Supplementary Information Available

Supplementary Table 1: The versions of databases used in the present study.

Database	**Release Version or Date**
Biogrid	01 May 2011, Version 3.1.75
CORUM	02 Sept 2009, Release Sept 2009
DIP	10 Oct 2010
HPRD	13 Apr 2010, Release 9
IntAct	21 Apr 2011, Release 141
HOMOMINT	07 Mar 2011, 2009 Update
MIPS	2008
Reactome	15 Mar, Version 36

Supplementary Table 2: The number of interactions (column 4) and nodes (column 5) used in the present study

Database	**Number of Interactions with UniprotKB/HGNC ids**	**Number of Nodes in the Original Network**	**Number of Interactions after Substitution with ENSEMBL Gene ids**	**Number of Nodes after Substitution with ENSEMBL Gene ids**
Biogrid	36004	8787	35548	8679
CORUM	36056	2524	35634	2497
DIP	12550	4756	12148	4565
HPRD*	39042	9607	37929	9301
IntAct	30801	7851	28848	7765
MIPS	280	356	276	353
HOMOMINT	20853	7383	18578	6485
Reactome	12146	2768	11943	2667

* Only in the case of HPRD, the original network was extracted with gene names (HGNC ids). These were then substituted with ENSEMBL Gene ids.

Supplementary Table 3: The number of interactions (column 4) and nodes (column 5) of RN, MM, DM and CE used in the present study

Organism	**No. of Interactions with UniprotKB/Refseq ids**	**Number of Nodes in the Original Network**	**Number of Interactions after Substitution with ENSEMBL Gene ids**	**Number of Nodes after Substitution with ENSEMBL Gene ids**
Rattus norvegius	9117	3136	1808	1046
Mus musculus	28115	8058	6011	3073
Drosophilla melanogaster	144830	10493	5837	1501
Caenorhabditis elegans	40597	5704	2456	1113

Figure S1A. Splice Variant count in hub versus non-hub genes of the HPRD PPI Network. Genes/nodes with top 10% of degree values were chosen as hubs. The hubs (5958 isoforms in total and ~6 per gene) are characterized by the presence of large number of isoforms as compared to non-hubs (40221 isoforms in total and ~5 per gene) (Wilcoxon ranksum test P<2.2e-16)

Figure S1B. Splice Variant count in hub versus non-hub genes of the HPRD PPI Network. Genes/nodes with top 15% of degree values were chosen as hubs .The hubs (8741 isoforms in total and ~6 per gene) are characterized by the presence of large number of isoforms as compared to non-hubs (37438 in total and ~5 per gene) (Wilcoxon ranksum test P<2.2e-16)

Figure S1C. Splice Variant count in hub versus non-hub genes of the HPRD PPI Network. Genes/nodes with top 20% of degree values were chosen as hubs .The hubs (11434 isoforms in total and ~6 per gene) are characterized by the presence of large number of isoforms as compared to non-hubs (34745 isoforms in total and ~5 per gene) (Wilcoxon ranksum test P<2.2e-16)

Figure S1D. Splice Variant count in hub versus non-hub genes of the BioGrid PPI Network. The hubs (2827 isoforms in total and ~6 per gene) are characterized by the presence of large number of isoforms as compared to non-hubs (40771 isoforms in total and ~5 per gene) (Wilcoxon ranksum test P= 3.187e-9)

Figure S1E. Splice Variant count in hub versus non-hub genes of the CORUM PPI Network. The non-hubs (12404 isoforms in total and ~5 per gene) seem to have greater number of splice variants than the hubs (681 isoforms in total and ~5 per gene) (Wilcoxon ranksum test P=0.9743)

Figure S1F. Splice Variant count in hub versus non-hub genes of the DIP PPI Network. The hubs (1321 isoforms in total and ~6 per gene) and non-hubs (22906 isoforms in total and ~5 per gene) have no significant difference in their splice variant counts (Wilcoxon ranksum test P=0.05258)

Figure S1G. Splice Variant count in hub versus non-hub genes of the IntAct PPI Network. The hubs (2534 isoforms in total and ~6 per gene) are characterized by the presence of large number of isoforms as compared to non-hubs (37329 isoforms in total and ~5 per gene) (Wilcoxon ranksum test P=9.71e-06)

Figure S1H. Splice Variant count in hub versus non-hub genes of the MIPS PPI Network. The hubs (110 isoforms in total and ~6 per gene) and non-hubs (2059 isoforms in total and ~6 per gene) have no significant difference in their splice variant count (Wilcoxon ranksum test P=0.1259)

Figure S1I. Splice Variant count in hub versus non-hub genes HOMOMINT PPI Network. The hubs are characterized by the presence of large number of isoforms (20699 isoforms in total and ~6 per gene) as compared to non-hubs (31225 isoforms in total and ~5 per gene) (Wilcoxon ranksum test P=0.0001568)

Figure S1J. Splice Variant count in hub versus non-hub genes Reactome PPI Network. The hubs are characterized by the presence of large number of isoforms (679 isoforms in total and 5 per gene) as compared to non-hubs (11045 isoforms in total and ~4 per gene) (Wilcoxon ranksum test P=0.0003584)

Figure S1K. Splice Variant count in hubs and non-hubs of the BioGrid PPI Network. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. Hubs are characterized by the presence of large number of isoforms as compared to non-hubs (Wilcoxon ranksum test P=0.0009006)

Figure S1L. Splice Variant count in hubs and non-hubs of the CORUM PPI Network. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. Hubs have lesser number of splice variants when compared to non-hubs (Wilcoxon ranksum test P=0.07994)

Figure S1M. Splice Variant count in hubs and non-hubs of the DIP PPI Network. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. The hubs and non-hubs have no significant difference in their splice variant count (Wilcoxon ranksum test P=0.4684)

Figure S1N. Splice Variant count in hubs and non-hubs of the IntAct PPI Network. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. Hubs are characterized by the presence of large number of isoforms as compared to non-hubs (Wilcoxon ranksum test P=2.509e-05)

Figure S1O. Splice Variant count in hubs and non-hubs of the HOMOMINT PPI Network. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. Hubs are characterized by the presence of large number of isoforms as compared to non-hubs. (Wilcoxon ranksum test P=0.0005369)

Figure S1P. Splice Variant count in hubs and non-hubs of the MIPS PPI Network. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. The hubs and non-hubs have no significant difference in their splice variant count (Wilcoxon ranksum test P=0.05592)

Figure S1Q. Splice Variant count in hubs and non-hubs of the Reactome PPI Network. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. Hubs are characterized by the presence of large number of isoforms as compared to non-hubs (Wilcoxon ranksum test P=0.01523)

Figure S1R. Splice Variant count in hub versus non-hub genes Worm (*Caenorhabditis elegans*) PPI Network. The hubs (154 isoforms in total and ~ 2 per gene) and non-hubs (2293 isoforms in total and ~ 2 per gene) have no significant difference in their splice variant count (Wilcoxon ranksum test P=0.4838)

