

POINT BY POINT ANSWER TO REVIEWERS

REVIEWER 1

In this study the authors want to understand the shape of interaction matrices between hosts and their parasites. Two features are of importance, nestedness and modularity. These are measured in 32 matrices of quantitative pathogenicity trait data gathered from 15 plant-parasite pathosystems consisting of either annual or perennial plants along with fungi or oomycetes, bacteria, nematodes, insects and viruses. After assessing the performance of several nestedness and modularity algorithms by simulation (in SI Material), they find that significant modularity in only six of the 32 matrices, with two or three modules detected. Significant nestedness is found in 30 of the 32 matrices.

Interestingly, nestedness was linked to a parasite strain effect and a plant accession effect, with no parasite-plant interaction term.

Comment : We did not mean exactly that, since we did not formally test the significance of the plant-parasite interaction and did not estimate its magnitude. The sentence in lines 218-220 “an additive model combining pathogenicity QTLs in the parasites and resistance QTLs in the hosts, with no QTL x QTL interactions between hosts and parasites” may be misleading (and was slightly modified).

Rather, we meant that the plant-parasite interaction, if any, should explain a minority of the pathogenicity variance, since the plant and parasite effects explained together from ~50%~100% of the variance. Consequently, a model without the interaction term could fit well with the data. We now provide estimates of the plant-parasite interaction for 27 of the 32 matrices (see below).

I found the paper very interesting, very well written and easy to follow. I am convinced by the analyses, and as far as I can check, the statistical analyses are robust and well conducted. The interpretation of the results is cautious and takes into account the uncertainties and statistical limitations of the methods. I therefore highly recommend this study for PCI Evol Biol, and have only some minor points to improve the clarity of the paper, or indicating some missing information, and links to the theoretical literature as suggestions.

Introduction:

Three topics could be better explained:

1) It would be nice to link nestedness and modularity to the general models by Boots (Boots et al. 2014 Evolution for example). These models allow very general switch from quantitative to qualitative traits based on underlying trade-offs and simple assumptions on the interaction matrix.

Answer :

It is indeed interesting to link the quantitative models in Boots et al. (2014) with ours. Models in Boots et al.'s Fig. 1A and 1B are nested (1A being similar to our « additive QTL » model ; 1B being a quantitative variation on the gene-for-gene model), while the model in Fig. 1C is modular and is a quantitative variation on the « matching allele » model.

Boots et al. (2014) is now quoted in the Introduction and the similarity of our quantitative models with the model in Fig. 1A of Boots et al. (2014) is mentioned in the Discussion. Consequences of this model in terms of host-parasite coevolution are also mentioned in Discussion.

2) The role of epistasis between genes of interaction could be mentioned. I would expect epistasis to generate modularity (but that is a guess). In animals several papers by the group of Dieter Ebert do show some example of epistatic interactions of GxG interactions. In plants, one could imagine the same for multi-locus plant-parasite model (e.g. in a multilocus GFG model with quantitative value, i.e. not 0/1, see for example papers by Sasaki).

Answer :

Several papers by Ebert's group deal with intra-genome epistasis (e.g. Ameline et al. 2020 Mol Biol Evol), but not with epistasis between genomes of interacting hosts and parasites (*i.e.* specificity) (to our knowledge). As we have no clues about the potential effect of intra-genome epistasis on nestedness or modularity, we would prefer not to mention its potential role.

Epistasis between genes of interaction (between-genome epistasis) could probably have an impact on the structure of plant-parasite interaction matrices. We would expect between-genome epistasis to blur nested (and maybe also modular) patterns (but it's also just a guess and would probably depend on the type of epistasis). Sasaki's model (e.g. Sasaki 2000, Proc. R. Soc. Lond. B 267:2183-2188) is a multilocus GFG model for quantitative trait variation which is quite similar to Fenton's two-step infection model (Fenton et al. 2012) and incorporates a particular type of between-genome epistasis. These models are certainly well-adapted to plant-parasite interactions and, as derivatives of the classical GFG model, correspond to nested rather than to modular patterns. We cannot exclude that other types of epistasis could generate modular patterns. We added these two references (Sasaki 2000 and Fenton et al. 2012) to the Discussion and present them as suitable alternative models.

3) The link to non-host resistance and parasite effectors to overcome this resistance is also not clear, would we expect some modularity then?

