EUROPEAN CHEMICALS AGENCY

~~Confidential~~

Helsinki, 12 January 2021

Addressee: [redacted]

Decision number: CCH-D-2114538555-43-01/F
Substance name: Butyl glycollate
EC number: 230-991-7
CAS number: 7397-62-8
Registration number: [redacted]
Submission number subject to follow-up evaluation: [redacted]
Submission date subject to follow-up evaluation: 17 January 2020

DECISION TAKEN UNDER ARTICLE 42(1) OF THE REACH REGULATION

By decision CCH-D-2114365547-39-01/F of 14 July 2017 ("the original decision") ECHA requested you to submit information by 21 January 2020 in an update of your registration dossier.

Based on Article 42(1) of Regulation (EC) No 1907/2006 (the 'REACH Regulation'), ECHA examined the information you submitted with the registration update specified in the header above, and concludes that

Your registration still does not comply with the following information requirement(s):

1. **Pre-natal developmental toxicity study (Annex X, Section 8.7.2.; test method: EU B.31./OECD TG 414) in a second species (rabbit), oral route with the registered substance;**

2. **Extended one-generation reproductive toxicity study (Annex X, Section 8.7.3.; test method: EU B.56./OECD TG 443) in rats, oral route with the registered substance specified as follows:**
   - **Ten weeks premating exposure duration for the parental (P0) generation;**
   - **Dose level setting shall aim to induce some toxicity at the highest dose level;**
   - **Cohort 1A (Reproductive toxicity);**
   - **Cohort 1B (Reproductive toxicity) without extension to mate the Cohort 1B animals to produce the F2 generation.**

You are therefore still required to provide this information requested in the original decision.

The reasons of this decision are set out in Appendix 1. The procedural history is described in Appendix 2. Advice and further observations are provided in Appendix 3.

The respective Member State competent authority (MSCA) and National enforcement authority (NEA) will be informed of this decision. They may consider enforcement actions to secure the implementation of the original decision and exercise the powers reserved to them under Article

126 of Regulation No 1907/2006 (penalties for non-compliance)[1].

Appeal

This decision can be appealed to the Board of Appeal of ECHA within three months of its notification. An appeal, together with the grounds thereof, shall be submitted to ECHA in writing. An appeal has suspensive effect and is subject to a fee. Further details are described under http://echa.europa.eu/regulations/appeals.

Approved[2] under the authority of Christel Schilliger-Musset, Director of Hazard Assessment

[1] See paragraphs 61 and 114 of the judgment of 8 May of the General Court of the European Court of Justice in Case T-283/15 Esso Raffinage v. ECHA

[2] As this is an electronic document, it is not physically signed. This communication has been approved according to ECHA's internal decision-approval process.

Appendix on general considerations

(i) Assessment of the Grouping of substances and read-across approach under Annex XI, Section 1.5.

You seek to adapt the following standard information requirements by applying a read-across approach in accordance with Annex XI, Section 1.5:

- Pre-natal developmental toxicity study in a second species (Annex X, Section 8.7.2.)
- Extended one-generation reproductive toxicity study (Annex X, Section 8.7.3.)

ECHA has considered the scientific and regulatory validity of your read-across approach in general before assessing the specific standard information requirements in the following appendices.

Grouping of substances and read-across approach

Annex XI, Section 1.5. specifies two conditions which must be fulfilled whenever a read-across approach is used. Firstly, there needs to be structural similarity between substances which results in a likelihood that the substances have similar physicochemical, toxicological and ecotoxicological properties so that the substances may be considered as a group or category. Secondly, it is required that the relevant properties of a substance within the group may be predicted from data for reference substance(s) within the group.

Additional information on what is necessary when justifying a read-across approach can be found in the ECHA Guidance[3] and related documents[4, 5].

Predictions for toxicological properties

You have provided a read-across justification document in IUCLID Section 13.

You read-across between the structurally similar substances, Ethane-1,2-diol (ethylene glycol, CAS 107-21-1, EC No 203-473-3) as source substance and the Substance as target substance.