Figure S1S. Splice Variant count in hub versus non-hub genes Fly (*Drosophila melanogaster*) PPI Network. The hubs are characterized by the presence of large number of isoforms (250 isoforms in total and ~3 per gene) as compared to non-hubs (2978 isoforms in total and ~ 2 per gene) (Wilcoxon ranksum test P=0.001002)

Figure S1T. Splice Variant count in hub versus non-hub genes Rat (*Rattus norvegicus*) PPI Network. The hubs are characterized by the presence of large number of isoforms (143 isoforms in total and ~ 3 per gene) as compared to non-hubs (1903 isoforms in total and ~ 2 per gene) (Wilcoxon ranksum test P=0.002384)

Figure S2. Distribution of splice variant count among all the human genes **(a)**, hub genes (442 in number) **(b)** and non-hub genes (8237 in number) **(c)** in the BioGrid PPI Network. As can be seen, hubs show a different distribution of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P= 0.0002847) whereas non-hubs show a distribution very similar to all the genes (Kolmogorov-Smirnov Test P=0.9995). The splice variant count distributions between hubs and non-hubs were significantly different (Kolmogorov-Smirnov Test P=0.0001101).

Figure S3. Distribution of splice variant count among all the human genes **(a)**, hub genes (151 in number) **(b)** and non-hub genes (2347 in number) **(c)** in the CORUM PPI Network. As can be seen, in this case hubs as well as non-hubs do not show significantly different distributions of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P values are 0.4545 and 1.0 respectively). The splice variant count distributions between hubs and non-hubs were not significantly different (Kolmogorov-Smirnov Test P=0.3801).

Figure S4. Distribution of splice variant count among all the human genes **(a)**, hub genes (230 in number) **(b)** and non-hub genes (4335 in number) **(c)** in the DIP PPI Network. As can be seen, in this case hubs as well as non-hubs do not show significantly different

distributions of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P values are 0.4395 and 1.0 respectively). The splice variant count distributions between hubs and non-hubs were not significantly different (Kolmogorov-Smirnov Test P=0.3762).

Figure S5. Distribution of splice variant count among all the human genes **(a)**, hub genes (410 in number) **(b)** and non-hub genes (7355 in number) **(c)** in the IntAct PPI Network. As can be seen, hubs show a different distribution of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P=0.004171) whereas non-hubs show a distribution very similar to all the genes (Kolmogorov-Smirnov Test P=1.0). The splice variant count distributions between hubs and non-hubs were significantly different (Kolmogorov-Smirnov Test P=0.002096)

Figure S6. Distribution of splice variant count among all the human genes **(a)**, hub genes (17 in number) **(b)** and non-hub genes (336 in number) **(c)** in the MIPS PPI Network. As can be seen, in this case hubs as well as non-hubs do not show significantly different distributions of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P values are 0.7576 and 1 respectively). The splice variant count distributions between hubs and non-hubs were not significantly different (Kolmogorov-Smirnov Test P=0.703)

Figure S7. Distribution of splice variant count among all the human genes **(a)**, hub genes (344 in number) **(b)** and non-hub genes (6142 in number) **(c)** in the HOMOMINT PPI Network. As can be seen, hubs show a different distribution of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P=0.01194) whereas non-hubs show a distribution very similar to all the genes

(Kolmogorov-Smirnov Test P=1.0). The splice variant count distributions between hubs and non-hubs were significantly different (Kolmogorov-Smirnov Test P=0.006729)

Figure S8. Distribution of splice variant count among all the human genes **(a)**, hub genes (136 in number) **(b)** and non-hub genes (2531 in number) **(c)** in the Reactome PPI Network. As can be seen, hubs show a different distribution of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P=0.001328) whereas non-hubs show a distribution very similar to all the genes (Kolmogorov-Smirnov Test P=1.0). The splice variant count distributions between hubs and non-hubs were significantly different (Kolmogorov-Smirnov Test P=0.0006047)

Figure S9. Distribution of splice variant count among all the genes **(a)**, hub genes (62 in number) **(b)** and non-hub genes (1051 in number) **(c)** in the Worm(*Caenorhabditis elegans*) PPI Network. As can be seen, in this case hubs as well as non-hubs do not show significantly different distributions of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P values are 0.1540 and 0.7386 respectively). The splice variant count distributions between hubs and non-hubs were not significantly different (Kolmogorov-Smirnov Test P=0.7017)

Figure S10. Distribution of splice variant count among all the genes **(a)**, hub genes (79 in number) **(b)** and non-hub genes (1422 in number) **(c)** in the Fly (*Drosophila melanogaster*) PPI Network. As can be seen, hubs show a different distribution of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P=0.01564) whereas non-hubs show a distribution very similar to all the

genes (Kolmogorov-Smirnov Test P=0.8102). The splice variant count distributions between hubs and non-hubs were not significantly different (Kolmogorov-Smirnov Test P=0.05209)

Figure S11. Distribution of splice variant count among all the mouse (*Mus Musculus*) genes **(a)**, hub genes (156 in number) **(b)** and non-hub genes(2919 in number) **(c)** present in PPI Network. As can be seen, hubs show a different distribution of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P=0.000696) whereas non-hubs show a distribution very similar to all the genes (Kolmogorov-Smirnov Test P=0.7666). The splice variant count distributions between hubs and non-hubs were significantly different (Kolmogorov-Smirnov Test P=0.001387)

Figure S12. Distribution of splice variant count among all the genes **(a)**, hub genes (57 in number) **(b)** and non-hub genes (989 in number) **(c)** in the Rat (*Rattus norvegicus*) PPI Network. As can be seen, hubs show a different distribution of splice variant count as compared to all the genes (Kolmogorov-Smirnov Test P=0.01465) whereas non-hubs show a distribution very similar to all the genes (Kolmogorov-Smirnov Test P=0.412). The splice variant count distributions between hubs and non-hubs were significantly different (Kolmogorov-Smirnov Test P=0.02060)

Figure S13 A. Splice variant count in hub and non-hub orthologous nodes Human (*Homo sapiens*) and Worm (*Caenorhabditis elegans*). Boxes shaded in dark-gray represent hubs and the ones in light-gray represent non-hubs. HS_Hubs and HS_Nonhubs represent the Hubs and Non-hubs of *Homo sapiens* respectively. CE_Hubs and CE_Nonhubs represent the Hubs and Non-hubs of *Caenorhabditis elegans*

respectively. Kruskal-Wallis Test P between all the groups is < 2.2e-16 and Wilcoxon Ranksum Test P value between CE_hubs and CE_Nonhubs is 0.4297

Figure S13 B. Splice variant count in hub and non-hub orthologous nodes Human (*Homo sapiens*) and Fly (*Drosophila melanogaster*). Boxes shaded in dark-gray represent hubs and the ones in light-gray represent non-hubs. HS_Hubs and HS_Nonhubs represent the Hubs and Non-hubs of *Homo sapiens* respectively. DM_Hubs and DM_Nonhubs represent the Hubs and Non-hubs of *Drosophila melanogaster* respectively. Kruskal-Wallis Test P between all the groups is < 2.2e-16 and Wilcoxon Ranksum Test P value between DM_hubs and DM_Nonhubs is 0.06632.