Answer : Nonhost resistance is the capacity of (almost) all genotypes of a plant species to impede infection by a given parasite. Clearly, our study does not correspond to this context, since for 31 of the 32 matrices, large differences in resistance scope and strength are observed at the within-plant species. An uncertainty remains for matrix 26 for which several *Solanum* species are represented by a single genotype.

More generally, "nonhost resistance essentially relies on the very same genes and pathways as other types of plant immunity" (Panstruga and Moscou 2020 Mol. Plant-Microbe Interact.). Hence, we would expect the same nested or modular patterns as for other types of resistance.

- Line 97: Older citations would be more appropriate than Thrall et al. 2016 (for example papers by Gandon and/or Nuismer who have already made this explicitly). There is a nice paper by Dybdhal et

al. (2014 Am Nat) which is also very relevant. Some definitions used are found in Antonovics et al. (2012, Evolution) which present some points of the introduction in a general way for plant and parasite systems.

Answer : We mentioned Thrall et al. 2016 because they described all three models of host-parasite interaction and evolution (gene-for-gene, matching-allele and inverse matching-allele) whereas many others deal with one or two models only. We also quoted several of the oldest papers having described these models. There are many articles that studied these models, either experimentally or by modelling and those mentioned by the reviewer are some interesting ones.

Dybdahl et al. (2014 Am Nat) was added. It presents the diversity of models discussed in the article and advocates for developing models for quantitative host-parasite interactions, which is highly relevant here. The other articles are more specific and were not added.

Results and Methods:

- Line 173: It is unclear whether in all systems, the minimum and maximum of disease severity are comparable (as the scale is then set to be the same for all system between 0 and 9). Does this mean that all systems have reported the value 0 as no infection at all, and value 9 as maximum disease severity? This clarification is needed to understand how quantitative or qualitative are the disease severity measures in the different systems.

Answer :

The 0 values correspond to an absence (or almost absence) of infection, of symptoms or a lack of effect on plant health, except marginally for matrices 5, 20, 31 and 32.

For matrix 5, the highest mean relative latency period (RLP) is 131% of that observed on a susceptible plant genotype and corresponds to a zero score. Of course, it does not correspond to a lack of infection since in that case, the RLP would be infinite. However, it is not far from the highest RLP observed for another plant genotype discarded from our study because showing HR or immunity against some of the isolates (143% ; Table 3 in González et al. 2012)

For matrix 20, the minimum average value is 0.86 on a scale from 0 (no symptoms) to 5 (dead plant). A lower average value (close to 0.0) was observed in matrix 19 involving the same parasite but a different set of plants.

For matrices 31 and 32, the phenotype is the dry matter weight of infected plants relative to mock-inoculated plants of the same genotype. The 0 score corresponds to values >100% (maximum mean value was 145% both for matrix 31 and 32), corresponding to overcompensation of the infection, as observed previously for the same pathosystem (Montarry et al. 2012, J. Evol. Biol.).

The 9 values correspond (or are close) to the maximal pathogenicity values for almost all matrices, (except matrices 19, 20, 32, and possibly matrices 1-4 and 9) either because most of the matrices included a susceptible plant genotype control and/or because at least one of the plant-parasite pairs had a mean pathogenicity value close to the maximal possible value. For many matrices, mean pathogenicity values higher than for the susceptible plant genotype control were observed,

suggesting that the 0-9 interval is not far from the minimal-maximal pathogenicity interval possible (although « absolute susceptibility » probably does not exist).

Matrices 1 to 4 and 9 contain lesion size data and did not explicitly include a susceptible plant genotype control. It is therefore difficult to ascertain that the maximal susceptibility scores were observed.

For matrices 19 and 20, the highest mean disease scores were 4.1 and 3.0, respectively, compared to a maximal score of 5 (dead plant).

For matrix 32, the lowest mean relative dry matter weight was 13% of the mock-inoculated control (and not 0%, which would be the maximal pathogenicity).

In any case, for all matrices, a continuous phenotype distribution was observed (this was achieved after log transformation for matrices 17b and 22 that contained a large number of zero-valued cells as mentioned in the text) which suggests strongly that we analysed quantitative and not qualitative plant-parasite interactions.

We do not provide all these details in the text but we indicate in the Materials and Methods that the 0 to 9 scale globally corresponds (or is close) to the minimum to maximum value scale.

