United States – Continued Suspension of Obligations in the EC – Hormones Dispute

(WT/DS320)

Rebuttal Submission of the United States of America

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I. INTRODUCTION

1. The United States maintains the measures at issue in this dispute in accordance with express authority from the Dispute Settlement Body ("DSB"). At this point in this dispute, it is clear that the European Communities ("EC") has not, and cannot, demonstrate that these measures breach U.S. obligations under the *Understanding on Rules and Procedures Governing the Settlement of Disputes* ("DSU") or the *General Agreement on Tariffs and Trade 1994* ("GATT 1994"), nor has nor can the EC demonstrate that other so-called "measures" that it challenges in fact existed as of the time this Panel was established.

2. As the United States has already pointed out, the EC's arguments relating to its DSU claims underscore its inability to meet its burden in this dispute, that is, to demonstrate that it has satisfied the Article 22.8 condition of removing the WTO-inconsistencies of its measures or providing a solution to U.S. nullification or impairment. Moreover, for the reasons already set forth in previous submissions and discussed further below, the EC's arguments that various DSU provisions can create obligations "in conjunction with" each other cannot change the fact that whether a provision is read on its own or "in conjunction with" another provision does not alter the substance of the provision. Nor can unilateral declarations by a Member concerned create a "presumption of good faith."

3. The United States has not breached Article 23 of the DSU. The United States was authorized to suspend concessions by the DSB, and the EC's declaration of compliance did not cause this authorization to lapse, to be revoked or to be suspended. The EC's declaration did not mean that the United States could no longer apply the suspension of concessions without breaching its obligations under the DSU. Nor did the EC's declaration and development of a "new" measure create a scenario whereby U.S. application of the suspension of concessions could be considered a "determination" as to the WTO-consistency or inconsistency of the amended ban.

4. Regarding the EC's purported demonstration of how it has come into compliance, the EC merely asserted in its first submission that it had come into compliance. Notwithstanding the EC's failure to even undertake the required demonstration, the United States responded in its first written submission by explaining in detail that the EC's import bans, despite DSB recommendations and rulings, continue not to be based on a risk assessment within the meaning of Article 5.1 of the *Agreement on the Application of Sanitary and Phytosanitary Measures* ("SPS Agreement"). Neither are they legitimate "provisional" bans within the meaning of Article 5.7 of the SPS Agreement. The materials put forward by the EC in its replies to questions from the Panel do little to change these conclusions. In fact, in several of its replies, the EC appears to have completely shifted its focus from theoretical risks posed by the six hormones themselves to a perceived "risk" of failure to satisfy good veterinary practices in administering the hormones to cattle in the United States. This submission touches briefly on the EC's claims of U.S. breach of the DSU, then turns to these materials and arguments and illustrates how they fail to demonstrate EC compliance with its WTO obligations.

II. LEGAL ARGUMENTS

A. The EC has failed to demonstrate a U.S. breach of DSU Articles 21.5, 22.8 or 23

1. Article 21.5

5. The EC has failed to demonstrate that the United States has breached its obligations under DSU Article 21.5. In fact, the EC has failed to link U.S. action or inaction to any obligation contained in Article 21.5's text whatsoever. Instead, it claims that the United States has breached Article 21.5 by acting in contravention of DSU Article 23. The United States has not acted in breach of Article 23, and therefore, even under the EC's theory, the United States can not have breached any obligations under Article 21.5 of the DSU.

6. Rather than pointing the Panel to a particular obligation in DSU Article 21.5 that it alleges the United States to have breached, the EC instead persists in its argument that a violation of Article 21.5 can only be found "in conjunction with" or when that Article is "read together" with DSU Article 23.[1] In support of this claim, the EC notes that "there is nothing unusual to cite various provisions to substantiate a claim. This follows actually the same approach the Panel took in the dispute *US - Certain Measures*."[2]

7. The EC is simply wrong about the approach that the panel took, however. When the Appellate Body examined the findings to which the EC is referring, the Appellate Body pointed out that "[o]ur reading of the Panel Report does not lead us to conclude that the Panel based its finding of the inconsistency of the 3 March Measure with Article 21.5 on its conclusion that the measure was inconsistent with Article 23.2(a). . .. The Panel's *references to Article 23.2(a) cannot be construed as the basis upon which the Panel reached its conclusions under Article 21.5*."[3]

8. Moreover, the approach that the EC proposes (and that the EC has wrongly ascribed to the *Certain Products* panel) would be inconsistent with the customary rules of treaty interpretation that this Panel must apply. In particular, so-called "read together" or "in conjunction" claims may not be employed to develop new obligations not actually found in the text of a particular provision of the DSU or the other covered agreements.

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[1] *See* Replies to Questions from the United States after the First Substantive Meeting by European Communities, paras. 4 and 8 (in which the EC fails to identify a claim of breach of obligations set out in Article 21.5 *per se*, and reiterates that its claims of U.S. breach of DSU Articles 21.5 and 22.8 are to be "read together" with DSU Article 23). U.S. arguments regarding the EC's "systemic" interpretive approach in this dispute extend to EC claims of U.S. breach of Article 22.8 "in conjunction with" Article 23.

[2] *See* Replies to Questions from the United States after the First Substantive Meeting by European Communities, paras. 5 and 7.

[3] Appellate Body Report, *United States – Import Measures on Certain Products from the European Communities*, adopted 10 January 2001, WT/DS165/R, para. 126 (*"Certain Products*") (emphasis added).

9. For example, in *India Mailbox*, the Appellate Body noted:

> [t]he legitimate expectations of the parties to a treaty are reflected in the language of the treaty itself. The duty of a treaty interpreter is to examine the words of the treaty to determine the intentions of the parties. This should be done in accordance with the principles of treaty interpretation set out in Article 31 of the Vienna Convention. But these principles of interpretation *neither require nor condone the imputation into a treaty of words that are not there or the importation into a treaty of concepts that were not intended*.[4]

The Appellate Body also referred to DSU Article 3.2, which provides that "[r]ecommendations and rulings of the DSB cannot add to or diminish the rights and obligations provided in the covered agreements," as well as DSU Article 19.2, which provides that, "[i]n accordance with paragraph 2 of Article 3, in their findings and recommendations, the panel and Appellate Body cannot add to or diminish the rights and obligations provided in the covered agreements."[5]

10. Similarly, in *United States – Import Prohibition of Certain Shrimp and Shrimp Products* ("*Shrimp Turtle*"), the Appellate Body stated, "*[a] treaty interpreter must begin with, and focus upon, the text of the particular provision to be interpreted*."[6] Contrary to this approach, the EC would have the Panel, in its Article 21.5 analysis, forego the actual text of the provision, by seeking a finding of a U.S. breach of Article 21.5 based on obligations set out in other provisions of the DSU. By arguing that "in conjunction" with DSU Article 23, new, non-textual obligations may now be imputed into the text of DSU Article 21.5, the EC attempts to achieve a result inconsistent with the customary rules of treaty interpretation and inconsistent with the guidance of the Appellate Body on the application of those rules.

11. As the United States has demonstrated, nowhere in DSU Article 21.5 is there an obligation for the United States to have sought recourse to an Article 21.5 compliance panel, and only such a panel, upon hearing the EC's declaration of compliance. Nor does Article 21.5 contain any time limitation or deadline by which a Member must initiate dispute settlement proceedings. Indeed, the EC does not claim that such obligations can be found in the text of Article 21.5.[7] Yet, it seeks a specific finding of a U.S. breach of Article 21.5 just the same.[8]

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[4] Appellate Body Report, *India – Patent Protection for Pharmaceutical and Agricultural Chemical Products*, adopted 16 January 1998, WT/DS50/AB/R, para. 45 ("*India Mailbox*") (emphasis added).

[5] *See* Appellate Body Report, *India Mailbox*, para. 47, *citing* DSU Articles 3.2 and 19.2.

[6] Appellate Body Report on *United States – Import Prohibition of Certain Shrimp and Shrimp Products*, adopted 6 November 1998, WT/DS58/AB/R, para. 114 ("*Shrimp Turtle*") (emphasis added).

[7] Indeed, the EC itself acknowledges that Article 21.5 does not specify which Member is tasked with initiating a compliance review. *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 20 ("Article 21.5 of the DSU does not itself mention who is to initiate the compliance review.").

[8] *See*, *e.g.*, Replies to Questions from the United States after the First Substantive Meeting by European Communities, para. 4.

Taking into account the Appellate Body's guidance in *India Mailbox* and *Shrimp Turtle*, the EC's theory in this dispute (*i.e.*, a violation of Article 21.5 read "in conjunction with" Article 23) has no textual basis and must therefore be rejected. Any analysis of whether U.S. actions have breached DSU Article 21.5 must be based on the text of that provision.[9]

12. In addition to its attempt to impute obligations into the text of Article 21.5, the EC provides other non-textual arguments in support of its claim of a U.S. Article 21.5 breach. Primary among these is the EC's reference to a presumption or principle of good faith. The EC considers that referring to such a presumption justifies imputing into Article 21.5 an unspecified and unwritten obligation that "[a] retaliating Member has at a minimum a *good faith obligation* to assess within a reasonable delay the compliance measure".[10] As noted above, the key to interpretation of the DSU, and Members' obligations under its provisions, lies in the actual text of the provisions. The text of Article 21.5 does not contain a time limitation, let alone what would amount to a case-by-case-determined "reasonable time period".[11] Neither does it contain an obligation that, in the post-suspension setting, the suspending Member initiate dispute settlement proceedings upon a declaration of compliance by the Member concerned. "Good faith" applies to implementing the obligations that <u>are</u> agreed upon by Members, evidenced in the text; "good faith" cannot serve to create <u>new</u> obligations that were never agreed by Members.

2. <u>Article 22.8</u>

13. Similarly, the EC has failed to make a *prima facie* case of a U.S. violation of Article 22.8 of the DSU. Rather than presenting any evidence of how it has satisfied the conditions of Article 22.8 (removal of WTO-inconsistent measure; provision of solution to nullification or impairment of benefits; mutually satisfactory solution), it posits its claim "in conjunction with" Article 23[12] and asserts that the "presumption of good faith" satisfies its burden of proof.[13] This argument not only highlights the EC's attempt to employ claims of good faith and bad faith in whichever manner serves it best at any given moment, it is also not consistent with Appellate Body findings on how burdens of proof function in dispute settlement. As we have explained in detail in our

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[9] The EC appears to accept this point, given that it says that it is a panel's task to "apply the existing procedural rules of the DSU to [a] case." *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 205.

[10] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 66.

[11] *See* paras. 17-18 below.

[12] *See* Replies to Questions from the United States after the First Substantive Meeting by European Communities, para. 8 ("the European Communities considers that the continued application of sanctions despite the unchallenged EC' compliance measure is in violation of Articles 23.1 and 22.8 read together.") *See* paras. 5 *et seq*. above.

[13] *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 74. ("It is true that within the Article 22.8, 23 claim the European Communities relies on the presumption of good faith.")

previous submissions to the Panel[14], there is no presumption of compliance or good faith in WTO dispute settlement that attaches to measures taken by WTO Members. Furthermore, even were one to presume that the EC implemented its amended bans in good faith, this fact would not in turn demonstrate that the EC's bans actually satisfy the elements of Article 22.8, *e.g.*, the EC could be acting in good faith, but still be wrong about the WTO-consistency or compliance of its amended measure.