Figure S13 C. Splice variant count in hub and non-hub orthologous nodes Human (*Homo sapiens*) and Rat (*Rattus norvegicus*). Boxes shaded in dark-gray represent hubs and the ones in light-gray represent non-hubs. HS_Hubs and HS_Nonhubs represent the Hubs and Non-hubs of *Homo sapiens* respectively. RN_Hubs and RN_Nonhubs represent the Hubs and Non-hubs of *Rattus Norvegicus* respectively. Kruskal-Wallis Test P between all the groups is < 2.2e-16 and Wilcoxon Ranksum Test P value between RN_hubs and RN_Nonhubs is 0.2472.

Figure 14 A. Splice variant count in hub and non-hub orthologous nodes of Human (*Homo sapiens*) and Mouse (*Mus musculus*) obtained after the alignment of two networks. Boxes shaded in dark-gray represent hubs and the ones in light-gray represent non-hubs. HS_Hubs and HS_Nonhubs represent the Hubs and Non-hubs of *Homo sapiens* respectively. MM_Hubs and MM_Nonhubs represent

the Hubs and Non-hubs of *Mus musculus* respectively. Kruskal-Wallis Test P between all the groups is <2.2e-16 and Wilcoxon Ranksum Test P value between MM_hubs and MM_Nonhubs is 0.001042

Figure S14 B. Splice variant count in hub and non-hub orthologous nodes of Human (*Homo sapiens*) and Rat (*Rattus norvegicus*) obtained after the alignment of two networks. Boxes shaded in dark-gray represent hubs and the ones in light-gray represent non-hubs. HS_Hubs and HS_Nonhubs represent the Hubs and Non-hubs of *Homo sapiens* respectively. RN_Hubs and RN_Nonhubs represent the Hubs and Non-hubs of *Rattus norvegicus* respectively. Wilcoxon Ranksum Test P value between RN_hubs and RN_Nonhubs is 0.6151 though Kruskal-Wallis Test P for all the groups is 6.143e-07

Figure S15 A. Average disorderedness in genes with high and low splice variant count. A: Genes with top 10% of the splice variant count (1140 genes), and B: Genes with bottom 90% of the splice variant count (8161 genes) (Wilcoxon Ranksum test P =8.267e-13)

Figure S15 B. Average disorderedness in genes with high and low splice variant count. A: Genes with top 15% of the splice variant count (1464 genes), and B: Genes with bottom 85% of the splice variant count (7837 genes) (Wilcoxon Ranksum test P =2.411e-12)

Figure S16A. Variation of Node Degree as a function of average disorderedness and splice variant count. Numbers from 1-4 on x-axis represent nodes/genes of different groups: 1) nodes/genes with high average disorderedness and high splice variant count, 2) nodes/genes with high average disorderedness and low splice variant count, 3) nodes/genes with low average disorderedness and high splice variant count and 4) nodes/genes with low average disorderedness and low splice variant count. Nodes/genes with average disorderedness

content of more than 20% were considered to be highly disordered. Nodes with average disordered content of less than 20% were considered to be structured. Nodes/genes with top 5% of splice variant count were considered as "high splice variant count nodes". Kruskal-Wallis Test P between all the groups is < 2.2e-16. Wilcoxon Ranksum Test P value between groups 1 and 4 is < 2.2e-16. While Wilcoxon Ranksum Test P value between groups 1 and 2 is 5.85e-12 and between groups 3 and 4 is 0.002414

Figure S16B. Variation of Node Degree as a function of average disorderedness and splice variant count. Numbers from 1-4 on x-axis represent nodes/genes of different groups: 1) nodes/genes with high average disorderedness and high splice variant count, 2) nodes/genes with high average disorderedness and low splice variant count, 3) nodes/genes with low average disorderedness and high splice variant count and 4) nodes/genes with low average disorderedness and low splice variant count. Nodes/genes with average disorderedness content of more than 30% were considered to be highly disordered. Nodes with average disordered content of less than 30% were considered to be structured. Nodes/genes with top 5% of splice variant count were considered as "high splice variant count nodes". Kruskal-Wallis Test P between all the groups is < 2.2e-16. Wilcoxon Ranksum Test P value between groups 1 and 4 is < 2.2e-16. While Wilcoxon Ranksum Test P value between groups 1 and 2 is 8.39e-11 and between groups 3 and 4 is 4.087e-05.

Figure S16C. Variation of Node Degree as a function of average disorderedness and splice variant count. Numbers from 1-4 on x-axis represent nodes/genes of different groups: 1) nodes/genes with high average disorderedness and high splice variant count, 2) nodes/genes with high average disorderedness and low splice variant count, 3) nodes/genes with low average disorderedness and high splice variant

count and 4) nodes/genes with low average disorderedness and low splice variant count. Nodes/genes with average disorderedness content of more than 30% were considered to be highly disordered. Nodes with average disordered content of less than 30% were. considered to be structured. Nodes/genes with top 10% of splice variant count were considered as "high splice variant count nodes". Kruskal-Wallis Test P between all the groups is < 2.2e-16. Wilcoxon Ranksum Test P value between groups 1 and 4 is < 2.2e-16. While Wilcoxon Ranksum Test P value between groups 1 and 2 is 9.755e-15 and between groups 3 and 4 is 1.232e-06.

Figure S16D. Variation of Node Degree as a function of average disorderedness and splice variant count. Only those Splice Variants (proteins) were used for calculation whose existence was experimentally validated. Presence of the proteins in the PRIDE database was considered as a proxy for experimental validation. Numbers from 1-4 on x-axis represent nodes/genes of different groups: 1) nodes/genes with high average disorderedness and high splice variant count, 2) nodes/genes with high average disorderedness and low splice variant count, 3) nodes/genes with low average disorderedness and high splice variant count and 4) nodes/genes with low average disorderedness and high splice variant count. . Nodes/genes with average disorderedness content of more than 30% were considered to be highly disordered. Nodes with average disordered content of less than 30% were considered to be structured. Nodes/genes with top 5% of splice variant count were considered as "high splice variant count nodes". Kruskal-Wallis Test P between all the groups is < 2.2e-16. Wilcoxon Ranksum Test P value between groups 1 and 4 is < 2.2e-16. While Wilcoxon Ranksum Test P value between groups 1 and 2 is < 2.2e-16 and between groups 3 and 4 is < 2.2e-16.