- Line 183, as well line 774-777: it would be nice to add some information on the null models (some names are given but the reader cannot assess what they mean without diving in the SI Material). It would be nice to indicate which null model test is the most stringent and which one is the most relaxed. A general description of the main null models and their assumptions (randomization procedure) could be included in the methods. The description is very good in the SI Material but lacks in the text. The description of some infection experiment could be reduced if these are published already to gain space for describing the null models.

Answer : We now provide information about the null models (and their constraint) in the Materials and Methods section. Note that it is not straightforward to rank the null models according to their constraints because there are several kinds of constraints (notably on the 0-valued cells of the matrix and on the row or column marginal sums) and a null model can be strongly constrained from one viewpoint and relaxed from the other viewpoint.

Description of experimental data was moved in Supplementary Methods 1 (except Table 1).

- Line 218: should it be “wine” instead of “wNODF”? otherwise this sentence is confusing.

Answer : No. *wNODF* shows limitations in terms of statistical power compared to *WINE* (means of 53% and 63% nestedness detection for values in Tables S6 and S5, respectively). As both methods share similar levels of type I error (15% and 16% mean false positives in Tables S1 and S2), we gave more importance to the results obtained with *WINE*.

Discussion:

- Line 441: The paper by Fenton, Antonovics and Brockhurst (Evolution 2012) also generates via a two-step infection process (similar to non-host resistance followed by infectivity/resistance) some interesting predictions. I guess that there modularity and/or nestedness generated by such model can be also predicted and applicable to plant systems?

Answer : Absolutely ! The reference was added in the Discussion and that model presented as an alternative for our data. Coevolution consequences of this, and other papers (Sasaki 2000 ; Boots et al. 2014) are added (briefly) in the Discussion. However, we did not provide details of these modelling approaches, because it is beyond the scope of this article, these approaches are more adapted to wild host-parasite systems than to crops and they have been reviewed elsewhere (Brown and Tellier 2011 ; which we added).

- I missed a discussion of the difference between annual/perennial and between different types of pathogens (Oomycetes, virus, bacteria, nematodes, ...) on why nestedness or modularity should be expected to be found. Is there a link between nestedness/modularity with life history traits such as obligate or facultative parasitism?

Answer : It is important to note that only one or a few pathosystems represent these different categories. For example, there was a single pathosystem involving a virus, a bacterium or an insect. In addition, the tendencies in nestedness and modularity were quite general (widespread nestedness and rare modularity). For example, 100% of pathosystems showed significant nestedness for at least one matrix. For these two reasons, it is difficult to derive any hypothesis from the experimental matrices analysed. It is also not possible to compare the nestedness and modularity scores in the different pathosystems because they are strongly influenced by the matrix dimensions.

Besides the results obtained with experimental matrices, it is also difficult to make general hypotheses. Naively, we could imagine that perennial plants have more durable immunity and therefore show little resistance/susceptibility specificity against a diversity of parasite strains. Hence, neither nestedness, nor modularity would be expected. The matrices corresponding to perennial plants (apricot and apple tree) look however similar to the others in terms of nested or modular structure.

We mention these points as interesting prospects in the Conclusion.

- What could be expectations for nestedness/modularity for wild plant systems versus crops? Could these make a nice set of predictions to be tested in other systems as conclusion of the paper?

Answer : That would be nice indeed. But again, it seems difficult to raise hypotheses with at least a minimal basis. Quantitative interactions are probably more frequent in natural systems compared to crop plants, where major-effect resistance genes have been primarily bred (Boots et al. 2014). So we could predict that similar matrix structures (frequent nestedness and rare modularity) would be frequently observed in wild plant-parasite systems. This comment was added in the Discussion.

REVIEWER 2

Reviewed by Rubén González, 2021-05-08 00:06

The manuscript entitled “The quasi-universality of nestedness in the structure of quantitative plant-parasite interactions” studies plant-pathogen interaction quantitative data by analyzing the structure of 32 matrices. The authors evaluated multiple algorithms, using them to analyze the structure and nestedness of the matrices. The findings point to a universal scarce modularity and high nestedness in the plant-parasite interactions. The strengths of the paper consist in the test of multiple algorithms, the use of a diverse set of plant-parasites, and the study of the structure of quantitative data. The authors have nicely described the methodology and datasets used, following a clear logic for their analysis. The examination of the biological significance of modularity and nestedness outcomes contributes to better understand the implications of the results. The analysis done uses a limited set of plant-parasites but, considering the scarce number of available data, the work makes relevant progress to indagate in the shared structures of plant parasite-interactions. This manuscript will be relevant for scientists working in the areas of plant disease, host-microbes interactions, and evolution.