14. Rather than a presumption of good faith, there is instead no presumption of bad faith that attaches to a WTO Member's measures in dispute settlement.[15] This is unexceptional, as it simply emphasizes the rules on burden of proof in WTO dispute settlement, *i.e.*, that a complaining Member may not simply allege the WTO-inconsistency of another Member's measure and prevail. Complaining Members are obligated to make a *prima facie* case of a breach, and not simply allege or assert a breach in order to place or shift the burden to the responding Member. Yet in this proceeding the EC, as the complaining party, argues that a presumption of good faith actually establishes its *prima facie* case against a responding party, thereby automatically shifting the burden of proof to the United States. This interpretation cannot be reconciled with the very Appellate Body findings cited by the EC in its first written submission.[16]

3. Article 23

15. As to the "heart"[17] of the EC's claims, DSU Article 23, there is little to add at this stage of the proceedings other than to touch upon an argument made by the EC in its replies to the Panel's questions and to make one comment regarding the EC's interpretation of Article 23's text. In its replies to the Panel's questions, the EC argues that the *Certain Products* panel "found that suspension of concessions (in order to seek redress of a violation) necessarily imputes a determination."[18] However, the panel finding cited by the EC was based on the lack of any DSB recommendations and rulings and so is inapt to the situation at hand. As noted by the *Certain Products* panel in the paragraph in question:

> When on 3 March the United States decided to take a remedial action against EC listed imports, there was no WTO determination (no findings by a panel, Appellate Body or arbitration body) concluding that the EC implementing measure was incompatible with the WTO Agreement. The unilateral action (as

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[14] *See* Responses of the United States to Questions from the Panel, paras. 47-54.

[15] *See also* Appellate Body Report, *Chile - Taxes on Alcoholic Beverages*, adopted 12 January 2000, WT/DS87/AB/R, WT/DS110/AB/R, para. 74 ("*Chile – Alcohol*"), cited in EC First Written Submission, para. 89.

[16] *See*, *e.g.*, EC First Written Submission, paras. 88-93.

[17] *See* Oral Statement by the European Communities in the First Substantive Meeting, para. 26 ("Our objective this morning is to refocus these proceedings on the provision of Article 23, which is at the heart of this case.")

[18] *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, at fn. 49.

> we concluded above . . .) taken by the United States implies necessarily a prior US unilateral determination that the EC implementing measure was inconsistent with the WTO.

16. The facts before the panel in the *Certain Products* dispute are markedly different from those in this proceeding. In *Certain Products*, the panel found that the United States acted (by suspending concessions) without having yet been authorized to do so by the DSB. Also, as opposed to these proceedings, the United States was not simply continuing to apply authorized suspension of concessions; the panel found that it had taken new action specifically in response to the EC's measure, dated after that measure. Through its analogy of the facts at issue in this dispute with those in the *Certain Products* dispute, the EC appears to insinuate that its declaration of compliance has cleaned the slate in the *Hormones* dispute[19] entirely, placing the parties back in the position that they found themselves in *Certain Products* – with no DSB authorization, no WTO determination and a U.S. measure targeted specifically at the EC's "amended" measure. This is not a colorable comparison and must be rejected.

17. Finally, as a procedural matter, the United States notes that the EC's interpretation of Article 23, and specifically Article 23.2(a), is complicated by a lack of clarity regarding when, exactly, a determination on the part of the suspending Member would be inferred. The EC has commented several times on a U.S. argument that, pursuant to the EC's interpretation of Article 23, a determination could be inferred "immediately" upon the declaration of the Member concerned that its measure conforms with DSB recommendations and rulings. Instead of "immediately", the EC argues that there is a "reasonable period" during which the suspending Member may review the measure before a determination is either inferred or due.[20] However, no such "reasonable period" time frame or obligation is set out in the text of Article 23. Rather, it is yet another construct of the EC's – an example of how the EC believes the DSU should be rewritten, by reading Article 23 "in conjunction" with Article 21.5.

18. Regardless of whether this fictional deadline is a "reasonable period" or immediate, the EC's interpretation establishing such a deadline is not sustainable. Not only would it beg litigation to determine on a case-by-case basis whether a Member has unreasonably delayed in making a determination, it would convert Article 23.2(a) from a prohibition on making determinations into an obligation to make them – ironically, a Member would in effect be required to make a determination upon learning of an Member concerned's declaration of compliance, and to do so within some unspecified time frame.[21]

19. Furthermore, as the United States has noted on several occasions, the United States was in the course of reviewing the EC's materials at the outset of this dispute. In light of this fact, it

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[19] *EC – Measures Concerning Meat and Meat Products (Hormones)*, Appellate Body Report and Panel Report adopted on 13 February 1998, WT/DS26/AB/R and WT/DS26/R ("*Hormones*" or "*EC – Hormones*").

[20] *See*, *e.g.*, Oral Statement by the European Communities in the First Substantive Meeting, para. 63.

[21] *See* Responses of the United States to Questions from the Panel, para. 10.

is difficult to comprehend how the United States could have made a "determination" as to the WTO-consistency of the EC's amended bans. A critical element of this U.S. evaluation is the review of the studies and Opinions ostensibly underpinning the EC's bans. Specifically, the EC refers to a number of studies which it commissioned after the Appellate Body proceedings in the *Hormones* dispute, referring to them as the "17 Studies".[22] The EC invokes these studies throughout its 2000 Review and 2002 Opinion, and a review of their methodology and results are therefore critical to an analysis of the EC's measure. However, as noted at the first substantive meeting with the Panel, the United States has not had the opportunity to review all of these documents, and referred to this fact in explaining why it had not yet been able to reach a determination of the EC's Opinions or its bans. Indeed, the EC has only recently informed us of a number of studies – which it contends comprise the basis for its claim of compliance with the DSB's recommendations and rulings – that were not referenced in the EC's response to the U.S. request for information under Article 5.8 of the SPS Agreement, through which the United States sought all relevant scientific information on which the EC premised its bans.[23]

20. In response to the U.S. explanation that it had not had the opportunity to review all of the EC's studies, and a question from the Panel[24] regarding the general availability of the studies, the EC commented that:

> It should be noted that Canada, Australia and the United Kingdom have made a review of these three opinions of the SCVPH and of the results of the 17 studies mentioned above, and issued their own reviews of these studies. The reviews of these three countries are also publicly available (and actually provided to the panel with the parties submissions). This means that these three countries *have had no problem whatsoever to obtain access to all relevant documentation pertaining to the European Communities' risk assessment*.[25]

Despite this assertion, the EC submitted several documents, studies and reviews with its replies to the Panel's recent set of questions which had not been included on previous lists of the "17 Studies", including the list of studies attached to its final "risk assessment", the 2002 Opinion.[26] The United States was not, before the EC's initiation of this dispute, privy to all of the materials

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[22] *See*, *e.g.*, "Opinion of the Scientific Committee on Veterinary Measures Relating to Public Health on Review of previous SCVPH opinions of 30 April 1999 and 3 May 2000 on the potential risks to human health from hormone residues in bovine meat and meat products", 10 April 2002 ("2002 Opinion"), § 1.2, p. 6. (Exhibit US-1).

[23] *See* EC Response to U.S. SPS Article 5.8 Request, Question 3 (requesting that the EC "please identify the risks the restriction addresses, identify the scientific evidence upon which the restriction is based, and provide any scientific studies or reports that support the restriction.") (Exhibit US-23).

[24] *See* Panel Question 16.

[25] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 81. (Emphasis added).

[26] *See* "Comments on the Availability of the EC's 17 Studies", attached as Table 1 to this submission, which lists materials recently put forward (and previously either unavailable or unreferenced) by the EC in support of its bans.

comprising the 17 Studies, let alone the hundreds of additional pages of materials the EC has now submitted in the middle of this proceeding. Indeed, the EC's skeletal response to the U.S. SPS Article 5.8 request for scientific information on the bans simply quoted the titles of the 1999 and 2002 Opinions and 2000 Review.[27] None of these documents cite to the new materials recently submitted by the EC.[28]

21. Furthermore, upon examination of the Australian Review of the EC's Opinions, cited above by the EC as an example of the ready availability of the studies, it is evident that Australia did not in fact obtain copies of all of the 17 Studies.[29] This difficulty in procuring materials was further emphasized by the 2005 U.K. Report.[30] This is not surprising, as the EC, while averring that these studies were readily available, added the following caveat to its answer to the Panel:

> The European Communities provides as an exhibit to this submission the results of all the 17 studies initiated by the European Commission and the numerous publications they have given rise to in various peer reviewed scientific journals. *When a couple of these studies are not published in peer reviewed scientific journals, a copy of the original of the study is provided*.[31]

22. In light of the EC's failure to provide to the United States all the relevant EC studies, it can come as little surprise that the United States was still in the course of reviewing documentation relevant to the EC's measure when the EC requested this Panel. As a consequence of this ongoing review, and the difficulty in obtaining these documents, it is perfectly understandable why the United States had not yet made a "determination" as to the

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[27] *See* EC Response to U.S. SPS Article 5.8 Request, Question 3. (Exhibit US-23).

[28] *See* Table 1 ("Comments on the Availability of the EC's 17 Studies").

[29] *See* "A Review to Update Australia's Position on the Human Safety of Residues of Hormone Growth Promotants (HGPs) Used in Cattle", Department of Health and Ageing, July 2003 ("Australia Review"), p. 43 ("A number of these 17 EC-commissioned studies have not been published to date, and were therefore unable to be evaluated. Furthermore, the original study data was not available from the Commission"), p. 32 ("All of the studies evaluated as part of this review were accessible only as published scientific papers, the main limitation of which was the general lack of reporting detail. Consequently, a truly independent evaluation of the original data was not possible.") (Exhibit US-16).

[30] *See* "Risks Associated with the Use of Hormonal Substances in Food-Producing Animals", Draft Report of the Veterinary Products Committee, May 2005 ("2005 U.K. Report") ("*One particular point to note was the effort required for the VMD Secretariat to obtain the 17 EU-funded reports* used by the SCVPH. The Working Group appreciated the *persistence of the Secretariat that eventually enabled them to be obtained from the Commission*. Full details of the 17 studies are given in Appendix B; individual studies are referred to in this report as studies 1 to 17. *In some cases, only the study report submitted to the EC was available*. In other cases, peer-reviewed publications were available, based on results within some of the study reports. *Some of the study reports have not, as yet, been published in journals and thus have not been subject to the normal peer-review process*.") (Emphasis added). (Exhibit US-20).

[31] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 84.

WTO-consistency of the EC's import bans. The EC bears the consequences for its delayed and piecemeal production of the underpinning materials for its bans.[32]

4. Article 3.7

23. Because the EC has failed to demonstrate a U.S. breach of its obligations under the DSU, there can be no "in conjunction with" breach of the objectives set out in DSU Article 3.7, even were such a claim possible.[33]

5. GATT 1994 Articles I and II

24. Because the United States has not breached its obligations under the DSU and continues to suspend concessions pursuant to DSB authorization, there can be no U.S. breach of GATT 1994 Articles I or II. Any finding of a breach of these provisions would be premised on a finding that the United States did not have authorization to suspend concessions to the EC.[34]

B. The EC has neither removed its WTO-inconsistent bans nor provided a solution to U.S. nullification or impairment within the meaning of DSU Article 22.8

1. Introduction

25. A determination of whether or not the EC has complied with the DSB's recommendations and rulings in the *Hormones* dispute is central to an analysis of whether or not it has satisfied the conditions of Article 22.8 by either removing its WTO-inconsistent measure or providing a solution to U.S. nullification or impairment. As we have demonstrated, a *prima facie* case of a U.S. breach of Article 22.8 would have to include a demonstration of how the EC has satisfied those recommendations and rulings, thereby either removing its measure or providing a solution within the meaning of Article 22.8. The EC has failed to make its *prima facie* case of a U.S. breach of DSU Article 22.8 because it has not demonstrated, other than by simple assertions that it deems its own measure to satisfy DSB recommendations and rulings, how its import bans are now WTO-consistent.