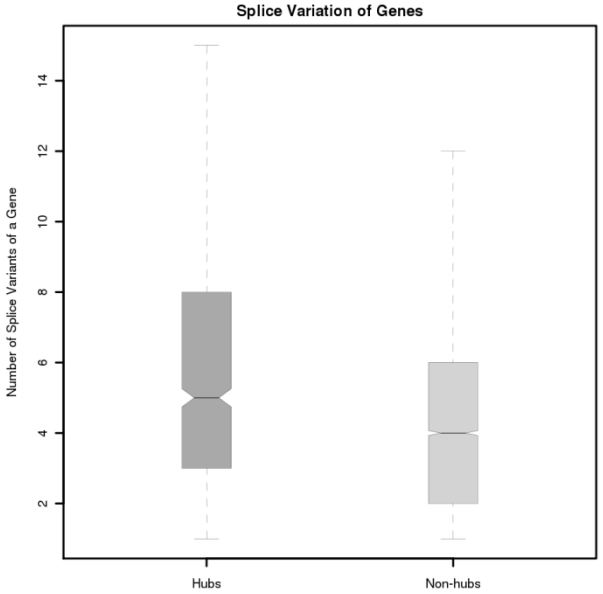
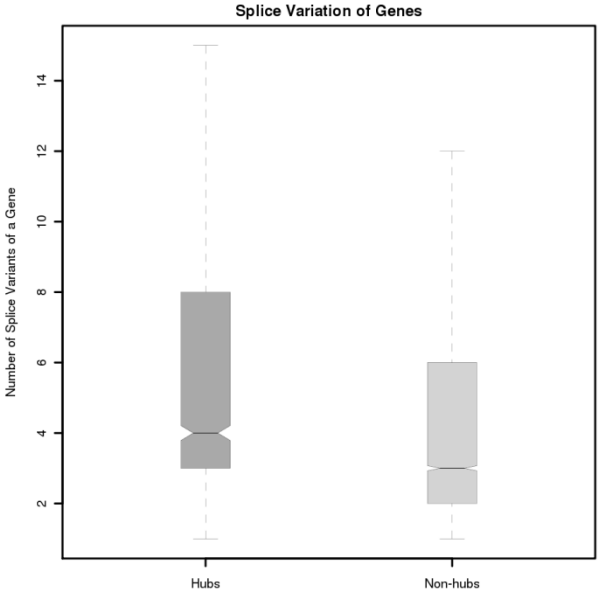


Figure S1A



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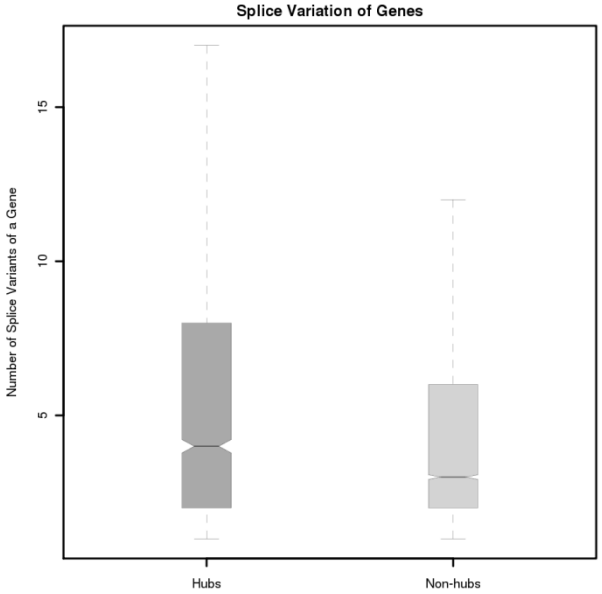

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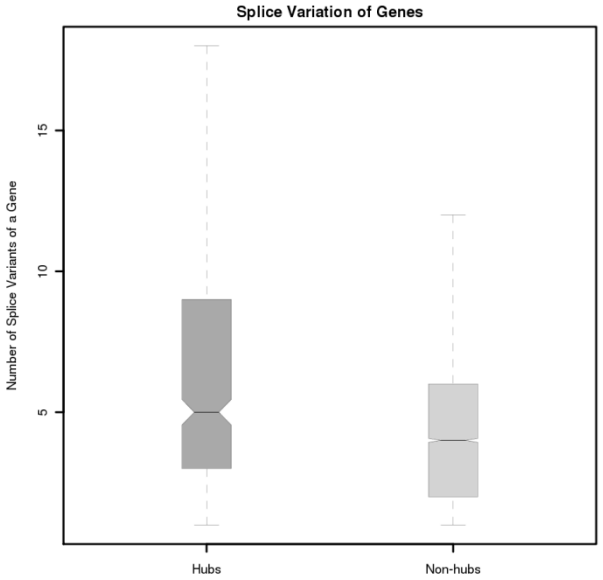


Figure S1D

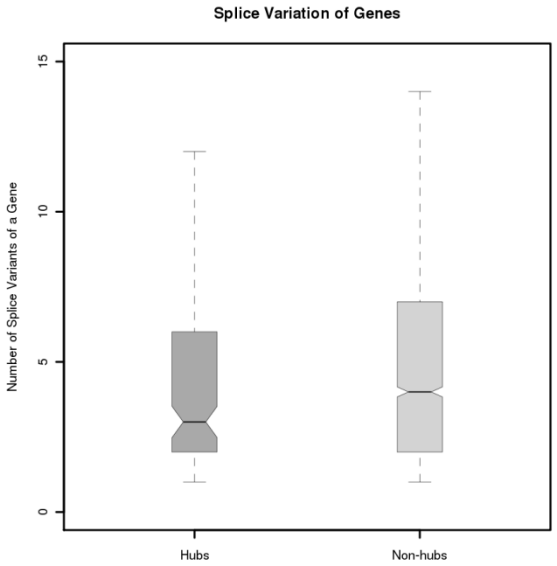
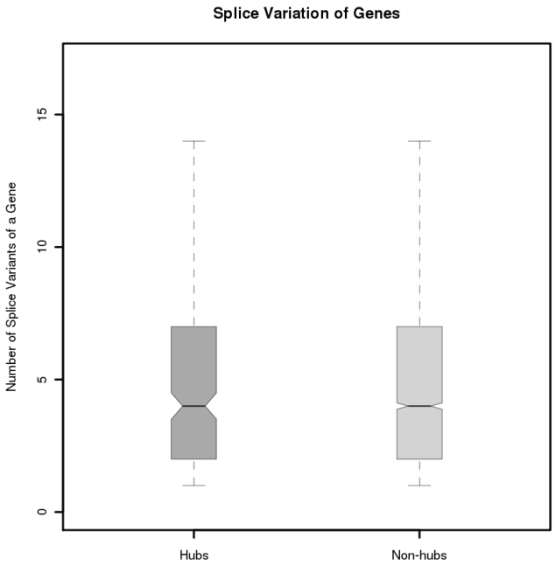