Comments:

1. In the discussion section there is no comment on the suitability of the algorithms used. The wNODF and WINE algorithm are discussed in the results sections. In the introduction it is said that one of the two goals of the work is "to assess the performance of available algorithms" (L152-153), but in the conclusion section there is no statement about the best suited algorithms.

Answer : As proposed also by reviewer 3, we introduced additional results about the performance of nestedness and modularity algorithms in Results and clarified the links between the « algorithm performance analysis » and the interpretation of the results obtained with the experimental matrices. We also present briefly the null models in the Materials and Methods section.

2. L264-265 The main text misses one reference when citing the algorithms: for label prop algorithm (Raghavan et al., 2007).

Answer : As we could not evaluate the performance of the walktrap and label prop (described by Raghavan et al., 2007) algorithms, we did not apply them to the experimental matrices.

3. L366-367 It is said that from the 32 matrices only two of them are not nested. The text speculates that the one of the two non-nested matrices could be nested but its little size may be hindering the nested pattern. As the size of the matrices were decided before the analysis, the results should be trusted.

Answer : As this explanation (small size matrix) was not tested further, we preferred to delete this interpretation.

4. L510-511 Datasets were selected under the criteria of having at least 6 plant accessions and 6 parasite strains. Is this minimum number arbitrary or was it required for the application of the algorithms?

Answer : This number was set arbitrarily as it is unlikely to detect nested and even more modular patterns in small-size matrices.

5. In Figure 2 all matrices are shown and the significant nested ones are numbered in red. Some identification for the matrix that showed modularity for infection or resistance scores would be appreciated.

Answer : Done.

6. For the Figure 3 it is stated that coefficient of correlation could not be calculated in some cases (L254-257). How many times did this happen? The figure is nice but I could not find the raw data in order to observe the coefficient value for each matrix depending on the threshold.

Answer : All coefficients of correlation are now provided in Table S22.

REVIEWER 3

In the manuscript « The quasi-universality of nestedness in the structure of quantitative plant-parasite interactions”, Moury and coauthors present an analysis of a compilation of plant-parasite interaction matrices and establish that the majority of them have a nested structure.

This is a timely and interesting manuscript which definitely deserves to be published. However, the production of this manuscript has involved a very high volume of work and the density of the information provided makes it a bit difficult to read and to follow. I suggest below ways of reorganising the manuscript and being more synthetic that should help having a better flow. I also ask some scientific and technical questions that should be answered in the manuscript.

1. As explicitly said at the end of the introduction, this paper has two goals: “(i) to assess the performance of available algorithms to identify nested and modular patterns in matrices of quantitative data and (ii) to determine if these patterns are specific to each pathosystem or show a general trend”. This paper is thus a combination of two papers in a sense, one with a methodological question and the other with a scientific question. The choice to write a single paper is one of the reasons why this manuscript is so information-rich. All the material and methods and results corresponding to the first goal are presented in the Supplementary material but discussed in the main discussion (1392-418), whereas everything related to goal 2 (including a very long material description) is in the main text. I would suggest to balance and synthesize a bit more the presentation of the two goals, to have at least an outline of the Materials and Methods and Results of the methodological comparison of the different algorithm to test for nestedness and modularity in the main text and to shorten the material section corresponding to the second goal (see next point).

Answer : This has been done (see details below).

2. The collection of plant-parasite quantitative interaction matrices analysed in this paper is composed of a mix of published and unpublished data. This results in a long Material section because for unpublished studies, technical details about the obtention of the matrix had to be given. I suggest to leave in the main text the information which is given for all matrices (number of plant accession, number of pathogen isolates, information on resistance/virulence QTL if available and pathogenicity variable(s) measured and given in the matrices) and move the technical details on how the matrix were obtained for unpublished studies to the supplemental material.

Answer : the pathosystem information available for all matrices is left in Table 1 but the technical details moved in Supplementary Methods S1. The relevant resistance QTL data (no virulence QTL data is available, for any matrix) is mentioned in the Results section.