26. In the following portion of our submission, the United States analyzes further the EC's claim of a "direct" U.S. violation of DSU Article 22.8. This analysis highlights that the EC's bans on meat and meat products from cattle treated with testosterone, progesterone, trenbolone acetate, zeranol and melengestrol acetate ("MGA") do not qualify as "provisional" bans within the meaning of SPS Article 5.7 and that the EC has failed to base its import ban on meat and

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[32] *See* Table 1 ("Comments on the Availability of the EC's 17 Studies").

[33] *See*, *e.g.*, Appellate Body Report, *US – Certain Products*, para. 120; *see also* U.S. First Written Submission, paras. 206-207.

[34] *See* U.S. First Written Submission, paras. 208-209.

meat products from cattle treated with estradiol 17β on a risk assessment within the meaning of SPS Article 5.1. The EC has thus failed to demonstrate that its amended import bans remove the WTO-inconsistent measure or provide a solution to U.S. nullification or impairment for purposes of DSU Article 22.8.

2. The EC has failed to demonstrate that its import ban on meat from cattle treated with testosterone, progesterone, trenbolone acetate, zeranol and MGA for growth promotion purposes according to good veterinary practices is a provisional measure within the meaning of SPS Article 5.7

27. Despite several opportunities to present evidence as to why its ban on five of the hormones (testosterone, progesterone, trenbolone acetate, zeranol and MGA) is a legitimate provisional measure, the EC fails to demonstrate how its ban on meat and meat products from cattle treated with these five hormones in fact satisfies the criteria of Article 5.7 of the SPS Agreement.[35] Because the EC's ban fails to meet the requirements of Article 5.7 (measure maintained in a situation where "relevant scientific evidence is insufficient;" measure adopted "on the basis of available pertinent information"; additional information for a more objective assessment of risks sought; measure reviewed within a reasonable period of time)[36], the EC is therefore not exempt from satisfying its obligations under Article 2.2 (measures not to be maintained without sufficient scientific evidence) and Article 5.1 (measures to be based on a risk assessment) of the SPS Agreement.

a. The EC fails to demonstrate that its ban on the five hormones is maintained in a situation where "relevant scientific evidence is insufficient"

28. The EC's ban on meat from cattle treated with the five hormones is not maintained in a situation where relevant scientific information regarding those hormones is insufficient. The Appellate Body in *Japan – Apples* noted that, in determining whether or not scientific evidence is insufficient, "the question is not whether there is sufficient evidence of a general nature or whether there is sufficient evidence related to a specific aspect of a [] problem, or a specific risk. The question is whether the relevant evidence, be it 'general' or 'specific' . . . is sufficient to permit [a risk assessment]."[37] In the case of the five hormones "provisionally" banned for use as

[35] Article 5.7 is a qualified exemption from Article 2.2 of the SPS Agreement, which stipulates, *inter alia*, that Members shall not maintain sanitary measures without sufficient scientific evidence "except as provided for in paragraph 7 of Article 5." *See* Appellate Body Report, *Japan – Measures Affecting the Importation of Apples*, adopted 10 December 2003 WT/DS245/AB/R, para. 170 ("*Japan – Apples*").

[36] *See* Appellate Body Report, *Japan – Apples*, para. 176, citing Appellate Body Report, *Japan – Measures Affecting Agricultural Products*, adopted on 19 March 1999, WT/DS76/AB/R, para. 89 ("*Japan – Varietals*").

[37] Appellate Body Report, *Japan – Apples*, para. 179.

growth promoters, there is more than sufficient scientific evidence to allow "performance of an adequate assessment of risks as required under Article 5.1."

29. The simple fact regarding the five hormones at issue is that international standards and a significant body of scientific studies exist on the risks posed by each hormone.[38] The Joint FAO/WHO Expert Committee on Food Additives ("JECFA") and several national regulatory bodies have determined that the scientific evidence regarding these hormones is adequate or sufficient to conduct a risk assessment. The EC alone alleges that this body of information is not "sufficient" to conduct a risk assessment, as required by Article 5.1 of the SPS Agreement, and the EC has only taken this position after firmly stating to the WTO several times that the information is sufficient and only after the WTO finding that the EC had breached its SPS obligations.[39] In so doing, however, the EC does little more than assert that this is the case,[40] failing to cite to any scientific evidence demonstrating risks to consumers from the five hormones when used for growth promotion purposes in meat according to good veterinary practices. Indeed, our review of the available materials comprising the 17 Studies has failed to uncover any new evidence of risk from the five provisionally banned hormones, further casting doubt on the EC's conclusion that evidence relating to these hormones is now somehow insufficient.[41]

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[38] *See* "Evaluation of certain veterinary drug residues in food", Fifty-Second Report of the Joint FAO/WHO Expert Committee on Food Additives, WHO Technical Report Series: 893 (2000) ("52nd JECFA Report"), p. 60 (the Committee considered "*numerous reports* of studies on progesterone in humans" as well as an "*extensive database* on the effects of the hormone"), p. 62 ("*[e]xtensive literature exists* on the use of synthetic progestogens in combination with estrogens for oral contraception") (Exhibit US-5); *see also* "Evaluation of certain veterinary drug residues in food", Fifty-Fourth Report of the Joint FAO/WHO Expert Committee on Food Additives, WHO Technical Report Series: 900 (2001) ("54th JECFA Report"), p. 65 ("[t]he Committee considered information from a *range of studies on melengestrol acetate*, including studies on its pharmacokinetics, biotransformation, acute, short-term and long-term toxicity, carcinogenicity, genotoxicity, and reproductive and developmental toxicity. It also considered the *results of studies in humans*"). (Exhibit US-24).

[39] The EC claims that its provisional ban is premised on "the most thorough and recent information obtained in the latest assessments of the risk for each of these five hormones [which] has been found to be insufficient, inconclusive and contradictory." *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 103.

[40] *See*, *e.g.*, Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 104-107 (in which the EC simply quotes "recitals" from its Directive that are nothing more than unsupported conclusions that the EC considers itself to have satisfied the conditions for a provisional ban. There does not appear to be any citation to scientific support for why, *e.g.*, scientific evidence is insufficient to conduct a risk assessment, or how the ban takes into account available pertinent information, as required by SPS Article 5.7).

[41] For example, to assess the genotoxicity of zeranol, trenbolone and MGA, the EC commissioned a study entitled "Hormones as growth promoters: genotoxocity and mutagenicity of zeranol and trenbolone" (study number two of the "17 Studies"). Yet, in an unpublished contract report (Exhibit EC-6(US)), only recently provided to the United States for review, the authors conclude that *none of the three synthetic growth promoters tested demonstrated evidence of genotoxicity* using 4 different endpoints of genetic damage. These results were reiterated in a review article later published by the same author and cited by the EC. *See* Metzler M. and Pfeiffer E., Genotoxic potential of xenobiotic growth promoters and their metabolites, APMIS 109 (2001): 89-95. (Exhibit EC-6(US)).

b. The EC fails to demonstrate that its ban was adopted "on the basis of available pertinent information"

30. As noted in our replies to questions from the Panel, while the Panel need not extend its analysis to a review of the second cumulative requirement for a provisional ban under Article 5.7 because there is sufficient scientific evidence for the EC to conduct a risk assessment for the five hormones, the EC has nonetheless failed to satisfy the second requirement of Article 5.7. Within the context of this dispute, "available pertinent information" refers to both general evidence relating to potential risks posed by the hormones, as well as information addressing the specific risk at issue – that associated the consumption of meat from animals treated with hormones for growth promotion purposes according to good veterinary practice.[42]

31. In the case of the five hormones "provisionally" banned by the EC, there is no such "pertinent information" upon which the EC's import ban can be based because none of the information presented by the EC in its Opinions suggests that meat and meat products from cattle treated with the five hormones for growth promotion purposes according to good veterinary practice pose a risk to consumers. The EC does not consider information pertaining to the specific risk in question (*i.e.*, that to consumers ingesting hormones in meat from cattle treated according to good veterinary practices), including relevant international standards for the five hormones and their underpinning studies, which indicates that hormone residues in such meat are safe.[43] Instead, the EC restricts its consideration to general information or evidence on the hormones – evidence that was considered by Codex and JECFA in determining that the hormones do not pose a risk to consumers.

32. As an example of the EC's failure to take into account available evidence, or "pertinent information", directly related to the expected doses from dietary exposures to hormones, the EC did not make use of relevant bioavailability data, take into account processes such as *in vivo* repair mechanisms, or examine actual regulatory practices in the United States as they pertain to growth promoting hormones.[44] These are examples of information relevant to an analysis of any hypothetical risk from the five hormones when used for growth promotion, and are examples of

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[42] *See* U.S. response to Panel question 70.

[43] As noted in the first sentence of Article 5.7, a Member's analysis of "available pertinent information" includes information "from the relevant international organizations as well as from sanitary or phytosanitary measures applied by other Members."

[44] *See*, *e.g.*, "Evaluation of certain veterinary drug residues in food", Thirty-Second Report of the Joint FAO/WHO Expert Committee on Food Additives, WHO Technical Report Series: 763 (1988) ("32nd JECFA Report"), pp. 23-26 (concluding that trenbolone has no effect on reproduction at low concentrations; trenbolone is not genotoxic; and any effects are consequent to concentrations above hormonal activity level) (Exhibit US-25); 52nd JECFA Report (2000) (concluding that "[t]he Committee noted that exogenous progesterone has been used to maintain pregnancy, with *no evidence of toxicity* and with *no effect on the normal conclusion of pregnancy*") (emphasis added) (Exhibit US-5); 54th JECFA Report (2001), p. 73 ("[m]elengestrol acetate has been *tested for genotoxicity in a range of assays in vitro and in vivo* . . . the Committee concluded that melengestrol acetate is *not genotoxic*") (emphasis added). (Exhibit US-24).

the types of considerations taken into account by numerous other regulatory and standard-setting bodies.[45] Rather than taking into account this information as required by SPS Article 5.7, however, the EC instead relies on hypothetical, unrealistic scenarios (*e.g.*, misuse or failure to satisfy good veterinary practices[46]), for which it fails to provide any scientific evidence, in order to calculate possible exposure estimates.[47] In the absence of evidence that these scenarios pertain to actual conditions, and given the wealth of information on more directly relevant exposure pathways, these scenarios (as well as environmental studies of hormone concentrations in animal effluent also relied upon by the EC[48]) do not constitute available pertinent information on risks associated with the consumption of meat from animals treated with hormones for growth promotion purposes according to good veterinary practice.

c. The EC has not "review[ed] the . . . measure accordingly within a reasonable period of time"

33. The EC has failed to demonstrate that it has reviewed its ban within a reasonable period of time. As noted by the Appellate Body, the "reasonable period of time" is not a fixed period, but rather reflects circumstances on a case-by-case basis.[49] Furthermore, in determining whether

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[45] *See* 52nd JECFA Report (2000), pp. 57-74 (Exhibit US-5); *see also* "Report of the CVMP on the Safety Evaluation of Steroidal Sex Hormones in particular for 17β-Oestradiol, Progesterone, Alternogest, Flugestone acetate and Norgestomet in the Light of New Data/Information made available by the European Commission", Committee for Veterinary Medicinal Products (EMEA/CVMP/885/99) ("1999 CVMP Report"), p. 11 ("General Considerations") (Exhibit US-13).