You have provided the following reasoning for the prediction of toxicological properties: *"based on the well-established and experimentally shown metabolism of esters, glycollic acid and n-butanol were selected as suitable read-across substances for butyl glycollate. These substances represent metabolic/chemical breakdown products of butyl glycollate. Ethylene glycol is selected as an appropriate source substance because it metabolizes to glycollic acid. Metabolic pathways for butyl glycollate and for ethylene glycol thus have the same main metabolite, i.e. glycollic acid."*

Furthermore, you state that "*Comparing the target organ and adverse effects of butyl glycollate, ethylene glycol, and glycollic acid shows that all three substances cause similar adverse effects in the kidneys (i.e. the same pattern of toxicological activity) [...] This corroborates the presence*

[3] Guidance on information requirements and chemical safety assessment Chapter R.6: QSARs and grouping of Chemicals. 2008 (May) ECHA, Helsinki. 134. pp. Available online: https://echa.europa.eu/documents/10162/13632/information_requirements_r6_en.pdf/77f49f81-b76d-40ab-8513-4f3a533b6ac9

[4] Read-Across Assessment Framework (RAAF). 2017 (March) ECHA, Helsinki. 60 pp. Available online: Read-Across Assessment Framework (https://echa.europa.eu/support/registration/how-to-avoid-unnecessary-testing-on-animals/grouping-of-substances-and-read-across)

[5] Read-across assessment framework (RAAF) - considerations on multi-constituent substances and UVCBs. 2017 (March) ECHA, Helsinki. 40 pp. Available online: https://doi.org/10.2823/794394

of similar metabolites, which then further supports use of these substances as read across source substances [...] Following the enzymatic hydrolysis of butyl glycollate via esterase to its metabolites, reproductive and developmental effects, if present, would most likely be related to the glycollic acid."

Finally, you consider that "*The hydrolysis is considered rapid as the half-life (t1/2) of butyl glycollate was estimated on average as 24 minutes (liver) and > 120 minutes (small intestines). The t1/2 for ethylene glycol has been estimated in a feeding study as between 1.4 and 1.9 hours*".

ECHA understands that you predict the properties of the Substance using a read-across hypothesis which is based on the formation of common (bio)transformation products. The properties of your Substance are predicted to be quantitatively equal to those of the source substance.

ECHA notes the following shortcoming with regards to prediction of toxicological properties.

Supporting information

Annex XI, Section 1.5 of the REACH Regulation states that "*physicochemical properties, human health effects and environmental effects or environmental fate may be predicted from data for reference substance(s)*". For this purpose "*it is important to provide supporting information to strengthen the rationale for the read-across*"[6]. The ECHA Guidance further states[7] "*In certain instances, the metabolism of the parent compound within barrier tissue (e.g. lung or gut tissue) occurs so rapidly that the initial primary metabolite is the predominant chemical found within the blood. Under these circumstances data from hazard identification studies conducted with that primary metabolite itself can be used to identify hazards for the parent compound.*"
The set of supporting information should allow to verify the crucial aspects of the read-across hypothesis and establish that the properties of the Substance can be predicted from the data on the source substance(s).

Supporting information must include toxicokinetic information on the formation of the common compound and information on the impact of exposure on the parent compound(s) on the prediction.

As indicated above, your read-across hypothesis is based on the (bio)transformation of the Substance and of the source substance(s) to a common compound(s). In this context, information characterising the rate and extent of (bio)transformation of the Substance and of the source substance(s) is necessary to confirm the formation of the proposed common (bio)transformation product and to assess the impact of the exposure to the parent compounds.

As further specified in the original decision, in order for the read-across approach to be acceptable, it is essential to provide the following evidence:

> "the rapid hydrolysis of the registered substance by esterases should be provided, which would confirm your claim that repeated oral exposure to the registered substance would lead to such a low systemic exposure that it is unlikely that the parent substance impacts the [pre-natal developmental toxicity/reproductive toxicity, in particular fertility and

[6] Guidance on information requirements and chemical safety assessment Chapter R.6: QSARs and grouping of Chemicals, Section R.6.2.2.1.f
[7] Guidance on information requirements and chemical safety assessment, Chapter R.6, Section R.6.2.5.2

perinatal developmental effects], i.e. that this toxicity is mediated only via the common metabolite glycolic acid"

You have provided an *in vitro* enzymatic hydrolysis study ([redacted] 2019) investigating the metabolism of the Substance in three matrices: rat liver S9 homogenate, rat small intestinal mucosa homogenate and rat caecum content.