3. The criterion to build the dataset are not clearly stated: a criterion of minimal size of the matrix is given (1510-511) but there are other published datasets that would satisfy this criterion and are not included. How were the studies to include chosen? Did the authors perform a systematic literature search? Which key words were used?

Answer : We could not find an efficient combination of keywords to perform a systematic literature search. Notably, plant-parasite quantitative studies almost never use the words « matrix », « network » or « graph », which could be due to the fact that network-based analyses were only very recently applied to host-parasite interactions, and still rarely to plant-parasite interactions. Datasets were identified by knowledge of the authors. We probably missed some of them, but we did not exclude any of them on purpose.

We are presently not aware of published matrices satisfying all our criteria : quantitative phenotypes (not qualitative phenotypic data, or mixture of qualitative and quantitative data), availability of phenotypic data for all plant-parasite pairs (no missing data), at least 6 x 6 dimension, intra-specific set of parasite strains and, preferentially, intra-specific set of plant genotypes (or closely-related plant species).

4. The results of the methodological part (best algorithms and null models in terms of type I and type II error) are taken into account in a blurry way in the analysis of the dataset: the results are presented in the tables for all algorithms and null models (even the ones shown to perform poorly) but then in the written part of the results more weight is given to the results provided by the “good” combinations of algorithm and null model and a unique final number is given. This integration of the results of goal 1 in the analyses for goal 2 needs to be clarified.

Answer :

We have added information on the performance of the algorithms in the Results section. To make it clearer, we focused on the most efficient algorithm in the main text and moved the results of the other algorithms in Supplementary Tables S19 and S20. Another option could be to delete also Supplementary Tables S19 and S20, but we think it is important to give the reader the opportunity to check that even less efficient algorithms do not contradict the most important results obtained with the most efficient ones.

For nestedness, we presented only WINE, which outperforms wNODF in terms of power. In any case, results of both methods are highly consistent, given the fact the wNODF performs poorly when 0-valued cells are rare in the matrix. These explanations were deleted for simplicity and clarity.

For modularity, we presented only spinglass, which outperforms the others in terms of type-I error rate. It should be noted that the other algorithms did not detect additional modularity consistently.

5. The main result of the study is that nestedness is very common in plant-parasite interactions and that statistical interaction between plant accession and parasite isolate is usually not significant (result section + I425-427, I445-448). The authors derive this second conclusion from the fact that statistical models with only the two individual factors (without the interaction between them) have a high R². The author should test directly whether the interaction is statistically significant or not; a model with single effects with an R² of 0.5 leaves “space” for a significant interaction.

Answer : We fully agree with this comment.

Again, we did not mean that the plant-parasite interaction was not significant, since we did not formally test its significance and did not estimate its magnitude, but that the plant-parasite interaction, if any, should explain a minority of the pathogenicity variance.

We had not performed the interaction analysis because it was not possible for all matrices. Indeed, to disentangle the interaction effect from the uncontrolled environmental and experimental variance, several independent phenotypic values should be available for each plant-parasite genotype pair. Such values are not available for all published data and for some of our experimental data. Consequently, for consistency between all matrices, we decided to not include such analyses.

However, we recognise the importance of evaluating the plant-parasite interaction, when feasible, in the context of this manuscript. We could perform that analysis for 26 of the 32 matrices and Soltis et al. (2019) provide it for a 27th (matrix 9). In Supplementary Table S21, we provide the estimates of the parts of variance explained by the plant effect, the parasite effect and the plant × parasite interaction effect. We used the ω^2 estimates (Kirk 1982) which are unbiased compared to classical R^2 . We took this opportunity to provide also the ω^2 estimates instead of R^2 in Table 1 for the model that does not include the interaction term.

The results confirm our previous statement that plant-parasite interactions explained a minority of the phenotypic variance. In the 24 nested matrices among the 26 analyzed, the plant-parasite interaction term explained on average 17.5% (minimum 0% ; maximum 41%) of the phenotypic variance, relatively to the total phenotypic variance that could be collectively explained by the plant, the parasite and the plant-parasite effects.