[46] *See* Section III.B.4 below; *see*, *e.g.*, Lange I. G. *et al.*, Hormone contents in peripheral tissues after correct and off-label use of growth promoting hormones in cattle: Effect of the implant preparations Finaplix-H, Ralgro, Synovex-H and Synovex Plus, APMIS 109 (2001): 53-65 (part of study number five of the "17 Studies"), which similarly concludes that, when used according to approved labeling conditions, residues of testosterone, zeranol, estradiol and trenbolone are less than U.S. tolerances or JECFA MRLs. (Exhibit EC-6(US)). From a methodological standpoint, several of the misuse studies conducted by the EC suffer from small test group sizes (number of cattle) and a consequent lack of statistical analysis. *See* Australia Review, p. 47. (Exhibit US-16).

[47] *See*, *e.g.*, "Opinion of the Scientific Committee on Veterinary Measures Relating to Public Health – Assessment of Potential Risks to Human Health from Hormone Residues in Bovine Meat and Meat Products", 30 April 1999 ("1999 Opinion"), § 3.3 (Exhibit US-4); 2002 Opinion, §§ 4.1.4, 9.1 (Exhibit US-1). For U.S. arguments relating to the EC's failure to satisfy the third prong of Article 5.7's four-element test (failure to "seek to obtain the additional information necessary for a more objective assessment of risks), please *see* U.S. First Written Submission, para. 132.

[48] *See* Schiffer B. *et al.*, The fate of trenbolone acetate and melengestrol acetate after application as growth promoters in cattle: environmental studies. Environmental Health Perspectives 2001; 109: 1145-1151 (study number fourteen of the "17 Studies"), in which, *inter alia*, liquid manure and manure from treated animals were sampled for trenbolone and MGA concentrations. The study's authors concluded that the two hormones should be investigated further for potential endocrine-disrupting activity in ecosystems. This study's results appear to be irrelevant to a human health assessment of trenbolone and MGA and therefore irrelevant in terms of support for the EC's ban on meat and meat products from cattle treated with the five hormones.

[49] *See* Appellate Body Report, *Japan – Varietals*, para. 93. ("The second part of the second sentence of Article 5.7 stipulates that the Member adopting a provisional SPS measure shall 'review the … measure accordingly within a reasonable period of time.' In our view, what constitutes a 'reasonable period of time' has to be established on a case-by-case basis and depends on the specific circumstances of each case, including the difficulty of obtaining

a reasonable time has elapsed, one of the factors that should be taken into account is the "difficulty of obtaining the additional information necessary for the review and the characteristics of the provisional SPS measure."[50] In the case of the five hormones banned by the EC, as noted above, there is already a substantial body of evidence available for completing a risk assessment, contradicting the suggestion that any "additional information" whatsoever might be required to review the amended ban. In addition, the "provisional" ban simply prolongs the original ban, marking over fifteen years that the import ban, the most trade-restrictive measure possible, has been in place. Taking into account the severity of the measure, and the ready availability of information on the five hormones, the EC has not reviewed its measure within a reasonable period of time within the meaning of Article 5.7.

3. The EC has failed to base its import ban on meat from cattle treated with estradiol 17β for growth promotion purposes according to good veterinary practices on a risk assessment within the meaning of SPS Article 5.1

34. The EC has failed to base its import ban on meat from cattle treated with estradiol 17β on a risk assessment within the meaning of SPS Article 5.1. Indeed, the EC's Opinions and underpinning studies fail to demonstrate a risk from residues in meat from cattle treated with estradiol 17β for growth promotion purposes according to good veterinary practices.[51] Instead, the studies on which the Opinions rely only succeed in demonstrating theoretical risks when the hormones are administered at doses or levels well-above those present in residues from hormone-treated meat; when good veterinary practices are not met; or in ways not germane to the relevant risk pathway. As a result, the EC's ban on meat and meat products from cattle treated with estradiol 17β for growth promotion purposes according to good veterinary practices is not "based on", or sufficiently warranted or rationally related to its purported risk assessment within the meaning of SPS Article 5.1. A measure premised on the existence of a risk from residues in such meat cannot rationally relate to a risk assessment that fails to identify scientific evidence of the relevant risk.[52]

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the additional information necessary for the review and the characteristics of the provisional SPS measure.")

[50] *See* Appellate Body Report, *Japan – Varietals*, para. 93.

[51] The United States addresses the EC's new arguments regarding the hypothetical failure to satisfy good veterinary practices at Section III.B.4 below. *See*, *e.g.*, Daxenberger *et al*., Detection of anabolic residues in misplaced implantation sites in cattle, AOAC International 83, No. 4, pp. 809-819 (part of study five of the "17 Studies") (the results of which assume in every instance that there is a failure of good veterinary practices in implanting cattle with hormones for growth promotion purposes). (Exhibit EC-6(US)).

[52] *See* Panel Report, *Japan – Apples (21.5)*, paras. 8.145-8.146 (finding that "[s]ince the scientific evidence relied upon by Japan does not support the conclusions reached by Japan in its 2004 PRA, we conclude that the 2004 PRA is not an assessment, as appropriate to the circumstances, of the risks to plant life or health, within the meaning of Article 5.1 of the SPS Agreement.") *See also* U.S. First Written Submission, at Section IV.C.2.a, describing how the EC's Opinions fail to satisfy the four elements of a risk assessment.

a. The EC's Opinions fail to take into account available scientific evidence relating to genotoxicity and carcinogenicity of estradiol 17β

35. The EC has failed to produce any scientific evidence that estradiol 17β, when used as a growth promoter in cattle according to good veterinary practices, poses a cancer risk to consumers. The EC's assumption that estradiol is genotoxic is essential to its overall conclusions regarding this hormone, as well as to its purported risk assessment, because, as noted in its response to a question from the Panel, "[m]ost importantly, carcinogenic activities of molecules can not be assessed with the reasoning and the biological thresholds available to assess their acute or chronic toxicity, as does the United States. This is *particularly the case when compounds have genotoxic activity*."[53] In other words, the EC reasons that if estradiol 17β is genotoxic, then the otherwise relevant considerations for evaluating its risk, such as by contemplating threshold levels or hormonal effect levels, are no longer adequate.[54]

36. Nevertheless, despite reaching the conclusion that estradiol 17β is genotoxic in its Opinions,[55] the EC does not in fact demonstrate through scientific evidence that this is the case.[56] It fails to provide evidence demonstrating that estradiol has carcinogenic effects other than through the receptor mediated, cell division stimulating activity of the hormone[57] – in other words, at levels exerting a hormonal effect on consumers, and not at the exponentially smaller levels that would be found in meat residues. The fact that effects may be observed at exposure

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[53] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 101.

[54] *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 160 ("[i]t should be clarified first that it is generally recognised that for substances which have genotoxic potential (as is the case with oestradiol 17β) the low dose used in animal growth promotion is not relevant, precisely because the possible genotoxic risks may arise at any dose.")

[55] *See* 2002 Opinion, p. 14 ("In summary, *additional and conclusive data* have now been published in the scientific literature to *demonstrate that 17β-oestradiol is genotoxic*"). (Exhibit US-1).

[56] *See* Panel Report, *Japan – Apples (21.5)*, para. 8.145.

[57] *See*, *e.g.*, 1999 CVMP Report, p. 9 (Exhibit US-13). The EC asserts that U.S. citation to the 1999 CVMP Report and its analysis of estradiol 17ß and progesterone is inappropriate because the CVMP only evaluated the hormones for therapeutic or zootechnical purposes and not for animal growth promotion purposes *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 133. However, we fail to see the distinction drawn by the EC. Use for either purpose can result in residues in beef. Our review of the CVMP document indicates that it was concerned primarily with hazards and risks arising from exogenous exposure of consumers to hormones and the possible need, in light of recent data, to perform new risk assessments for estradiol and progesterone. The CVMP concluded that new risk assessments were not necessary and that certain residue levels of progesterone and estradiol were safe, based on some very basic conclusions on these hormones (*e.g.*, lack of genotoxicity, carcinogenic action only after prolonged exposure at high exposure levels) which contradict fundamental – but unsupported – scientific conclusions set out in the EC's Opinions. It should be emphasized that the basic scientific information considered by the CVMP with respect to the risks of therapeutic and zootechnical use of hormones was the same information used by the SCVPH to assess the risks of hormones used for growth promotion, but the CVMP and SCVPH reached very different conclusions. Thus the CVMP evaluation is indeed relevant to this dispute and an analysis of the EC's Opinions.

levels above the hormonal effect level or threshold is well established,[58] and is one of the reasons that groups such as Codex set acceptable daily intakes ("ADIs") and maximum residue levels ("MRLs") at levels exponentially lower than this threshold.

37. The EC seeks support for its argument that estradiol 17β is genotoxic in a JECFA conclusion that "estradiol 17β has genotoxic potential",[59] yet fails to cite to the rest of the relevant paragraph, in which JECFA notes, "[t]he Committee reviewed studies of the genotoxic potential of estradiol-17β. Estradiol-17β *did not cause gene mutations in vitro*. In some other assays, sporadic but unconfirmed positive results were obtained."[60] Furthermore, the EC's citation to the JECFA safety assessment ignores its ultimate conclusion, *i.e.*, that a maximum residue level for estradiol 17β in meat need not be specified because there is a "wide margin of safety for consumption of residues in food when the drug is administered according to good practice in the use of veterinary drugs."[61] JECFA's conclusion corresponds to that of the EC's own Center for Veterinary Medicinal Products ("CVMP"), which summed up the relevant, and recent, evidence on the genotoxicity of estradiol 17β when it concluded as follows:

> **3.5. CONCLUSIONS ON MODE OF ACTION CARCINOGENICITY**
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> As already demonstrated earlier, the *recent studies show* that hormonal carcinogens in humans and experimental animals are characterized by (i) tumorigenic action typically in various endocrineresponsive organs and/or tissues, and (ii) *the need for a prolonged exposure to high concentrations before tumorigenic effects become apparent*. The *studies are also consistent with the notion of hormone-receptor mediated increase in cell division and proliferation* in epithelial cells of the target tissues. *This points to a non-genotoxic mode of action*, which is in concurrence with (i) *the negative results of both earlier and recently performed genotoxicity tests*, and (ii) *the absence of structural alerts for genotoxicity in the molecule*. As cited in the introduction, *the recent extensive reviews by IARC and JECFA* also confirmed that the *tumorigenic action* of hormones, in particular 17β-oestradiol, in animals and man are *the consequence of the receptor-mediated, cell division stimulating activity of these compounds in somatic target cells*, and that the *potential genotoxic properties of the compounds would not be expressed in vivo and/or not play a role in the tumorigenic activity*.[62]

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[58] *See*, *e.g.*, 52nd JECFA Report (2000), pp. 58-60 (discussing effects of hormone replacement therapy, which involved exposure to estrogens at concentrations above the hormonal effect level). (Exhibit US-5).

[59] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, paras. 97, 129, 160, 168.

[60] 52nd JECFA Report (2000), p. 58. (Exhibit US-5).

[61] *See* 52nd JECFA Report (2000), p. 74, fn. 1. This conclusion also applies to the other two natural hormones, testosterone and progesterone. (Exhibit US-5).