The results of the *in vitro* study show that "*Test item was converted on average 97% in rat liver S9 and 22% in rat small intestinal mucosa > 120±1 min of incubation and highly stable in rat caecum content incubations. In vitro t1/2 values: 24 min liver and >120 intestinal mucosa and caecum content.*" and "*In liver S9 homogenate incubations, the butyl glycolate was completely metabolized after 180 minutes of incubation whereas in small intestinal mucosa homogenate incubations the conversion was approximately 20% after 180 minutes of incubation.*"

The toxicokinetics section of your dossier (expert judgment, [redacted] 2010) concludes that the systemic bioavailability of the Substance after oral uptake "*is assumed as 100% with rapid absorption*".

ECHA considers that even though the Substance is eventually (>120 min) converted to the major metabolite glycolic acid and the minor metabolites glyoxylic acid and n-butyric acid in the rat liver S9 homogenate, a half-life of 24 minutes (liver) indicates that systemic exposure to the unchanged parent compound cannot be excluded. Furthermore, a conversion rate of approximately 20% in the rat intestinal mucosa, and no conversion in the caecum, further indicate potential for the systemic exposure to the unchanged parent compound. Therefore, it is not demonstrated that "*repeated oral exposure to the registered substance would lead to such a low systemic exposure that is it unlikely that the parent substance impacts the pre-natal developmental toxicity, i.e. that this toxicity is mediated only via the common metabolite glycolic acid*". Therefore, the provided information does not support the rationale for the read-across and you have not demonstrated that the properties of the Substance can be predicted from the properties of the source substance.

You submitted comments as the only addressee of the decision. In your comments you confirm that it was indeed your intention to apply read-across approach even though the information in the dossier refers to weight of evidence. You emphasise that, as also acknowledged in the decision, your read-across approach considers the Substance as target substance, and ethylene glycol as well as glycollic acid are the source substances.

You further consider that you have fulfilled the information requirements via read-across. You disagree with ECHA's summary that a lack of reliable information does not allow drawing conclusions on prenatal developmental toxicity and reproductive toxicity (functional fertility). You consider that "*Compared to the original decision, ECHA introduced a different interpretation of the data necessary to strengthen the read across approach and changed the initial information requirements.*" You further clarify that in your opinion, "*The requirement has thus been changed from 'demonstrate no risk' to 'demonstrate no exposure*'."

As explained above, ECHA noted in the original decision that in order for the read-across approach to be acceptable, evidence of rapid hydrolysis of the registered substance should be provided, which would confirm that repeated oral exposure to the Substance would lead to such a low systemic exposure to the parent substance that it is unlikely that the parent substance impacts pre-natal developmental toxicity/reproductive toxicity, i.e. that this toxicity is mediated only via the common metabolite glycolic acid (emphasis added). ECHA did not require you to 'demonstrate no risk'.

As you claim that "*The expected toxicity of butyl glycollate from systemic exposure is primarily based on the effects of glycollic acid*", i.e. the toxicity is mediated only via the metabolite glycolic acid, it would need to be demonstrated that the hydrolysis of the parent substance is rapid and complete, i.e. systemic exposure to the parent substance, following repeated oral exposure, is negligible and does not contribute to toxicity. ECHA agrees that glycollic acid is the main metabolite of butyl glycollate. However, the *in vitro* hydrolysis study showing a half-life of 24 minutes, and complete hydrolysis in >120 minutes, indicates systemic exposure to the parent substance butyl glycollate, and therefore it does not support your read-across hypothesis that the toxicity would be mediated only via the metabolite glycolic acid. The dossier does not contain any *in vivo* toxicokinetics studies on the Substance. Therefore, you have not provided *in vitro* or *in vivo* information which would demonstrate that repeated oral exposure to the Substance would lead to low systemic exposure of the parent substance.

In your comments you explain that in absence of *in vivo* toxicokinetics data, 100% absorption is typically used as a default assumption. You explain that "*The absorbed registered substance is then transported to the liver where metabolism occurs.*" You further consider that "*in vitro metabolism is likely to underestimate the rate of metabolism in an intact animal system*".

ECHA acknowledges your explanation that the metabolism of the Substance is activated in the liver rather than in the intestines. However, the 'likely underestimation' of *in vivo* metabolism is speculative and does not contribute to evidence on rapid hydrolysis following repeated oral exposure.

