data for Common Sources (EPA, 2019g)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Quality Evaluation for Consumer Exposure (EPA, 2019a)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Extraction for Consumer Exposure (EPA, 2019c)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Quality Evaluation of Ecological Hazard Studies (EPA, 2019d)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Updates to the Data Quality Criteria for Epidemiological Studies (EPA, 2019j)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies – Epidemiologic Studies (EPA, 2019i)
- Draft Risk Evaluation for 1-Bromopropane (1-BP), Systematic Review Supplemental File: Data Quality Evaluation of Human Health Hazard Studies (EPA, 2019h)

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Q 2.1	*Please comment on the approaches and/or methods used to support and inform the gathering, screening, evaluation, and integration of data/information used in the Draft Risk Evaluation for 1-Bromopropane (1-BP).*
Q 2.2	*Please also comment on the clarity of the information as presented related to systematic review and suggest improvements as warranted.*

3. *Occupational Exposure Assessment* (Section 2.3.1 of the Draft Risk Evaluation)

EPA evaluated acute and chronic exposures to workers for conditions of use in industrial and commercial settings. For exposure via the inhalation pathway, EPA quantified occupational exposures for both workers and occupational non-users based on a combination of monitoring data and modeled exposure concentrations. For exposure via the dermal route, EPA modeled exposure for workers, accounting for the effect of volatilization and glove use. EPA assumed dermal exposure would not occur for occupational non-users.

EPA assumed that workers and occupational non-users would be adults of both sexes (>16 and older, including women of reproductive age) based on occupational work permits.

Q 3.1	*Please comment on the approaches and estimation methods, models, and data used in the occupational exposure assessment.*
Q 3.2	*Please provide any specific suggestions or recommendations for alternative data, or estimation methods that could be considered by the Agency for conducting occupational exposure assessment.*

4. *Consumer Exposure Assessment* (Section 2.3.2 of the Draft Risk Evaluation)

EPA evaluated acute inhalation exposure to consumers for the following product use scenarios including adhesive accelerants, general spray cleaners, spot cleaner/stain removers, mold cleaning/release, general cleaners-degreasers, degreasers-electronics, coin/scissors cleaner, automobile AC flush products, and insulation which contain 1-BP. EPA also evaluated acute dermal exposure to consumers using general cleaners-degreasers, coin/scissors cleaner, and automobile AC flush products containing 1-BP. Dermal exposure was evaluated for these three uses based on the assumption that use could be a constant supply of a product in contact with the skin such that evaporation from the skin does not occur (submersion into a pool of product, or a product-soaked rag covering the skin). Consumer uses of the above listed products are not expected to be chronic in nature and therefore EPA did not evaluate chronic inhalation or dermal exposure.

EPA evaluated exposure to consumers in residential settings following acute exposure and considered both users of a product and bystanders within the residence where the product

was used. EPA considered users to be either adult (>21 years of age) or youth (16-20 years of age). A second youth group (11-15 years of age) are included in the evaluation although this age group is not expected to be a significant user of most product uses evaluated. Bystanders within a residence where product is used include individuals of any age group (infants, children, youth, adults, elderly).

Three models (CEM, MCCEM, and IECCU) were used to evaluate acute inhalation exposure depending on the condition of use evaluated. These three models are defined and discussed in detail within the 1-BP risk evaluation. Dermal exposure was evaluated using CEM.

Product specific consumer monitoring information was not identified during the systematic review process, therefore, model inputs related to consumer use patterns (duration of use, mass of product used, room of use, and similar inputs) are based on survey data found in the literature as described and referenced within the 1-BP risk evaluation. Weight fraction of chemical within products are based on product specific SDS sheets. Default values utilized within the models are based on literature reviewed as part of model development as well as EPA's Exposure Factors Handbook.

Q 4.1	*Please comment on the approaches, models, exposure or use information (e.g., information on duration, number of user events, amount used) and estimates for the nine consumer uses evaluated for this risk evaluation.*
Q 4.2	*Please provide any specific suggestions or recommendations for alternative approaches, models, exposure or use information (e.g., information on duration, number of user events, amount used) that could be considered by EPA in developing and /or refining the exposure assumptions and estimates for the nine consumer uses evaluated for this risk evaluation.*
Q 4.3	*Dermal exposure was evaluated using a permeability method within CEM based on the availability of a permeability coefficient found within the literature in a study by NIOSH. The permeability method within CEM does not consider evaporation when estimating exposure which is the primary basis for EPA evaluating dermal exposure only for consumer uses where there is a constant supply of product against the skin during use or a barrier prohibiting evaporation. Please comment on the chosen approach and provide any suggestions or recommendations for alternative approaches, dermal methods, models, or other information which may guide EPA in developing and refining the dermal exposure estimates.*

5. *Environmental Hazard and Risk Characterization* (Sections 3.1 and 4.1 of the Draft Risk Evaluation)

Available data indicates that 1-BP exhibits a moderate environmental hazard to aquatic species. A screening-level analysis of potential risk to aquatic species indicates that expected environmental concentrations are below hazard thresholds for aquatic species. In addition, a

qualitative consideration of physical-chemical properties and the conditions of use in this assessment indicate that risks to sediment-dwelling invertebrate species and terrestrial species are not expected.