Figure S1E



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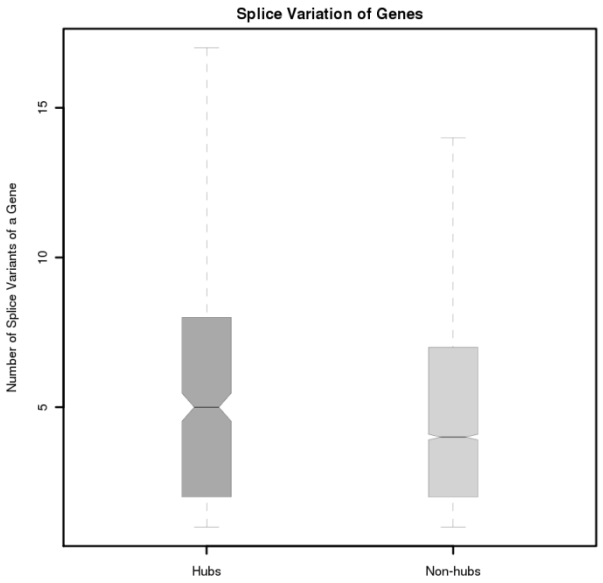


Figure S1G

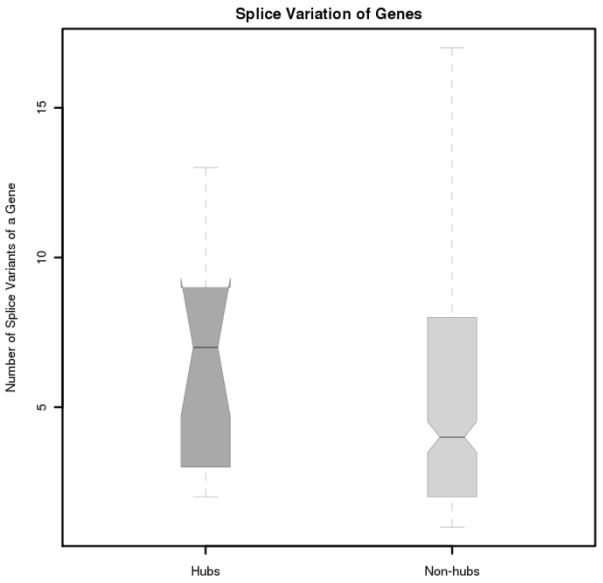


Figure S1H

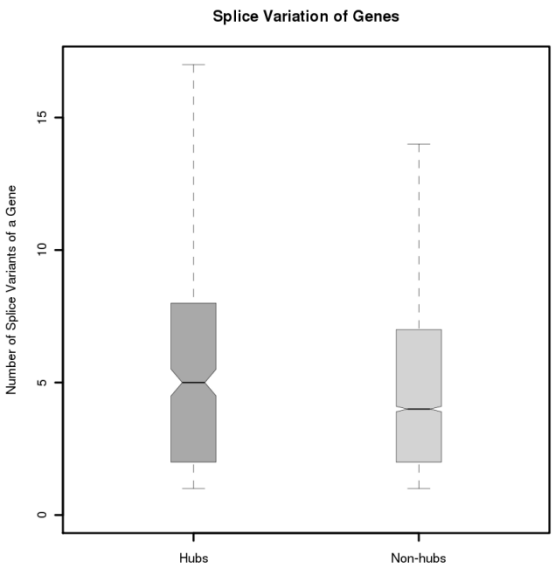


Figure S1I

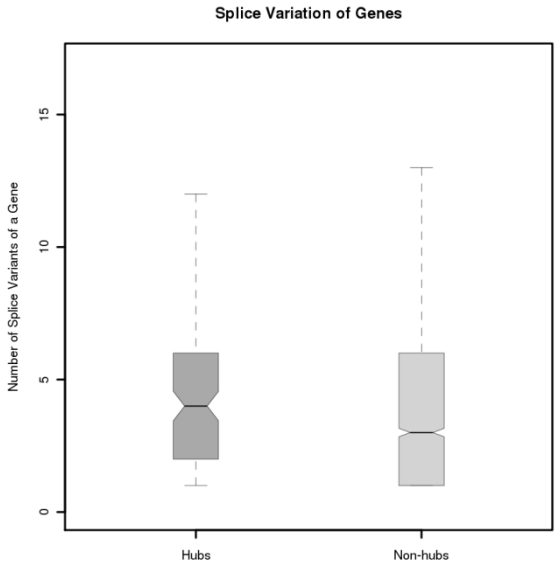

Splice Variation of Genes
Number of Splice Variants of a Gene
0
5
10
15
Hubs
Non-hubs

Figure S1J

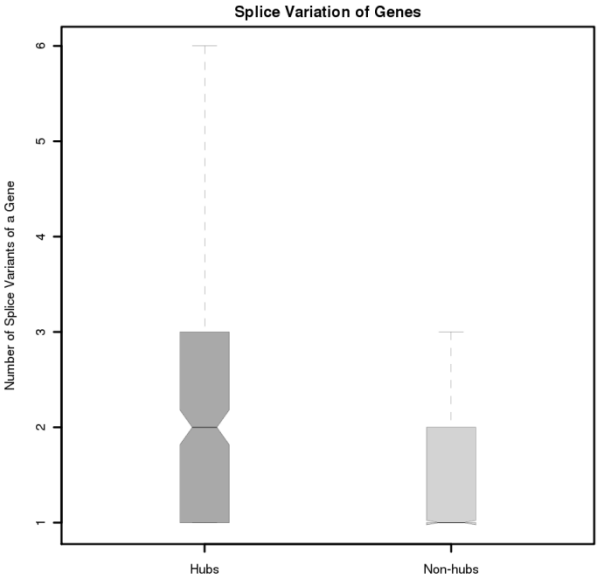
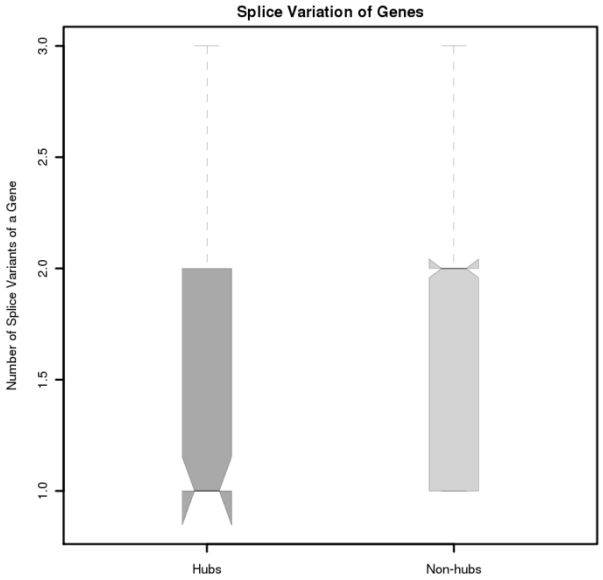