In your comments you also list the available *in vivo* data for the Substance, namely the acute toxicity study, 28-day and 90-day repeated dose studies as well as a developmental toxicity study. All studies were conducted in rats. In the context of this decision, ECHA notes that these studies provide information on prenatal developmental toxicity in one species, and no information on reproductive toxicity (functional fertility). They do not provide information which would support your read-across approach for developmental toxicity on a second species, or reproductive toxicity (functional fertility).

Conclusions on the read-across approach

As explained above, you have not established that relevant properties of the Substance can be predicted from data on the analogue substance. Therefore, your adaptation does not comply with the general rules of adaptation as set out in Annex XI, Section 1.5. and your grouping and read-across approach is rejected.

Appendix 1: Reasons

1. Pre-natal developmental toxicity study (Annex X, Section 8.7.2.) in a second species

Pre-natal developmental toxicity (PNDT) studies (OECD TG 414) in two species is a standard information requirement under Annex X to REACH.

In the original decision you were requested to submit information derived with the Substance for pre-natal developmental toxicity in a second species (rabbit), via oral route.

In the updated registration dossier subject to follow-up evaluation, you have adapted the standard information requirement mentioned above according to Annex XI, Section 1.2. of REACH (weight of evidence). Within the weight of evidence, you applied a read-across approach based on a common breakdown product.

In support of your adaptation, you have provided the following sources of information:

1. Non-guideline developmental toxicity study, rat ([redacted] 1999). One dose level of ethylene glycol (the source substance), glycolic acid (the common metabolite) and sodium glycolate (not identified as a source substance or a metabolite of the Substance in the read-across justification document).
2. Non-guideline developmental toxicity study, rabbit, with ethylene glycol ([redacted] 1993)
3. OECD TG 414 study, rat and mouse, with ethylene glycol ([redacted] 1995)

Based on the presented sources of information, you argue that the available data gives sufficient information to conclude on the prenatal developmental toxicity for the Substance. In IUCLID section 7.8, you state that *"the extensive amount of data available on ethylene glycol and glycollic acid in addition to butyl glycollate data allows the conclusion that there is no reason to expect that conducting a pre-natal developmental toxicity study (OECD 414) in rabbits [...] with butyl glycollate will reveal new information or additional adverse developmental and/or reproductive effects in animals exposed to butyl glycollate"*.

Furthermore, you have attached documentation in IUCLID section 13[8]. This documentation includes evaluation and weighing of available information on the analogue substance ethylene glycol and concludes that *"there is negligible concern of adverse developmental toxicity from EG at exposures below 125 mg/kg bw."*

In your comments you confirm that sodium glycollate (test material in source study 1) was not identified as a source substance or a metabolite of butyl glycollate in the read-across justification document because sodium glycollate cannot be a metabolite of butyl glycollate or ethylene glycol.

Annex XI, Section 1.2 states that there may be sufficient weight of evidence weight of evidence from several independent sources of information leading to assumption/conclusion that a substance has or has not a particular dangerous (hazardous) property, while information from a single source alone is insufficient to support this notion.

[8] [redacted], January 2004

According to ECHA Guidance R.4, a weight of evidence adaptation involves an assessment of the relative values/weights of the different sources of information submitted. The weight given is based on the reliability of the data, consistency of results/data, nature and severity of effects, and relevance and coverage of the information for the given regulatory information requirement. Subsequently, relevance, reliability, coverage, consistency and results of these sources of information must be balanced in order to decide whether they together provide sufficient weight to conclude that the Substance has or has not the (dangerous) property investigated by the required study.

Annex XI, section 1.2 requires that adequate and reliable documentation is provided to describe your weight of evidence approach.

Relevance of the information provided

Relevant information that can be used to support weight of evidence adaptation for information requirement of Section 8.7.2 at Annex X includes similar information that is produced by the OECD TG 414 on a second species (two species taking the first species into account to address the potential species differences). The following aspects are covered: prenatal developmental toxicity in two species, maternal toxicity in two species, and maintenance of pregnancy in two species.

ECHA notes that you have provided information on prenatal developmental toxicity in one species (rat) on the Substance. The sources of information (1-3) provide relevant information on prenatal developmental toxicity, maternal toxicity and maintenance of pregnancy in other species (mouse, rabbit) for the analogue substance ethylene glycol.