Q 5.1	*Only a few environmental test data endpoints (including ECHA) are available in the public domain for 1-BP. Most are from the ECHA website. EPA attempted to obtain the full ECHA studies with no success. Since the studies were in French and Japanese (and no U.S.A. sponsor), EPA decided not to make further attempts to find the studies. Given that the ECHA environmental test data results are in the public domain, EPA decided to use the experimental data. Please comment on the reasonableness of this approach for the environmental hazard assessment of 1-BP.*
Q 5.2	*EPA determined that there are no environmental risks based on a screening-level assessment of risk using environmental hazard data, TRI exposure data, fate information, and physical/chemical properties. Please comment on whether the information presented supports the findings outlined in the draft risk characterization section.*

6. Hazard and Dose-Response Assessments (Section 3.2 of the Draft Risk Evaluation)

EPA considered the adverse human health effects for 1-BP across organ systems and screened to those that are relevant, sensitive, and found in multiple studies. The 1-BP human health hazard systematic review process screened 813 studies and obtained 29 studies that were relevant and applicable to the PECO statement. Five of these studies were unacceptable based on data evaluation criteria. The remaining database of 24 studies included epidemiological studies that examined associations between 1-BP exposure and endpoints related to effects on the nervous system, as well as repeat-dose experimental animal studies. For hazard identification and dose-response, EPA reviewed the evidence for 1-BP toxicity and selected liver toxicity, kidney toxicity, reproductive/developmental toxicity, neurotoxicity, and cancer. Data on toxicity following acute exposures, and genotoxicity were also considered. From these effects, EPA selected endpoints supported by the weight-of-the-scientific evidence for non-cancer and cancer that demonstrated the most robust, sensitive and consistent adverse human health effects for risk characterization, that were amenable to quantitative analysis for dose-response assessment, and that were appropriate toxicological studies to be used for acute and chronic exposure scenarios. EPA used benchmark dose (BMD) modeling where practicable and, when BMD values were adequate, they were used to generate the Point of Departure (POD) for characterizing chronic and acute exposure scenarios. EPA determined that using developmental toxicity and neurotoxicity endpoints for dose-response calculation would be protective of the most sensitive life stages, including the developing fetus for non-cancer PODs and risk estimates.

For the non-cancer assessment, EPA identified liver toxicity, kidney toxicity, reproductive/developmental toxicity, and neurotoxicity in the risk assessment as adverse

human health effects for risk characterization. EPA used these endpoints to calculate PODs to assess non-cancer risks associated with chronic inhalation exposures.

Q 6.1	*As part of the review, please comment on the choice of these endpoints as PODs for assessing risks in humans associated with acute and chronic inhalation exposures to 1-BP. Specifically, are there other data that EPA could have considered for the hazard identification and dose-response associated with acute inhalation exposures? If so, please provide specific data and references. Are there other data that EPA could have considered for the hazard identification and dose response associated with chronic inhalation exposures? If so, please provide specific data and references.*
Q 6.2	*Please comment on the WOE analysis for the choices of non-cancer endpoints for the acute and chronic risk scenarios. Please provide additional data, data interpretation or information that would have informed the WOE analysis and selection of critical studies for the PODs.*

The CSAC Peer Review of the *Application of Systematic Review in TSCA Risk Evaluations* document (U.S. EPA, 2018a) recommended that while the majority of exposures are occurring via inhalation, and inhalation exposure is the most important, dermal exposures might be an important contributor to overall exposure and an estimate for dermal exposure should be included in the evaluation, with gaps/limitations clearly stated to address another potential workplace exposure pathway. Limited toxicological data is available by the oral route, and no repeated-dose toxicity studies by the dermal route were identified on 1-BP. Physiologically based pharmacokinetic/pharmacodynamic (PBPK/PD) models that would facilitate route-to-route extrapolation have not been identified, and there are no relevant kinetic or metabolic information for 1-BP that would facilitate development of dosimetric comparisons. Therefore, when EPA derived HEDs for dermal exposures, the limited oral studies were not used and HEDs for dermal exposures were derived by extrapolating from the inhalation PODs.