Figure S1K



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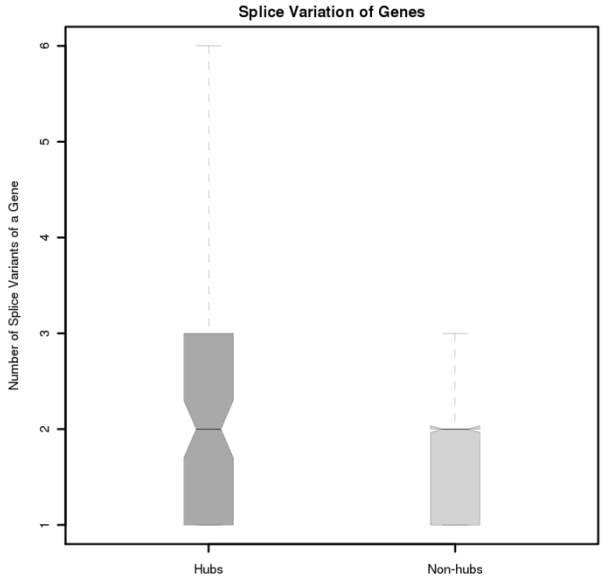

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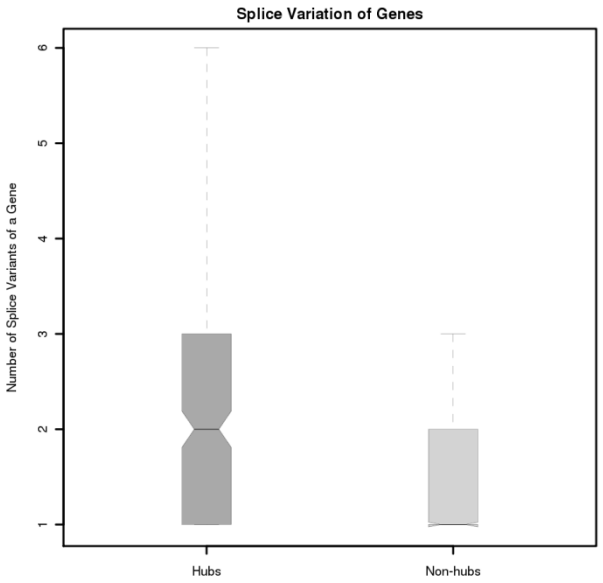

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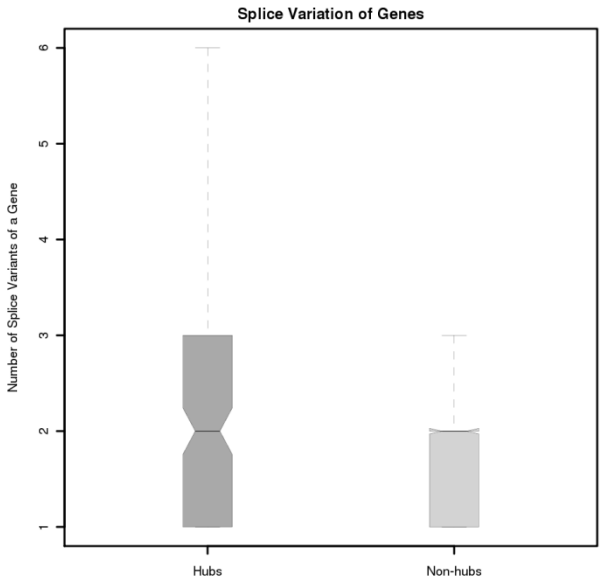

Figure S1O

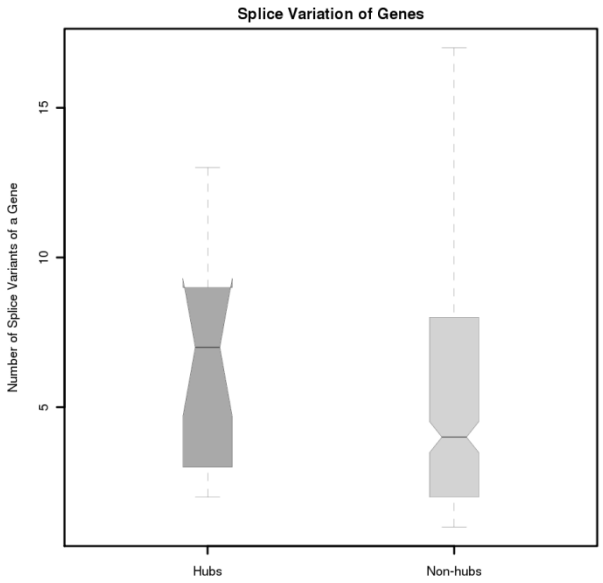

Figure S1P

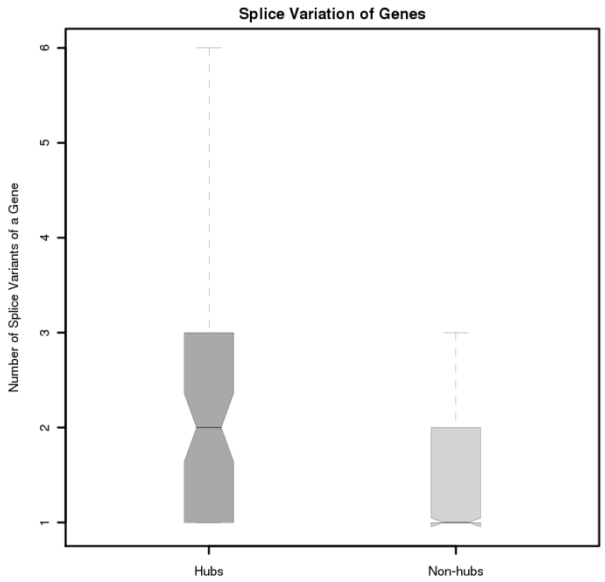
Splice Variation of Genes
Number of Splice Variants of a Gene
1
2
3
4
5
6
Hubs
Non-hubs