Reliability of the information provided

The following deficiencies affect the reliability of the sources of information:

Section (i) 'Assessment of the Grouping of substances and read-across approach under Annex XI, Section 1.5.' above identifies deficiencies of the grouping and read across approach used in your dossier. These findings apply equally to the sources of information relating to analogue substances submitted under your weight of evidence adaptation. As explained above, your adaptation does not comply with the general rules of adaptation as set out in Annex XI, Section 1.5. and your grouping and read-across approach is rejected.

In your comments you refer to the systemic toxicity pattern of butyl glycollate, glycolic acid and ethylene glycol, concluding that '*all three substances cause similar nephrotoxicity (i.e. the same pattern of toxicological activity)*'. ECHA notes that nephrotoxicity cannot alone predict pre-natal developmental toxicity.

You also refer to similar developmental effects: "*Butyl glycollate effects are equivalent to the developmental adverse effects seen when rodents (rats, mice) are exposed to glycollic acid or ethylene glycol.*" and "*Rabbits (the species suggested for OECD 414 in the DD) did not show developmental toxicity when exposed to glycollic acid or ethylene glycol.*"

ECHA notes that similar effects in rodents do not automatically predict similar effects in non-rodents. Furthermore, ECHA notes that your dossier contains a PNDT study in rabbits conducted with ethylene glycol, with a conclusion "*EG resulted non-teratogenic in rabbits.*". The dossier does not contain any information on developmental toxicity of glycollic acid in rabbits. You have not provided any information on developmental toxicity on the Substance in a second species

(rabbits). Hence, there is no evidence supporting prediction based on similar properties for developmental toxicity between the Substance and the source substances in rabbits.

Finally, ECHA notes that you have self-classified the Substance as Repr. 2 "*based on the observed developmental toxicity in rats.*" As your read-across adaptation is rejected, this concern cannot be clarified by a PNDT study in a second species conducted with ethylene glycol.

In the absence of reliable information, no conclusion can be drawn on prenatal developmental toxicity, maternal toxicity and maintenance of pregnancy in a second species, as required by the information requirement.

It is not possible to conclude, based on any source of information alone or considered together, whether your Substance has or has not the particular dangerous properties foreseen to be investigated in an OECD TG 414. Therefore, your adaptation is rejected.

Based on the above, the information you provided does not fulfil the information requirement, and you are still required to provide information on the prenatal developmental study in rabbits, oral route (Annex X, Section 8.7.2); test method: EU B.31/OECD TG 414 with the Substance.

2. Extended one-generation reproductive toxicity study (Annex X, Section 8.7.3.)

The basic test design of an Extended one-generation reproductive toxicity (EOGRT) study (OECD TG 443) is a standard information requirement under Annex X to REACH. Furthermore Column 2 of Section 8.7.3. defines when the study design needs to be expanded.

In the original decision you were requested to submit information derived with the Substance for an extended one-generation reproductive toxicity study, in rats, via oral route.

In the updated registration dossier subject to follow-up evaluation, you have adapted the standard information requirement mentioned above according to Annex XI, Section 1.2. of REACH (weight of evidence). Within the weight of evidence, you applied a read-across approach based on a common breakdown product.

In support of your adaptation, you have provided the following sources of information, all conducted with the analogue substance ethylene glycol:

4. Three-generation study, rat ([redacted] 1986)
5. Continuous breeding study, mouse ([redacted] 1985)
6. Continuous breeding study, mouse ([redacted] 1986)

Based on the presented sources of information, you argue that the available data gives sufficient information to conclude on the reproductive toxicity (fertility) for the Substance. In IUCLID section 7.8, you state that "*the extensive amount of data available on ethylene glycol and glycollic acid in addition to butyl glycollate data allows the conclusion that there is no reason to expect that conducting [...] an extended one year generation reproductive study (OECD 443) in rats with butyl glycollate will reveal new information or additional adverse developmental and/or reproductive effects in animals exposed to butyl glycollate*".

Furthermore, you have attached documentation in IUCLID section 13[9]. This documentation includes evaluation and weighing of available information on the analogue substance ethylene glycol and concludes that "*there is negligible concern of adverse reproductive toxicity from EG*".

Annex XI, Section 1.2 states that there may be sufficient weight of evidence weight of evidence from several independent sources of information leading to assumption/conclusion that a substance has or has not a particular dangerous (hazardous) property, while information from a single source alone is insufficient to support this notion.