Q 6.3	*Please comment on the assumptions, strengths, and weaknesses of this approach for determining dermal PODs in the non-cancer assessment.*

In the 2016 Draft Risk Assessment (U.S. EPA, 2016), decreased live litter size (i.e. reduced number of live pups per litter) was the endpoint selected as most relevant for calculating risks associated with developmental toxicity following chronic, exposures (WIL Research, 2001). A BMR of 5% was used to address the severity of this endpoint (U.S. EPA, 2012). This endpoint choice is a combination of reproductive effects where a BMR 10% relative deviation would be used and developmental effects of post implantation loss which is considered a severe effect like mortality where a BMR of 1% relative deviation would be used and so an intermediate value of 5% was used. The POD for the decreased live litter size was a BMDL of 43 ppm. The CSAC Peer Review of the 2016 Draft Risk Assessment (U.S. EPA, 2016) recommended using nested modeling on this developmental endpoint to account for intra-litter correlations and litter specific covariates. In response to this recommendation, EPA used the BMDS nested dichotomous model, evaluated multiple covariates of dam weight and

number of implantation sites and selected the NCTR model. However, this model can only be applied to increases in effects and therefore, increased post-implantation loss was the endpoint selected as most relevant for calculating risks associated with developmental toxicity following chronic exposures (WIL Research, 2001) using nested modeling. A BMR of 1% was used to address the severity of this endpoint which is considered a severe effect like mortality (U.S. EPA, 2012). The POD for the increased post-implantation loss was a BMDL of 23 ppm.

Q 6.4	*Please comment on the nested modeling approach and the selection of endpoint and whether the risk evaluation has adequately described the use of this model.*

For the cancer risk assessment, EPA derived the inhalation unit risk (IUR) based on lung tumors in female mice. The precise mechanism(s)/mode(s) of action of 1-BP carcinogenesis are not clearly understood. There are, however, an abundance of data, including *in vitro* tests, metabolism across species, SAR and other potential mechanisms of action, that provide a basis for a weight-of-evidence (WOE) evaluation. The WOE evaluation presented in the draft 1-BP Risk Evaluation proposes a mutagenic, and possibly additional, modes of action for carcinogenesis. Other possible mechanisms of action – oxidative stress, immunosuppression, and cell proliferation—could act synergistically to complete the multi-stage process of carcinogenesis. Per EPA Guidelines for Carcinogen Risk Assessment, overall, the totality of the available data/information and the WOE analysis for the cancer endpoint was sufficient to support a mutagenic mode of action for 1-BP carcinogenesis.

Q 6.5	*EPA concluded in the risk assessment that 1-BP carcinogenesis occurs through a mutagenic mode of action (MMOA) based on the totality of the available data/information and the WOE. Please comment whether the cancer hazard assessment has adequately described the WOE regarding the MMOA.*
Q 6.6	*Typically, EPA uses the benchmark dose modeling software (BMDS) with a BMR of 10% and the models are restricted to multistage models or the broader suite of dichotomous models in BMDS and a single best model is chosen for the POD. EPA used an alternative approach to calculate the cancer POD versus the standard approach of choosing best fit model and to assess the impact of model uncertainty. Briefly, EPA used two model averaging approaches (frequentist and Bayesian) considering multiple benchmark dose models to calculate the POD at benchmark response (BMR) levels of 0.1% and 10% and for added and extra risk. Please comment on the assumptions, strengths and weaknesses of the model averaging approach for determining the POD in the cancer assessment.*
Q 6.7	*In agreement with EPA's long-standing approach, all three tumor types from the NTP study (NTP, 2011) were dose-response modeled with multistage models using the typical constrained model coefficients ≥0 (EPA, 2012). Under the U.S. EPA's 2005 cancer guidelines (U.S. EPA, 2005), quantitative risk estimates from cancer bioassay data were calculated by modeling the data in the observed range to estimate a BMCL for a BMR of 10% extra risk, which is generally near the low end of the observable range for standard cancer bioassay data. The*

	BMCs and BMCLs are shown for each of the three cancer datasets. The results for a BMR of 0.1% added risk are presented for comparison. Please comment on the assumptions, strengths and weaknesses of the multistage modeling approach for determining the POD in the cancer assessment.

7. *Human Health Risk Characterization* (Section 4.2 of the Draft Risk Evaluation)

EPA quantified non-cancer risks based on the Margin of Exposure (MOE), which is the product of dividing the scenario specific exposure into the hazard point of departure which is no adverse effect level, based on animal and/or human studies. EPA calculated MOEs for acute or chronic exposures separately based on the appropriate noncancer POD and estimated exposure concentrations adjusted for durations. To determine if unacceptable risks were present for relevant exposure scenarios, the endpoint-specific MOEs were compared to the endpoint-specific benchmark MOEs. The benchmark MOEs were the product of all of the relevant UFs identified for each non-cancer POD. If the MOE estimate was less than the benchmark MOE, the exposure scenario for non-cancer endpoints was interpreted as a human health risk. Cancer risk estimation consisted of multiplying the occupational scenario-specific exposure estimates by the cancer IUR to estimate the extra cancer risk. Extra lifetime cancer risk estimates from 1-BP exposure were compared to benchmark cancer risk levels of 10^{-6}, 10^{-5} and 10^{-4} (i.e., 1 in 10,000, 1 in 100,000 and 1 in 1,000,000).