Figure S1Q

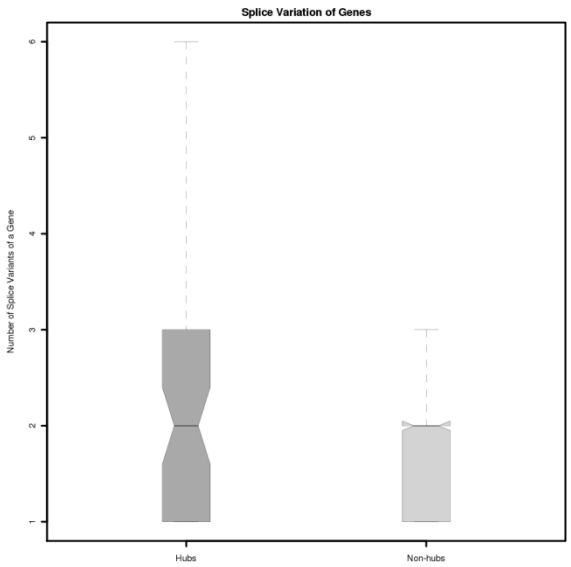

Figure S1R

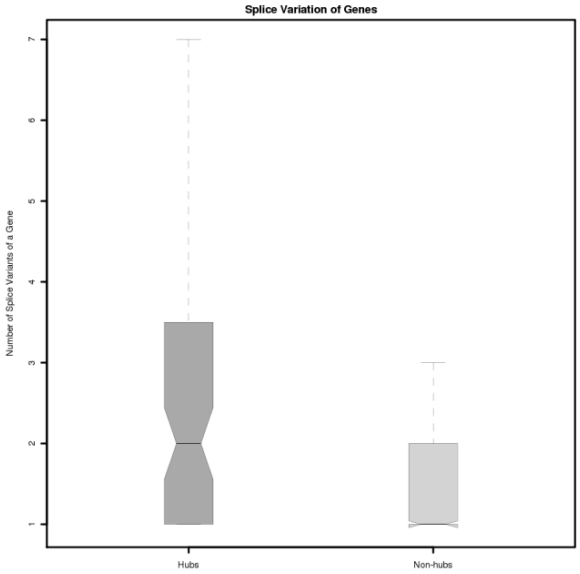

Figure S1S

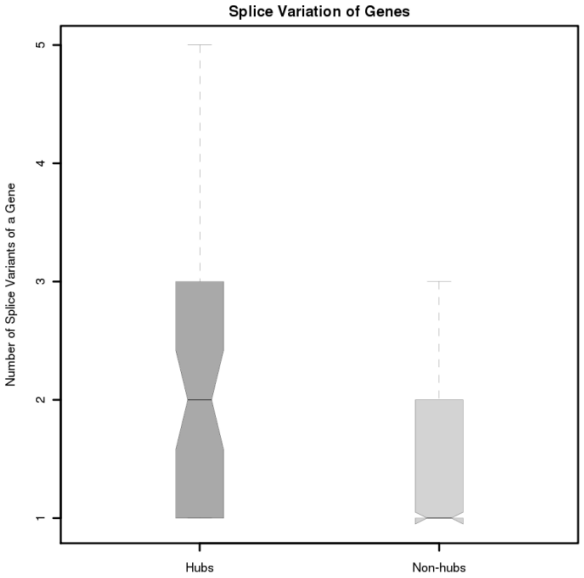

Figure S1T

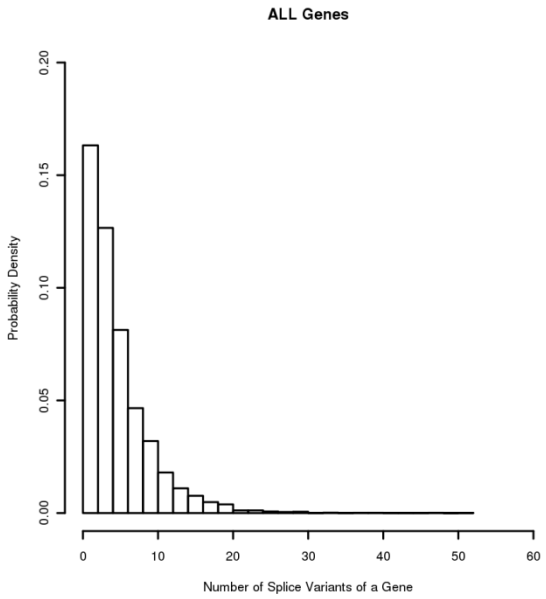

Figure S2 (a)

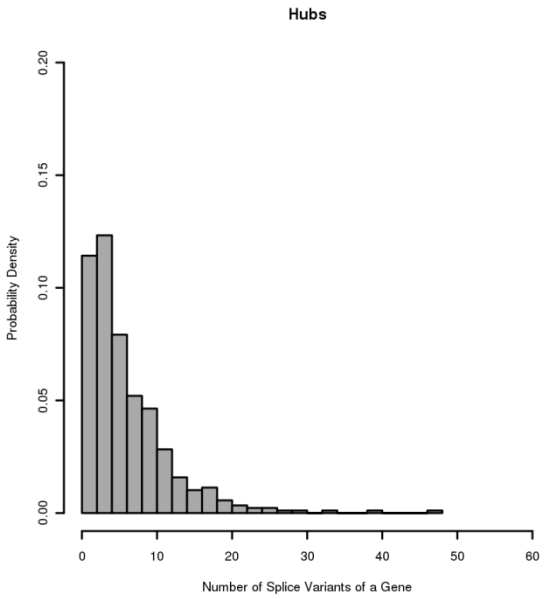

Figure S2 (b)

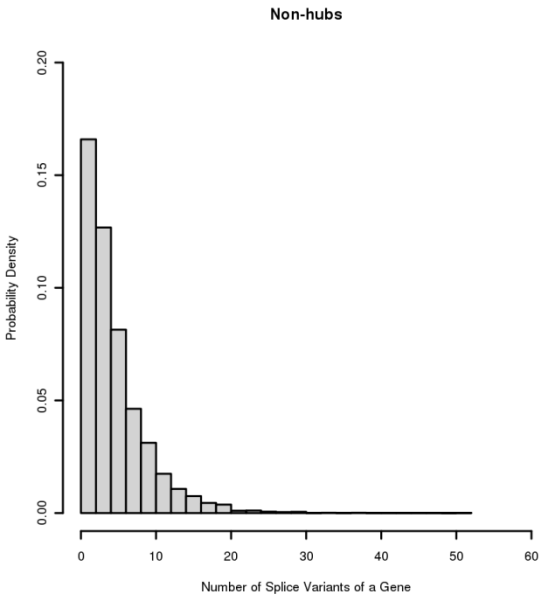

Figure S2 (c)

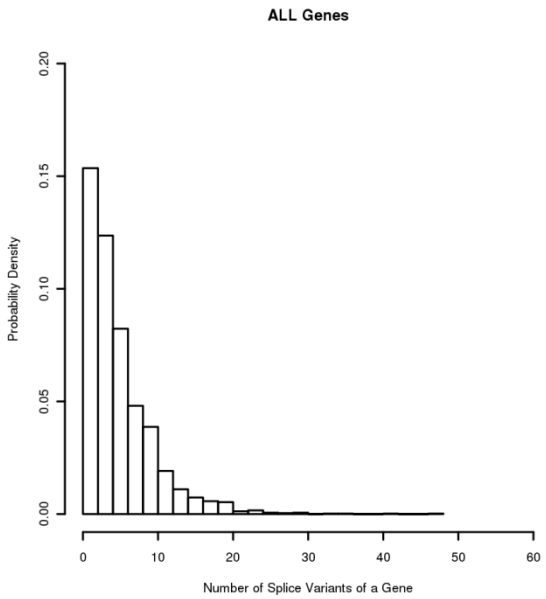

Figure S3 (a)