[62] 1999 CVMP Report, pp. 8-9, citing the 52nd JECFA Report and the 1999 Report of the International Agency for Research on Cancer. *See also* 2005 U.K. Report, § 1.5.1, ("Overwhelming evidence suggests that sex steroids exert effects that are dose-dependent and that a threshold dose exists, below which, no biological effect will

38. In addition, the EC cites to the U.S. Department of Health and Human Services' "Report on Carcinogens" in support for the conclusion that estrogens are "known to be human carcinogens."[63] However, this statement is unexceptional when applied to estrogens generally, as it is in the cited Report.[64] As noted in our first written submission, there has been a battery of epidemiological tests focused on women and the use of hormone replacement therapies and oral contraception, both of which contain estrogens.[65] The Report on Carcinogens takes these studies into account in its analysis, as well as the conclusions of the 1999 Report of the International Agency for Research on Cancer ("IARC").[66] JECFA took these same studies into account in 1999, noting the "[e]pidemiological studies on women who took estrogens alone or in combination with progesterone and androgens, showed that the risks for cancers at most sites were unaffected; however, the risks for cancers of the endometrium and breast were increased."[67] However, it attributed these effects to the "hormonal effects of estrogens", *i.e.*, to levels of estradiol 17β or other estrogens high enough to have a hormonal effect on the consumer.[68] This is one of the reasons that JECFA determined that levels of estradiol 17β found in meat from cattle treated with the hormone for growth promotion purposes according to good veterinary practices (levels exponentially lower than those causing hormonal effects) is safe.

39. Similarly, the EC's CVMP, upon review of the same 1999 IARC monograph cited in the U.S. Report on Carcinogens,[69] as well as the scientific materials comprising the EC's 1999 Opinion, concluded that "[i]n confirmation of earlier studies provided . . ., most of the available recent data indicate that oestradiols and/or their synthetic analogues are *devoid of the ability to*

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occur.")

[63] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, paras. 98-99.

[64] *See* U.S. Department of Health and Human Services Report on Carcinogens, Eleventh Edition, "Estrogens, Steroidal", p.1 ("This listing of steroidal estrogens . . . applies to all chemicals of this steroid class.") (Exhibit US-26). Indeed, the 1987 Report of the IARC reached a similar conclusion regarding estrogens generally, but the *Hormones* panel determined that this conclusion had been taken into account by the relevant JECFA safety assessments addressing the relevant risk – that from estradiol 17β residues in meat from cattle treated with the hormone for growth promotion according to good veterinary practices. *See* Panel Report, para. 8.129.

[65] *See* U.S. First Written Submission, paras. 56, 69, 127, 146.

[66] *See* U.S. Department of Health and Human Services Report on Carcinogens, Eleventh Edition, "Estrogens, Steroidal" ("Report on Carcinogens"), p.1. (Exhibit US-26).

[67] 52nd JECFA Report (2000), p. 60. (Exhibit US-5).

[68] 52nd JECFA Report (2000), p. 60. (Exhibit US-5). Note that the Report on Carcinogens reached the same conclusion, stating that "[t]he evidence is strong that estrogen carcinogenesis is mediated through activation of the estrogen receptor", in other words, by the much smaller concentrations of estrogens sufficient to have hormonal effect. Not, as the EC appears to insinuate, by concentrations found in meat products from cattle treated according to good veterinary practices. Report on Carcinogens, p. 1. (Exhibit US-26).

[69] *See* 1999 CVMP Report, Section 3 ("Genotoxicity and Carcinogenicity"), in which the CVMP clearly sets out and analyzes a number of the studies relied upon by the EC in this dispute before reaching the conclusion that the evidence that estradiol 17β is non-genotoxic. (Exhibit US-13).

induce gene mutations or chromosome aberrations in vitro",[70] *i.e.*, that estradiol 17β is *"mainly devoid of genotoxic activity* and [] exerts its carcinogenic activity after prolonged exposures and/or at *levels considerably higher than those required for a physiological response*."[71] In other words, estradiol 17β "belong[s] to the group of non-genotoxic carcinogens" and "exogenous exposure to hormones would need to be substantial (*i.e.*, in the order of post-menopausal therapy levels[72]) before carcinogenic effects would be detectable in humans."[73] These conclusions do not ignore the fact that, at physiological, hormonal-effect concentrations, there are carcinogenic risks from estrogens. However, they do not support a theory that estradiol 17β is either genotoxic, or will have carcinogenic effects, at concentrations present in meat from cattle treated with the hormone for growth promotion purposes according to good veterinary practices.

40. The severe limitations of the alleged "evidence" for genotoxicity of estradiol 17β was also noted in the recent 2005 U.K. Report. In that Report, the Veterinary Products Committee ("VPC") concluded that only limited evidence was available to indicate that estradiol 17β is capable of inducing gene mutations, and cautioned that even this "evidence" has been obtained using non-standard assays, some of which suffer from flawed experimental design. The VPC noted the existence of limited evidence to indicate that metabolites of estradiol 17β have genotoxic potential *in vitro*; however, they could find no evidence to indicate that these metabolites are produced *in vivo* in sufficient amounts to overwhelm endogenous DNA repair pathways.[74]

b. The EC's Opinions fail to take into account available scientific evidence relating to the bioavailability of estradiol 17β, evidence relating to susceptible populations, and evidence relating to *in vivo* repair mechanisms

41. The EC also asserts that the U.S. argument that estradiol 17β is generally inactive when given orally, while "well known", is "still controversial and not consensually accepted by the scientific community."[75] To the contrary, estradiol's low oral bioavailability has found international support in Codex and JECFA ("[i]n general, estradiol 17β is inactive when given

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[70] 1999 CVMP Report, § 3.2.3 ("Conclusions on mode of action genotoxicity"), p. 7; *see also* 1999 CVMP Report, § 3.5. (Exhibit US-13).

[71] 1999 CVMP Report, § 5.1.2, p. 11. (Exhibit US-13).

[72] In oral post-menopausal therapies that contain estradiol 17β, doses range from 0.5-2.0 milligram/person/day [http://www.fda.gov/cder/ob/default.htm] while the highest excess intake of total estrogens from meat from cattle treated with estradiol 17β for growth promotion are 0.00003-0.00005 milligrams/person/day. *See* 52nd JECFA Report (2000), p. 73.

[73] 1999 CVMP Report, § 5.2.1, p. 12. (Exhibit US-13).

[74] *See* 2005 U.K. Report, § 6.5 ("Conclusions and recommendations"). (Exhibit US-20).

[75] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 100.

orally because it is inactivated in the gastrointestinal tract and liver"),[76] as well as support within the EC from the CVMP, which noted that "the *bioavailability of 17*β*-oestradiol* esters after oral administration *is low (3% as unchanged oestradiol)*, but might be higher if estron, an estrogenic metabolite, is included."[77] The EC's assertion is also surprising in light of unpublished, EC-generated data which the EC only recently provided to the U.S. which confirmed the internationally-accepted principle that bioavailability of estradiol 17β is low in humans.[78]

42. In an attempt to bolster its argument on the bioavailability of estradiol 17β, the EC cites to data it has developed on estrogen levels in young children.[79] While it is unclear how this comparison relates in any way to a discussion on bioavailability, we can only assume that the EC makes this argument in an attempt to cast doubt on previously established standards for estradiol 17β and the other hormones at issue, *i.e.*, that the relevant standard setting groups were not taking into account populations identified as more sensitive than previously thought. The EC's argument fails for two fundamental reasons: (1) populations such as young children were indeed taken into account in establishing international standards and domestic requirements for the six hormones; and (2) the studies cited by the EC by which it attempts to cast such groups as even more susceptible than previously thought are flawed.

43. As to the first point, JECFA, in its safety assessment for the hormones, including estradiol 17β, took into account data on most sensitive populations. In order to accommodate for these groups, in the case of estradiol 17β, it applied a "safety factor" of 10 "to account for normal variation among individuals" to its initial determination as well as an additional factor of 10 "to

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[76] *See* 52nd JECFA Report (2000), p. 58. (Exhibit US-5). *See also* 2005 U.K. Report, § 1.5.2 ("Moreover, as human exposure will be via food, the absorption and metabolism of the compound in the gut becomes very important. Oral absorption of oestradiol is good, however, the quantity reaching the systemic circulation is greatly reduced by extensive first-pass metabolism in the intestines and liver, and oestradiol is *generally considered to be inactive when administered orally*.") (Emphasis added). (Exhibit US-20).

[77] 1999 CVMP Report, p. 2. (Exhibit US-13).

[78] *See* Hoogenboom, Investigations on the metabolism of 17β-estradiol by bovine hepatocytes, human intestinal and breast cells, and the genotoxic and estrogenic properties of the metabolites (unpublished). (Exhibit EC-6(US)). In this study, estradiol 17β was added to immortalized human intestinal cells (Caco-2 cells). These cells can be grown as a monolayer *in vitro* and thus represent a model for the human gastrointestinal tract, the known site of estradiol 17β metabolism *in vivo*. The author found that *no estradiol 17*β *was transported across the intestinal cell monolayer*; only estrone, a toxicologically benign metabolite of estradiol 17β, was detected on the other side of the monolayer. These results indicate that human intestinal cells convert estradiol 17β to estrone, and confirm the internationally-accepted principle that bioavailability of estradiol 17β is low in humans. This interpretation of the data is clearly shared by the author of the study, who concluded that "[t]his indicates that 17β-estradiol is not absorbed intact in the human intestinal tract." Hoogenboom, p. 5.

[79] *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 156.

protect sensitive populations."[80] The CVMP, in determining that estradiol 17β is safe within certain concentrations also took into account data on prepubertal boys.[81]

44. As to the second point, the EC's own CVMP made the following comments on one of the main assays used by the EC in purportedly establishing a lower circulating estrogen levels in boys, the "Klein assay":

> It was noted that the report by Klein et al. (1994)[82] indicated much lower plasma levels of oestradiol when measured with a new method, based on β-galactosidase gene expression in genetically modified yeast, compared to the classical RIA requirements (Klein et al., 1994). However, (i) *the measure was made only in plasma and needs to be carried out in other tissue(s) to enable to comparison between the intake of residual oestradiol and the endogenous levels*, [and] (ii) the *methodology needs validation and is not (yet) generally accepted*.[83]

The U.K. Group further emphasized this concern, setting out as one of its main conclusions that it had "concerns about the validity and selective applicability of a key analytical approach cited in the Opinion, which was based on an assay for apparent oestrogenic activity done in genetically modified yeast. The concerns were sufficient to throw doubt upon the values derived from this analytical technique and therefore also on the conclusions of the Opinion."[84]

45. The EC's Opinions also conclude that meat from cattle treated with estrogens may accelerate the onset of puberty in children.[85] The EC attempts to support this conclusion with a publication describing an outbreak of breast enlargement (gynecomastia) in school children in Milan in 1977. The study's authors state that estrogenic contamination of meat served in the school canteen was the "suspected" cause of breast enlargement. However, the presence of estrogen in meat consumed by the students was never confirmed and a causal link between estrogen in meat and gynecomastia was never demonstrated. Indeed, in a retrospective study conducted some twenty years later, the original study's author questions his own earlier

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[80] 52nd JECFA Report (2000), p. 60. (Exhibit US-5).

[81] 1999 CVMP Report, p. 12 ("In an earlier evaluation, *CVMP had based its risk assessment on* the relation between any possible excess of hormones from zootechnically treated animals in the diet and *the endogenous daily production of oestradiol in prepubertal boys*, the latter value being estimated as 6μg per day.") (Emphasis added). (Exhibit US-5).