The Frank R. Lautenberg Chemical Safety for the 21st Century Act (2016; amended TSCA (TSCA §§ 6b[4a]) states that "potentially exposed or susceptible subpopulations" (PESS) be considered in the risk evaluation process. *"The Administrator shall conduct risk evaluations pursuant to this paragraph to determine whether a chemical substance presents an unreasonable risk of injury to health or the environment, without consideration of costs or other non-risk factors, including an unreasonable risk to a potentially exposed or susceptible subpopulation identified as relevant to the risk evaluation by the Administrator, under the conditions of use."* Further, amended TSCA specifically includes infants, children, and pregnant women in its definition of PESS (TSCA §§ 3[12])– *"The term 'potentially exposed or susceptible subpopulation' means a group of individuals within the general population identified by the Administrator who, due to either greater susceptibility or greater exposure, may be at greater risk than the general population of adverse health effects from exposure to a chemical substance or mixture, such as infants, children, pregnant women, workers, or the elderly.''*

EPA interpreted the endpoint of decreases in live litter size following exposure to 1-BP before and during gestation, as a surrogate for frank developmental effects, as relevant to humans per EPA's Guidelines for Developmental Toxicity Risk Assessment. EPA used this endpoint to calculate a point of departure (POD) to assess non-cancer risks associated with acute inhalation exposures to 1-BP.

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Q 7.1	*Please comment on the assumptions, strengths and weaknesses of the MOE approaches used to estimate the non-cancer risks to workers and occupational non-users (e.g. adults of reproductive age) following acute inhalation exposures to 1-BP, including the MOEs presented in the document. Specifically, please suggest alternative data that could be used. Please comment on the selection of uncertainty factor values in deriving the benchmark MOE for acute inhalation exposures.*
Q 7.2	*Please comment on the assumptions, strengths and weaknesses of the MOE approaches used to estimate the non-cancer risks to consumers following acute inhalation exposures to 1-BP, including the MOEs presented in the document. Specifically, please suggest alternative data that could be used. Please comment on the selection of uncertainty factor values in deriving the benchmark MOE for acute inhalation exposures.*
Q 7.3	*Please comment on the assumptions, strengths and weaknesses of the MOE approaches used to estimate the non-cancer risks to workers and occupational non-users following chronic inhalation exposures to 1-BP, including the MOEs presented in the document. Please comment on the selection of uncertainty factor values in deriving the benchmark MOE for chronic inhalation exposures.*
Q 7.4	*Please comment on the assumptions, strengths and weaknesses of the approach used to estimate extra lifetime cancer risks to workers which EPA-derived from an inhalation unit risk based on lung tumors in female mice for estimating incremental or extra individual lifetime cancer risk.*
Q 7.5	*Please comment on whether the risk characterization has adequately described the assumptions, uncertainties and data limitations in the methodology used to assess risks from 1-BP. Please comment on whether this information and risk conclusions are presented in a logical, transparent manner and provide suggestions that could increase clarity in the risk characterization.*
Q 7.6	*Please comment on whether the risk characterization has adequately identified and characterized the "potentially exposed or susceptible subpopulations" (PESS) based on a thorough review of the available 1-BP exposure and health effects data on both potentially exposed and biological susceptible subpopulations.*

8. General Risk Characterization (Sections 4.1, 4.2, and 4.3 of the Draft Risk Evaluation)

After consideration of all information identified by EPA that pertains to 1-BP, EPA concluded that 1-BP presents an unreasonable risk of injury to health or the environment under the conditions of use. EPA made these determinations considering risk to potentially exposed or susceptible subpopulations identified as relevant, under the conditions of use without considering costs or other non-risk factors.

Q 8.1	*Please comment on the objectivity of the underlying data used to support the risk characterizations and the sensitivity of EPA's conclusions to analytic assumptions.*
Q 8.2	*Please comment on the characterization of uncertainties and assumptions including whether EPA has presented a clear explination of underlying assumptions, accurate contextualization of uncertainties and, as appropriate, the probabilities associated with both optimistic and pessimistic projections, including best-case and worst-case scenarios.*
Q 8.3	*Please provide information on additional uncertainties and assumptions that EPA has not adequately presented.*
Q 8.4	*Please comment on whether the information presented supports the findings outlined in the draft risk characterization section. If not, please suggest alternative approaches or information that could be used to develop a risk finding in the context of the requirements of EPA's Final Rule, Procedures for Chemical Risk Evaluation Under the Amended Toxic Substances Control Act (82 FR 33726).*

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