According to ECHA Guidance R.4, a weight of evidence adaptation involves an assessment of the relative values/weights of the different sources of information submitted. The weight given is based on the reliability of the data, consistency of results/data, nature and severity of effects, and relevance and coverage of the information for the given regulatory information requirement. Subsequently, relevance, reliability, coverage, consistency and results of these sources of information must be balanced in order to decide whether they together provide sufficient weight to conclude that the Substance has or has not the (dangerous) property investigated by the required study.

Annex XI, section 1.2 requires that adequate and reliable documentation is provided to describe your weight of evidence approach.

Relevance of the information provided

Relevant information that can be used to support weight of evidence adaptation for information requirement of Section 8.7.3 at Annex X includes information similar to what is produced by the OECD TG 443 design as specified in this decision. This includes information on sexual function and fertility, toxicity to offspring and systemic toxicity.

ECHA notes that you have not provided any information on reproductive toxicity (fertility), i.e. sexual function and fertility, toxicity to offspring and systemic toxicity, on the Substance. The sources of information (4-6) provide relevant information on sexual function and fertility, toxicity to offspring and systemic toxicity for the analogue substance ethylene glycol.

Reliability of the information provided

The following deficiencies affect the reliability of the sources of information:

Section (i) 'Assessment of the Grouping of substances and read-across approach under Annex XI, Section 1.5.' above identifies deficiencies of the grouping and read across approach used in your dossier. These findings apply equally to the sources of information relating to analogue substances submitted under your weight of evidence adaptation. As explained above, your adaptation does not comply with the general rules of adaptation as set out in Annex XI, Section 1.5. and your grouping and read-across approach is rejected.

In your comments you refer to the systemic toxicity pattern of butyl glycollate, glycolic acid and ethylene glycol, concluding that '*all three substances cause similar nephrotoxicity (i.e. the same pattern of toxicological activity)*' ECHA notes that nephrotoxicity cannot alone predict reproductive toxicity (functional fertility).

[9] [redacted] January 2004

In your comments you further state that "[redacted] *(2004) review on reproductive and developmental toxicity of ethylene glycol did not find lesions in reproductive tissues in rodents*". Furthermore, referring to glycollic acid and ethylene glycol, you state that "*these two substances exhibit a lack of reproductive toxicity in rats.*" ECHA emphasises that you have not provided any information on reproductive toxicity (functional fertility) on the Substance. Hence, there is no evidence supporting prediction based on similar properties for reproductive toxicity (functional fertility) between the Substance and the source substances.

In the absence of reliable information on reproductive toxicity (fertility), no conclusion can be drawn on sexual function and fertility or toxicity to offspring as required by the information requirement.

It is not possible to conclude, based on any source of information alone or considered together, whether your Substance has or has not the particular dangerous properties foreseen to be investigated in an 443 study with a design described in this decision. Therefore, your adaptation is rejected.

Based on the above, the information you provided does not fulfil the information requirement, and you are still required to provide information on Extended one-generation reproductive toxicity study in rats, oral route (Annex X, Section 8.7.3.), test method: OECD TG 443, with the Substance.

Appendix 2: Procedural history

In accordance with Article 42(1) of the REACH Regulation, the Agency examined the information submitted by you in consequence of decision CCH-D-2114365547-39-01/F. The Agency considered that this information did not meet one or more of the requests contained in that decision. Therefore, a new decision-making process was initiated under Article 40 of the REACH Regulation.

For the purpose of the decision-making, this decision does not take into account any updates of your registration after the date when the draft of this decision was notified to the Member States Competent Authorities according to Article 51(1) of the REACH Regulation.

The decision making followed the procedure of Articles 50 and 51 of the REACH Regulation, as described below:

ECHA notified you of the draft decision and invited you to provide comments.

ECHA took into account your comments and did not amend the decision.

ECHA notified the draft decision to the competent authorities of the Member States for proposals for amendment.

As no amendments were proposed, ECHA adopted the decision under Article 51(3) of REACH.

Appendix 3: Further information, observations and technical guidance

1. This decision does not prevent ECHA from initiating further compliance checks on the present registration at a later stage.

2. The Article 42(2) notification for the original decision is on hold until all information requested in the original decision has been received.