[82] *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 156 (in which the EC invokes the Klein assay as its scientific evidence for lower than previously thought estrogen levels in prepubertal boys).

[83] 1999 CVMP Report, p. 12. (Exhibit US-13).

[84] 1999 U.K. Report, pp. 2, 28 *et seq*. (Exhibit US-13); *see also* U.S. First Written Submission, fn. 92.

[85] *See* 1999 Opinion, p. 14. (Exhibit US-4). The findings of the study cited in support of this claim in the 1999 Opinion (Fara G. M. *et al*., Epidemic of breast enlargement in an Italian school, Lancet (1979) 11:295-297) were subsequently included in a review article that was published as part of Study 12 of the EC's 17 Studies (Chiumello G. *et al*., Accidental gynecomastia in children, APMIS (2001) 109, Suppl. 103: S203-209). (Exhibit EC-6(US)).

conclusions and recognizes the likelihood that some other factor caused the early onset of puberty, asking:

> Does the presumed cause of the outbreak (eating contaminated food) seem likely, or should some other environmental factor affecting the entire school population, and not just the subjects who ate in the canteen, be sought, given the similarity of the alterations found in all the subjects of the exposed school?[86]

The results of this unpublished, and previously unavailable study clearly demonstrate that the conclusion that hormones in meat are causative factors for early onset of puberty is unfounded.

46. In addition, the EC's Opinions ignore the scientific evidence relating to human *in vivo* DNA repair mechanisms, specifically that genotoxic effects of relevant residue levels of growth promoting hormones would not be expressed *in vivo* based in part on the existence of the efficient DNA repair mechanisms that exist in all mammalian cells. These repair mechanisms protect cells from genotoxicity. Genotoxic effects are manifest only when DNA repair mechanisms are overwhelmed by a genotoxic agent. The efficacy of these repair mechanisms is exemplified in one of the unpublished reports recently provided by the EC.[87] In this study, it was suggested that the lack of genotoxic effects of estradiol 17β on human intestinal cells was due to a very efficient and rapid repair system.[88] Despite these relevant findings, however, the EC's Opinions completely ignore the influence of endogenous DNA repair mechanisms, and instead attempt to implicate genotoxicity as a basis for the purported human health risk associated with estradiol 17β residues in meat and meat products at any concentration.

47. For these reasons, as well as those set out in the other U.S. submissions to the Panel, the EC's ban on meat and meat products from cattle treated with estradiol 17β for growth promotion purposes according to good veterinary practices is not "based on", or sufficiently warranted or rationally related to its purported risk assessment within the meaning of SPS Article 5.1. The EC's Opinions fail to adduce scientific evidence of a risk to consumers posed by meat from cattle

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[86] *See* Chiumello G., Long-term effects in children of exposure to estrogen-contaminated meat: a retrospective group study, p. 19. (Exhibit EC-6(US)). This retrospective study of the student population was conducted some 20 years after the incidence of gynecomastia, and was included in the unpublished information recently provided to the U.S. by the EC. Although the author reports a trend for both sexes toward earlier puberty in females in the exposed group, there were no statistically significant differences in the age of puberty between exposed individuals and control subjects (control subjects were former students of another school in Milan). Moreover, there were no significant differences between the exposed group and the control group with respect to endocrine parameters or gynecological or urological pathologies.

[87] *See* Hoogenboom, Investigations on the metabolism of 17β-estradiol by bovine hepatocytes, human intestinal and breast cells, and the genotoxic and estrogenic properties of the metabolites (unpublished). (Exhibit EC-6(US)). *See also* 2005 U.K. Report, § 6.1 ("The study by Hoogenboom and colleagues showed that 17β-oestradiol and several of its metabolites were *negative* in a series of apparently well-conducted bacterial mutagenicity assays using a variety of strains . . .. Furthermore, they also reported *negative responses* in the Comet assay using human intestinal cells (CaCo-2).") (Emphasis added). (Exhibit US-20).

[88] Hoogenboom, p. 31. (Exhibit EC-6(US)).

treated with estradiol 17β for growth promotion purposes according to good veterinary practices. Assessments which conclude otherwise (*i.e.*, that such a risk exists), such as the EC's 1999 Opinion, 2000 Review and 2002 Opinion, therefore do not "take into account available scientific evidence,"[89] and are not risk assessments as appropriate to the circumstances within the meaning of Article 5.1 of the SPS Agreement.

4. The EC fails to demonstrate that there is a risk of failure of controls or failure to satisfy good veterinary practices

48. The EC's replies to the Panel's questions were enlightening regarding what, exactly, is the perceived "risk" against which the EC has imposed its import bans on U.S. meat and meat products. From the outset, the United States has argued that the scientific evidence, and the EC's Opinions and 17 Studies, do not demonstrate a risk from the six growth promoting hormones when used for growth promotion purposes according to good veterinary practices. Our focus on this specific risk and exposure pathway seemed obvious because this is the legally permitted use of growth promoting hormones in the United States. This focus also seemed obvious since, if a WTO Member were concerned about a failure of controls, or a failure to satisfy good veterinary practices, there are countless less trade restrictive methods for mitigating against this perceived risk than an absolute ban on another Member's goods, and Article 5.6 of the SPS Agreement requires Members to ensure that their SPS measures are not more trade-restrictive than required. Furthermore, a logical extension of the EC's argument is that since the EC cannot be confident its own controls will never fail (indeed, as discussed below, there is evidence that these controls have failed), the EC should ban all EC meat and meat products.

49. Nevertheless, the EC's replies consistently invoke the "risk" of a failure to satisfy good veterinary practices.[90] Indeed, it is as a result of this additional perceived "risk" that the EC appears to discount the conclusions reached in previous JECFA risk assessments and set out in Codex standards as MRLs and ADIs. For instance, the EC dismisses the JECFA conclusions on the safety of the six hormones when used for growth promotion purposes according to good veterinary practices because it "could not adopt the risk management options proposed by JECFA",[91] adding that:

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[89] SPS Agreement, Article 5.2.

[90] *See*, *e.g.*, Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 95 ("another important qualification comes from the proper implementation of GVP and the possibilities available to control good veterinary practice, as discussed in the answer to the previous question"); para. 129 (distinguishing its conclusions from JECFA's, noting that the "European Communities came to the conclusion that the JECFA's recommendations could not achieve the level of health protection considered appropriate by the European Communities in its territory from *residues of these hormones under realistic conditions of use for animal growth promotion*.") *See also*, Replies to Questions from the Panel after the First Substantive Meeting by European Communities, paras. 76, 83, 136 *et seq*., 151, 168, 172.

[91] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 129.

> the European Communities regulatory authorities concluded that the prohibition of the use of hormones for growth promotion within the European Communities and the exclusion of import of meat derived from animals treated with hormonal growth promoters was the most appropriate measure in order to protect its consumers from the risks identified from excess intake of hormone residues and their metabolites and *the potential for abuse*, inter alia, *through non-observance of good husbandry practices*. In other words, *the European Communities came to the conclusion that the JECFA's recommendations could not achieve the level of health protection considered appropriate by the European Communities in its territory from residues of these hormones under realistic conditions of use for animal growth promotion*.[92]

As we will discuss in detail, the examination of the potential for failures of control or a failure to meet good veterinary practices are legitimate factors for consideration in risk assessments. Nevertheless, the EC's focus on "risk management" and its assumption that good veterinary practices will not be met raises serious concerns regarding the trade restrictiveness of their chosen measure, an outright ban on all meat and meat products, as well as with their purported analyses of the possibility of such failures, which lack any support.

a. "Risk management" and Article 5.2 of the SPS Agreement

50. In its replies to the Panel's questions, the EC frequently cites to the processes of "risk analysis" and "risk management",[93] neither of which are explicitly referred to in the text of the SPS Agreement. As noted by the Appellate Body in the *Hormones* dispute, "[w]e must stress . . . that Article 5 and Annex A of the SPS Agreement speak of 'risk assessment' only and *that the term 'risk management' is not to be found either in Article 5 or in any other provision of the SPS Agreement*."[94] This is why, to quote the EC, "[t]he United States . . . make[s] little reference to risk management" in its submissions.[95]

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[92] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 129 (emphasis added); *see also* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 165 (noting that "*the exclusion of import of meat derived from animals treated with hormonal growth promoters was the most appropriate measure in order to protect its consumers from* the risks identified from excess intake of hormone residues and their metabolites and *the potential for abuse, inter alia, through non-observance of good husbandry practices*.") (Emphasis added).

[93] *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 76 ("The European Communities will indicate separately the basis of its 'risk management' that led to the adoption of the new Directive 2003/74.")

[94] Appellate Body Report, *EC – Hormones*, para. 181.

[95] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 136.

51. This is not to say that concepts such as "risk management" and "risk analysis" find no expression in the SPS Agreement whatsoever. Rather, they may be found in, *e.g.*, Article 5.2 of the SPS Agreement,[96] which states:

> In the assessment of risks, Members shall take into account available scientific evidence; relevant processes and production methods; relevant inspection, sampling and testing methods; prevalence of specific diseases or pests; existence of pest- or disease-free areas; relevant ecological and environmental conditions; and quarantine and other treatment.

Article 5.2 provides examples of what should be taken into account in a risk assessment for purposes of Article 5.1 and paragraph 4 of Annex A to the SPS Agreement. Many of these examples may be linked to what the EC refers to as "risk management."[97]

52. For instance, an examination of "relevant processes and production methods" would presumably incorporate an analysis of how growth promoting hormones are approved for use as well as how cattle are actually treated with growth promoting hormones in the United States. Similarly, an analysis of "relevant inspection, sampling and testing methods" would include an examination of the types of safeguards in place in the United States to prevent against abusive use of growth promoting hormones in cattle, as well as an examination of the efficacy of those safeguards. Whether or not the EC engaged in a proper evaluation of these factors, within the meaning of SPS Article 5.2, would inform a decision on whether or not they have indeed properly assessed the risk of failure to satisfy good veterinary practices within the meaning of Article 5.1 and Annex A of the SPS Agreement. As discussed below, the EC has not engaged in the necessary evaluation of these factors as required by Articles 5.1, 5.2 and Annex A of the SPS Agreement.

53. The EC avers that its Opinions examine the theoretical risk from residues in meat from cattle treated with hormones for growth promotion purposes according to good veterinary practice.[98] Yet, the ensuing analysis set out by the EC does not examine the actual residue levels that would be found in meat from cattle treated according to good veterinary practices, the subject of an inquiry by the Panel, but rather addresses a hypothetical failure to satisfy good veterinary practices on the part of U.S cattle producers. The EC subscribes to the theory that "if a product is said to be safe if GVP is followed, then it may or may not be safe if GVP is not implemented."[99] This theory is unobjectionable. In fact, it appears to acknowledge that the

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[96] *See*, *e.g.*, Appellate Body Report, *EC – Hormones*, para. 206.

[97] *See*, *e.g.*, Replies to Questions from the Panel after the First Substantive Meeting by European Communities, paras. 76, 83, 129, 136 *et seq*., 151, 168, 172.

[98] *See* Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 85.