Moreover, for matrix 9, Soltis et al. (2019) indicate a nonsignificant interaction effect between pathogen isolate and plant genotype. Concerning the 5 remaining matrices for which no estimate of the plant-parasite interaction could be obtained (matrices 5, 7, 10, 11 and 16), it should be noted that the plant and parasite effects explained >80% of the phenotypic variance for 4 of them (Table 1) and a lower proportion of phenotypic variance for the last one.

These analyses were included in the Results section and in Supplementary Table S21.

6. For many pathosystems, there is more than one matrix in the dataset. It is not always clear whether the different matrices have been obtained by measuring different traits after the same inoculation events or whether they result from different inoculation experiments and in this second case, whether the plant accessions and the parasite isolates used are identical for all experiments/matrices.

Answer : Pairs of matrices (1,3), (2,4), (12, 13), (14, 15) and (21, 22) and triplets (27, 29, 31) and (28, 30, 32) result from the same inoculation experiment.

All other matrices result from independent experiments, usually with different sets of plant and parasite genotypes, except matrices 27 to 32 that include almost the same genotypes.

This information is now included in Supplementary Methods S1.

7. In the cases where several matrices have been obtained on the same pathosystem, it would be great to have comments on the similarity of the results between the different matrices (as for example in I330-350).

Answer : For some pathosystems, results varied depending on the matrix.

For nestedness, matrix 21 was not significantly nested whereas matrix 22 is nested. Similarly, among matrices 27 to 32, only matrix 32 is not significantly nested.

For modularity, matrices 6 and 7 are significantly modular, whereas matrix 8 is not. Matrices 10 and 11 are both modular. Matrix 14 is significantly modular whereas matrices 12, 13 and 15 are not. Matrix 17 is significantly modular whereas matrix 16 is not.

Finally, for inverted matrices, matrices 10 and 11 are both modular. Also, matrices 14 and 15 are modular whereas matrices 12 and 13 are not.

These differences could be due to the specific pathogenicity trait that vary between matrices and/or the statistical power to detect the nested or modular structures in these matrices.

As we could not find more clues about these discrepancies, we just mentioned this fact in the end of the Results section.

8. L65-74: this paragraph of definitions does not seem necessary; the two meanings of “interaction” are well-known to readers of this kind of papers.

Answer :

In fact, we added this paragraph lately because it appeared that it was not so clear for several colleagues in the field of phytopathology or plant genetics. We agree that it may be obvious to many others. We would rather keep it to make it explicit for the largest audience.

9. L96-133: the link between models of interaction and the modularity/nestedness has been discussed in other papers (de Vienne et al. 2009, Gallet et al. 2016). And this section of the introduction could be more synthetic.

Answer : The reference by Gallet et al. 2016 was added and is indeed a nice illustration of that link. de Vienne et al. (2009) does not explicitly analyze the modularity or nestedness of interaction matrices, so its links with the present study is more distant and it was not added.

We also reduced the mentioned section.

10. L375-391: this paragraph would be better in the introduction.

Answer : We moved it accordingly.

11. L562: what do the authors mean by “differential lines”?

Answer : The word « differential » is unnecessary (it meant that the melon lines exhibited contrasted resistance levels against the *P. xanthii* isolates).

12. Supp mat, l39-40: explain why you could not apply certain algorithms to your data.

Answer :

In fact, we applied them but we could not use the results.

Most of the time, the *walktrap* and *label prop* algorithms provided modularity scores of 0 for both the matrix analyzed (experimental data or simulated matrices) and the corresponding null-model matrices. Hence, no p-values can be provided and the analysis is useless to assess the significance of modularity.

In addition, as mentioned in the text, we could not evaluate the performance of the *leading eigenvector* algorithm because it is an iterative algorithm that sometimes does not converge towards a modularity estimate. It did not converge for many test matrices, as well as for the actual matrix 12 and for many null model matrices corresponding to matrix 12.

The sentences in Suppl. Methods 2 were slightly modified to make it clearer.

Minor comments:

- L91: to be rephrased

Answer : Done.

- L546 and 551: apple tree accessions.

Answer : Done.

- L758: similar results as

Answer : Done.

- Supp mat l10: weighted version of

Answer : Done.

- Supp mat l94: “or” has to be replaced by “of”.

Answer : Done.

Additional modifications :

In Table 1, corrections have been made in the « Matrix size » column (numbers of hosts and parasites in matrices 28, 29 and 31 had been inverted).

Raw data : Corrections were made to some matrices (0-valued cells had been erroneously duplicated).