[99] Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 87.

growth promoting hormones *per se* are indeed safe when used according to good veterinary practices, and that any actual risk only follows from a failure to use the hormones accordingly. However, the EC fails in its Opinions to conduct the necessary evaluation of a risk from failure of controls.[100]

b. The EC's Opinions fail to take into account available scientific evidence, relevant processes and production methods and relevant inspection, sampling and testing methods as they relate to the hypothetical risk of failure to satisfy good veterinary practices as required by Article 5.2 of the SPS Agreement

54. Specifically, contrary to the requirements of Article 5.2 of the SPS Agreement, the EC has failed to examine available scientific evidence, relevant processes and production methods and relevant inspection, sampling and testing methods as they relate to a hypothetical risk of a U.S. failure to satisfy good veterinary practices.[101] Had the EC engaged in such an analysis it would have, for example, examined relevant processes and production methods and relevant inspection sample and testing methods as they actually exist in the United States. This would include, as required by Article 5.2, analysis of the efficacy of processes currently in place in the United States. Significantly, the EC's bans do not vary by, or look at, the conditions in the country of export, demonstrating that it in fact is unrelated to the question of the efficacy of good veterinary practices.

55. The United States has rigorous controls in place, which include the establishment of tolerances (maximum allowable levels) for hormone residues in food by the Food and Drug Administration, and USDA/Food Safety and Inspection Service ("FSIS") enforcement of these tolerances through (1) residue control programs; and (2) ante mortem, post mortem, and processing inspection, to which all cattle entering the human food supply are subjected.[102] This system provides extremely efficient safeguards against a hypothetical failure of controls in the United States, while at the same time being significantly less trade restrictive than an outright ban on U.S. meat and meat products. A review of these relevant factors in its Opinions would have assisted the EC in making its ultimate determination of whether a ban on U.S. meat and meat products is "not maintained without sufficient scientific evidence" and is not "significantly

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[100] Interestingly, the EC appears to recognize this shortcoming by informing the Panel of its need to provide "specific evidence from misuse of these hormones that has recently arise [sic] in the territory of the defending Members" in its rebuttal submission, rather than simply citing to its purported "risk assessment", where such information should be included. Replies to Questions from the Panel after the First Substantive Meeting by European Communities, para. 91. It remains unclear how the can EC unilaterally decide to provide this information for the first time in its rebuttal submission and remain in compliance with paragraph 13 of the Working Procedures.

[101] *See* SPS Agreement, Article 5.2; *see also* Panel Report, *Japan – Apples (21.5)*, paras. 8.145.

[102] As a final deterrent, the United States ultimately has the authority to seek an injunction prohibiting the sale of cattle by any producer who fails to satisfy good veterinary practices.

more trade restrictive than required to achieve the appropriate level of sanitary or phytosanitary protection" as required by the SPS Agreement.[103]

56. Instead, the EC's Opinions focus on several hypothetical "failure of control" scenarios that ignore actual regulatory processes in the United States, and for which it presents no support. It asserts that these scenarios "clearly identify a risk for excessive exposure of consumers to residues from misplaced or off-label used implants and incorrect dose regimes."[104] Yet, the EC fails to produce any evidence identifying a real risk of failure of controls or failure to satisfy good veterinary practices in the United States. As noted by the compliance panel in *Japan – Apples*, the evidence relied upon by a Member must actually support the conclusions reached in that Member's risk assessment.[105] In other words, the EC may not simply set out conclusions in its Opinions that are not actually grounded in the studies or evidence it cites as support.

i. Scenario 1: Implanted ears enter the human food chain

57. The EC's 1999 Opinion alleges that "from 6% to 30% of the original dose [of a hormone implant] remained in the ears from 65 to 150 days after implantation . . .. These data indicate that consumption of tissue from implantation sites would result in substantial exposure."[106] This hypothetical assumes that ears containing implants will enter the human food chain, but provides no evidence in support of this scenario. Contrary to the EC's assumption, very clear instructions are provided on manufacturers' labels on all FDA-approved growth promoting implants, indicating that implants must be placed beneath the skin of the middle third of ears of cattle. Because ears are then discarded at slaughter, excess dietary exposure to hormone residues via consumption of implant sites does not occur. USDA inspectors confirm through ante- and post-mortem inspections that ears are discarded and that no hypothetical misplaced implants enter the human food chain. Therefore, the EC's conclusion that cattle ears containing hormone implants will enter the human food chain is unsupported by relevant scientific evidence and real world conditions.

ii. Scenario 2: Misplaced implants enter the human food chain

58. The EC's Opinions also contemplate a scenario whereby growth promoting hormone implants are placed in parts of cattle other than the ear. The EC discusses this scenario in two of its "17 Studies", in which, for purposes of the experiment, EC scientists placed implants in the muscle of the necks of cattle and pigs (a species in which the use of implants is not even

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[103] *See* SPS Agreement, Article 5.6. If the EC were to assess the risk of a failure to observe good veterinary practices, then it was not an option for the EC to review these factors, it was a requirement for the development of its risk assessment. *See* SPS Agreement, Article 5.2.

[104] 2002 Opinion, pp. 11-12. (Exhibit US-1).

[105] *See* Panel Report, *Japan – Apples (21.5)*, paras. 8.145.

[106] 1999 Opinion, pp. 30-32. (Exhibit US-4).

approved in the United States).[107] The studies' hypothesis is apparently that intramuscular injection of hormone implants would make them invisible from the surface of the carcass, thus facilitating multiple treatments of an animal, or permitting hidden implants to enter the food chain. In support of its claim that this is a realistic scenario, the EC asserts that "correct implantation can neither be guaranteed nor expected."[108] However, the EC provides no evidence in support of this claim. The scenario developed by the EC in its laboratories is just that – a hypothetical laboratory experiment with no grounding in or evidence relating to the real world use of implants. The findings of such experiments, which are not grounded in the real world use of implants and growth promoting hormones, are unresponsive to the Panel's request that the EC provide evidence in support of its conclusion that "misplaced implants and repeated implanting seem to occur frequently."[109]

59. In the real world, the likelihood that a U.S. beef producer would intentionally misplace hormone implants in muscle is negligible given the economic and enforcement considerations at stake. First, the implants are specifically designed to achieve maximum effect when inserted into the animal's ear. There is therefore no economic benefit for injecting cattle in other parts of the body. Second, misplaced implants would ruin surrounding muscle tissue, thereby decreasing the value of the carcass. Furthermore, placement of an implant in muscle would require the use of a large needle that would result in considerable damage and permanent scarring at the site of injection.[110] Third, discovery of any intramuscular (non-ear) implants at slaughter by a federal inspector would cause the entire carcass to be condemned, resulting in not only zero profit, but significant economic loss to the producer.

iii. Scenario 3: Multiple implanting of cattle

60. In another portion of the failure of controls discussion, the EC's Opinions allege that overdosing cattle with hormone implants is commonplace in the United States. For instance, the EC's 1999 Opinion concludes that "[v]arious examples of overdosing by simultaneous and/or repeated implantation of various implants in USA feedlots have also been documented by EC

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[107] *See* Daxenberger A. *et al*., Detection of Anabolic Residues in Misplaced Implantation Sites in Cattle, J. AOAC International (2000), 83:154-158 (part of study five of the "17 Studies") (Exhibit EC-6(US)); and Daxenberger A *et al*., Suppression of androstenone in entire male pigs by anabolic preparations, Livestock Production Science (2001) 69:139-144 (part of study five of the "17 Studies") (Exhibit EC-6(US)); *see also* Australia Review, p. 39 ("[t]he major finding from residue analyses in cattle is that proper use of registered HGPs (as per label directions) does not generate violative levels of residues (ie. above MRLs) (Daxenberger et al 2000; Daxenberger et al 2001; Lange et al 2001). In fact, off- label use of zeranol and testosterone at up to 10-fold the label dose did not lead to violations of Codex MRLs or FDA allowable residue levels (Lange et al 2001).") (Exhibit US-16). (Note that the Lange study is also part of study five of the "17 Studies").

[108] 2002 Opinion, p. 11. (Exhibit US-1).

[109] Panel Question 27 to the EC.

[110] *See* Daxenberger A *et al*., Application of anabolic agents to food producing animals health risks caused by disregarding the requirements of good veterinary practices (part of study number five of the "17 Studies"), p. 36 (noting that misplacement of implants causes "hygienic impairment" of the carcass). (Exhibit EC-6(US)).

inspection missions."[111] However, overdosing constitutes an "off-label" use of growth promoting hormones, *i.e.*, use of the hormones for purposes other than those for which they are approved. Such use is not permitted in the United States. The EC fails to provide evidence to support its conclusion that off-label use actually occurs in the United States.

61. The EC Opinion does, however, cite to one publication from which it extrapolates its conclusion that off-label use of hormones occurs, but it appears to misinterpret the data and information provided in that document.[112] While the publication in question does indeed illustrate potential implant programs involving re-implanting cattle at defined intervals, there is a period of several weeks between administration of each (single) implant, during which time the concentration of the hormone in the animal has progressively decreased, and each of the programs constitutes proper, on-label, authorized use of hormone implants in the United States.[113] Furthermore, there is no economic incentive for the off-label implant use alleged by the EC; to the contrary, such use would have negative economic effects on cattle producers.[114] Therefore, the Opinions' conclusion that off-label use of hormone implants occurs in cattle for export to the EC in the United States is unsupported by available scientific evidence and real world conditions.

62. Indeed, the conclusion that multiple implanting poses a hypothetical health risk to consumers appears to ignore the findings of some of the very laboratory studies commissioned by the EC ostensibly in support of its claim that multiple implanting actually occurs and poses a risk to consumers in the real world. Accordingly, the EC fails to take into account available scientific evidence related to this "risk" within the meaning of SPS Article 5.2. For example, in one of the EC's unpublished contract reports[115] recently reviewed by the United States, the EC presents data clearly supporting the human food safety of growth promoting implants in cattle. In this study, cattle were treated with 0, 1, 2 or 4 implants containing estradiol 17β and the amounts of the hormone in edible tissues were measured at slaughter. The authors concluded that ingestion of a standard portion of meat from an animal treated with a single implant amounted to only 1.4% of the ADI for estradiol 17β. In the case of multiple implanting, the researchers concluded that consumption of a standard portion of meat from cattle treated with two or four implants would contribute less than 10% of the ADI for the hormone.[116]

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[111] 1999 Opinion, p. 31.

[112] *See* Griffin and Mader, Beef Cattle Implant Update (1997) (http://ianrpubs.unl.edu/beef/g1324.htm). (Exhibit US-27).

[113] Dr. Griffin, one of the authors of the 1997 Update, upon learning of the conclusions for which his publication has been cited, provided a brief letter clarifying the data in the document. This letter has been attached as Exhibit US-28.

[114] *See*, *e.g.*, para. 59 above.

[115] *See* Metabolic pathways of estrogens used as steroidal growth promoting agents (Study no. XL/98/EUROESTR) (unpublished). (Exhibit EC-6(US)).

[116] Similar results were published as part of study three of the "17 Studies" (1.3% of the ADI for a single implant and less than 7% of the ADI for up to 4 implants). *See* Maume D. *et al*., Assessment of estradiol and its metabolites in meat, APMIS (2001); 109: 32-38 (Exhibit EC-6(US)).

63. In another study commissioned by the EC, overdosing was again simulated by treating cattle with multiple implants containing zeranol and testosterone propionate, and residue levels in tissues then measured at slaughter. Even in a simulation of extreme misuse (ten implants per animal), residue levels of testosterone and zeranol did not exceed Codex MRLs. As noted by the authors "[t]reatment with zeranol and testosterone propionate, even after multiple application, does not cause any problems, as far as infringement of threshold levels is concerned."[117]

iv. Scenario 4: "Black market" for growth promoting hormones

64. The EC's Opinions also cite to the existence of "black market" drugs, other non-authorized pharmaceutical formulations, or hormone "cocktails" as contributing to the risk of a failure of controls.[118] However, the EC again provides no evidence of such a black market actually existing in the United States.[119] Indeed, the analysis set out in the EC's Opinions ignores available evidence relating to black markets, as well as relevant processes and production methods and relevant inspection, sampling and testing methods as they exist in the United States. Available materials focusing on the black market use of growth promoting hormones only discuss evidence of such a market for their use within the EC (and thus would indicate that the EC, under its own approach in this dispute, should ban the sale of EC meat and meat products).[120] EC inspection missions appear to confirm this fact.[121]

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[117] *See* Lange I. G. *et al*. (part of study five of the "17 Studies"), Residue levels of trenbolone acetate were also considered by the authors, who determined that the MRL for trenbolone was only exceeded after overdose with ten implants per animal, and then only in liver tissues (*i.e.*, not in muscle, kidney or fat). (Exhibit EC-6(US)).

[118] *See* 1999 Opinion, § 3.3.3, "Black-market drugs" ("The possibility that non-authorized pharmaceutical formulations of hormones will be used in animals can not be excluded. These black market drugs may comprise an even higher risk to public health, as their quality is inadequate.") (Exhibit US-4).

[119] *See* Stephany, Hormones in meat: different approaches in the EU and in the USA, APMIS 109, p. S 357 (2001) ("It has to be concluded that in some EU Member States an exten[d]ed black market exists. For the USA, *no experimental evidence is available for such a black market.*") (Exhibit US-29).

[120] *See* Stephany, Hormones found in meat samples from regular controls within the European Union and from U.S. imports, Chemical Awareness 9, p. 13 (July 5, 2000) ("In the EU dozens of illegal hormones are used, and the number of active compounds is continuously changing.") (Exhibit US-30); Stephany, Hormones in meat: different approaches in the EU and the USA, APMIS 2001, pp. 359, 361 ("In the EU in the past three decades dozens of illegal hormones are used," and "[a] defensible overall estimate for the use of these compounds in the European Union based on results from annual regulatory testing programmes could be in the range of 5-15 percent.") (Exhibit US-29).

[121] *See* Final Report of a Mission Carried Out in Germany from 07/05/01 to 18/05/01 in Order to Evaluate the Control of Residues in Live Animals and Animal Products (DG(SANCO)/3267/2001), p. 21 ("*The recent incidents of illegal drug traffic*: Already in 1999 there had been investigations in Bavaria suspecting that some veterinarians were distributing drugs by mail without checking animals . . . [i]t became obvious that these veterinarians had sold veterinary drugs to farms almost all over Germany.") (Exhibit US-31). *See* Final Report of a Mission Carried Out in Portugal from 17 to 21 November 2003 in Order to Evaluate the Control of Residues in Live Animals and Animal Products (DG(SANCO)/9209/2003) (noting several residue violations in § 5.4.1 ("Group A violations.")) (Exhibit US-32).

65. Nevertheless, despite the fact that there is no evidence of a black market in the United States, the reference to a growth promoting hormone "black market" in the EC's Opinions is ultimately informative, because the lack of such a market in the United States highlights the shortcomings of instituting a ban in response to a theoretical risk of a failure of controls or failure to satisfy good veterinary practices. There is evidence that the EC, which chose to ban the use of growth promoting hormones, suffers from varying degrees of misuse or failure to abide by good veterinary practices in the form of "black market" use of hormones whereas the United States, which permits their appropriate use, does not.[122] The presence of this market emphasizes that a total ban is not necessarily the most effective (and certainly not the least trade restrictive) means of preventing a theoretical failure of good veterinary practices. The *Hormones* panel reiterated this concern, noting that "the banning of a substance does not necessarily offer better protection of human health than other means of regulating its use."[123]

c. Conclusion

66. In conclusion, it is not surprising that the EC now argues that there is a risk of failure of controls since, as we have demonstrated, the preponderance of the EC's studies (in particular the "17 Studies") assume subject exposure to hormones at levels orders of magnitude higher than would be ingested from meat from cattle treated with hormones according to good veterinary practice.[124] The next logical step, upon failure to demonstrate evidence of adverse effects at relevant exposure levels (those from meat from cattle treated with growth promoting hormones according to good veterinary practices), is to argue that good veterinary practices are not in fact satisfied, or that there is a failure of controls on the part of the meat exporter. However, as demonstrated above, the EC has failed to demonstrate in its Opinions that such a risk exists by failing to take into account available scientific evidence and relevant processes and production methods and relevant inspection, sampling and testing methods as they exist in the United States. The EC has therefore failed to base its measure on a risk assessment, such that it is sufficiently warranted by or rationally related to such a hypothetical risk as required by Article 5.1 of SPS Agreement.

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[122] *See* Stephany, Hormones in meat: different approaches in the EU and in the USA, APMIS 109, p. S 357 (2001) ("For the USA, *no experimental evidence is available for such a black market.*") (Emphasis added). (Exhibit US-29).

[123] Panel Report, *EC – Hormones*, para. 8.146. *See also* 2005 U.K. Report, p. 9 (noting that "illegal or improper use of growth-promoting substances, in the form of implants and/or in feed might present an added exposure to humans who consumed meat or meat products from animals so treated. However, this would be no different from the illegal or inappropriate use of any veterinary products . . ..").

[124] *See*, *e.g.*, U.S. First Written Submission, at paras. 144-146.

III. CONCLUSION

67. In light of the foregoing, the United States asks the Panel to find that:

(1) The EC has failed to demonstrate that the United States has breached DSU Article 22.8, and that the United States continues to suspend concessions to the EC consistent with the requirements of that provision;

(2) The United States has not breached DSU Articles 3.7, 21.5, 23.1 or 23.2(a); and

(3) The United States has not breached GATT 1994 Articles I or II.

TABLE 1

Comments on the Availability of the 17 Studies

As the Panel is aware, the United States requested an extension in filing its rebuttal submission in this dispute to afford it more time to evaluate materials put forward by the EC as exhibits to its replies to questions from the Panel. In particular, the United States noted that several of the materials had not previously been made available to the United States for review. In this Table, we provide an accounting of these new materials.

As a preliminary point, we would make the general comment that there appears to have been a change in the EC's perspective of what materials comprise the "17 Studies". The EC's original list of the 17 Studies, which was appended to the EC's 2002 Opinion (*see* 2002 Opinion, pp. 28-30 (Exhibit US-1)), provided the public list of studies of which the United States was aware, and which the EC referred to in response to the U.S. SPS Article 5.8 information request in May, 2005. A side-by-side comparison of this original list with the list of the 17 Studies recently provided to the Panel in this dispute reveals differences (*see* table listing the 17 Studies in Exhibit EC-6(US)). Specifically, the "new" list of the 17 Studies (Exhibit EC-6(US)) contains eleven studies that were either not listed or not publicly available as of the publication of the original, 2002 Opinion list. These studies are:

- Materials comprising Study number four, "Metabolites of melengestrol acetate, trenbolone acetate & zeranol in bovine & humans" (in the 2002 Opinion, the EC noted that "[p]ublication [of the study is] foreseen", yet no publication appears to have taken place).

- "Biochemistry and physiology of anabolic hormones used for improvement of meat production" (APMIS 109: 1-8, 2001) (part of Study number nine).

- Materials comprising Study number ten, "Interaction of xenobiotics with sex hormone binding globulin; impact on endogenous steroid transport, bioavailability, mechanism of action" (in the 2002 Opinion, the EC commented that unavailability was due to the fact that the "scientist has not yet indicated name of journal and publication date", yet no publication appears to have taken place).

- Materials comprising Study number eleven, "Reproductive sequelae of developmental exposure of rabbits to trenbolone, zeranol & MGA; emphasis on differential & neoplastic transformation of germ, cells" (in the 2002 Opinion, the EC noted that "[p]ublication [of the study is] foreseen by the end 2001", yet no publication appears to have taken place).

- "Possible health impact of animal estrogens in food" (Human Reproduction, Vol. 7, no 3, 340-355: 2001) (part of Study number fourteen).

- "Possible health impact of phytoestrogens and xenoestrogens in food" (APMIS, 109, 161-184: 2001) (part of Study number fourteen).

- "Human exposure to endocrine disrupters: standardisation of a marker of oestrogenic exposure in adipose tissue" (APMIS, 109, 185-197: 2001) (part of Study number fourteen).

- Materials comprising Study number fifteen, "Screening samples for estrogenic & androgenic anabolic chemicals" (in the 2002 Opinion, the EC noted that the "scientist has not yet indicated name of journal and publication date", yet no publication appears to have taken place).

- "Mobility of the growth promoters trenbolone and melengestrol acetate in agricultural soil: column studies" (Science of the Total Environment 326, 225-237: 2004) (part of Study number sixteen).

- "Androgenic and estrogenic activity in water bodies receiving cattle feedlot effluent in Eastern Nebraska - USA" (Environmental Health Perspectives, Vol. 112, no 3, pp. 346-352, March 2004) (part of Study number sixteen).

- "Endocrine-disrupting effects of cattle feedlot effluent on an aquatic sentinel species, the fathead minnow" (Environmental Health Perspectives, Vol. 112, no 3, pp. 353-358, March 2004 (part of Study number sixteen).

Prior to this recent listing and inclusion of these studies in the EC's replies to questions from the Panel, the United States was not aware that the EC relied on these studies as support for its import bans (nor could it have been since these materials are not referenced in the list of the "17 Studies" attached to the EC's Opinions nor were they mentioned in the EC's response to the U.S. SPS Article 5.8 request). As a result, we could not have reviewed these publications prior to this point. To the best of our knowledge, due to their lack of publication, the materials comprising Studies four, ten and eleven would not have been available even had we been aware of their completion.

In addition, we would note that study one of the EC's "17 Studies" ("Presence of estrogens in meat (delivery of samples)") has never been made available for review by the United States.

TABLE OF EXHIBITS

Exhibit US-	Title of Exhibit
23	EC Response to U.S. SPS Article 5.8 Request
24	Fifty-Fourth Report of the Joint FAO/WHO Expert Committee on Food Additives, WHO Technical Report Series: 900 (2001) ("54th JECFA Report")
25	Thirty-Second Report of the Joint FAO/WHO Expert Committee on Food Additives, WHO Technical Report Series: 763 (1988) ("32nd JECFA Report")
26	U.S. Department of Health and Human Services Report on Carcinogens, Eleventh Edition, "Estrogens, Steroidal" ("Report on Carcinogens")
27	Griffin and Mader, Beef Cattle Implant Update (1997) (http://ianrpubs.unl.edu/beef/g1324.htm).
28	Letter from Dr. Dee Griffin explaining results of Beef Cattle Implant Update
29	Stephany, Hormones in meat: different approaches in the EU and in the USA, APMIS 109, p. S 357 (2001)
30	Stephany, Hormones found in meat samples from regular controls within the European Union and from U.S. imports, Chemical Awareness 9, p. 13 (July 5, 2000)
31	Final Report of a Mission Carried Out in Germany from 07/05/01 to 18/05/01 in Order to Evaluate the Control of Residues in Live Animals and Animal Products (DG(SANCO)/3267/2001)
32	Final Report of a Mission Carried Out in Portugal from 17 to 21 November 2003 in Order to Evaluate the Control of Residues in Live Animals and Animal Products (DG(SANCO)/9209/2003)