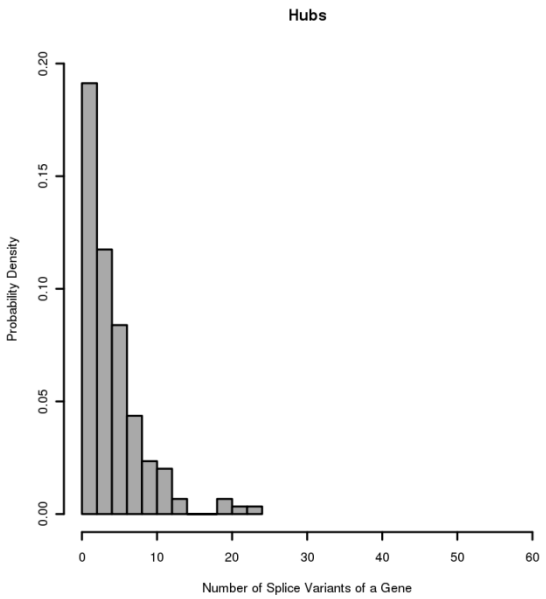

Figure S3 (b)

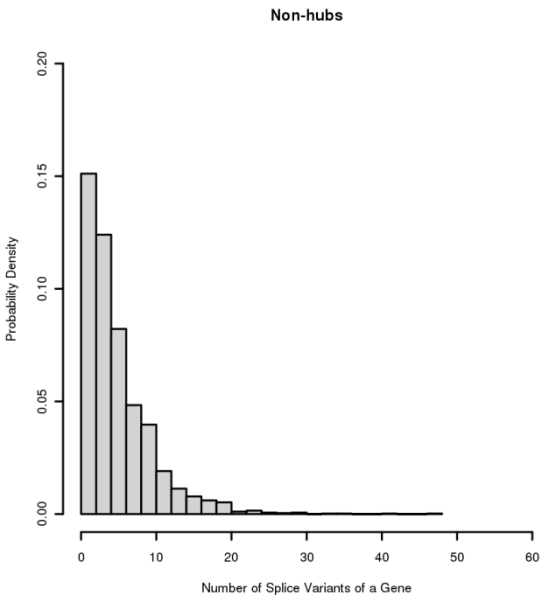

Figure S3 (c)

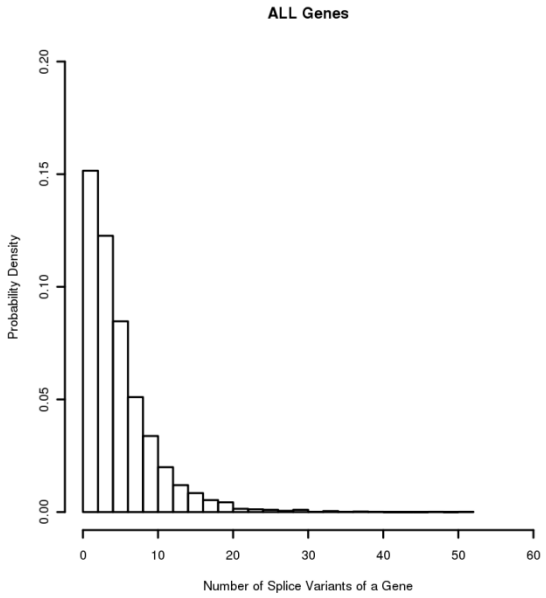

Figure S4 (a)

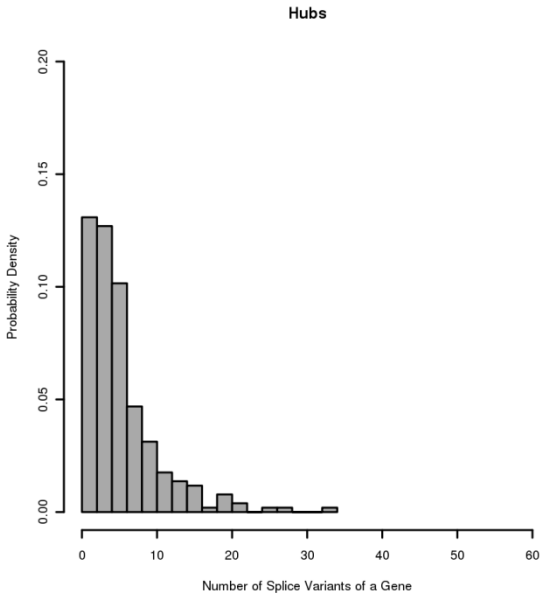

Figure S4 (b)

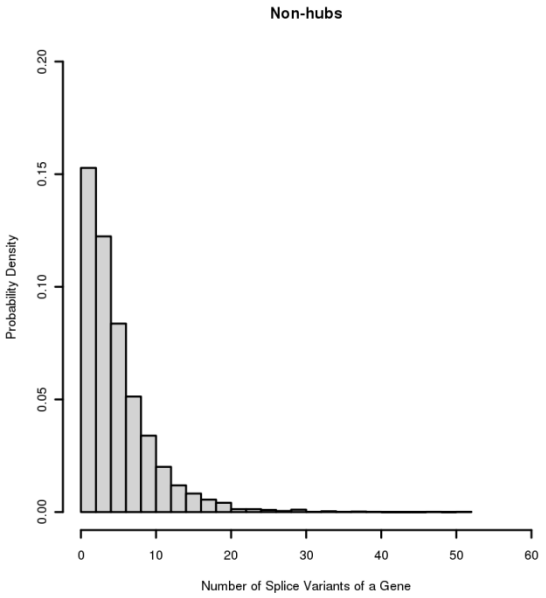

Figure S4 (c)

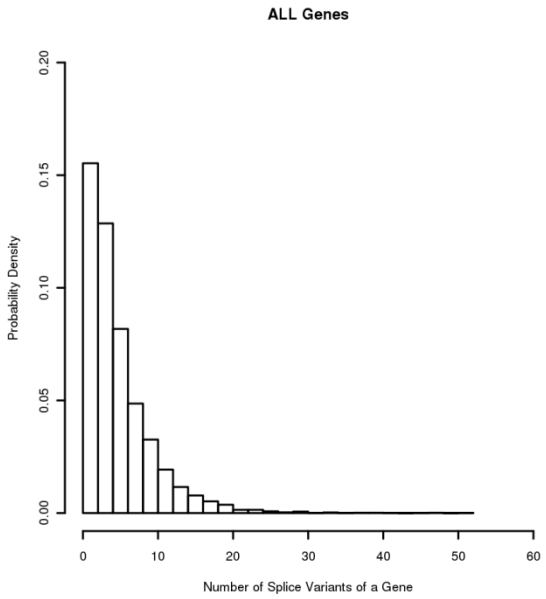

Figure S5 (a)

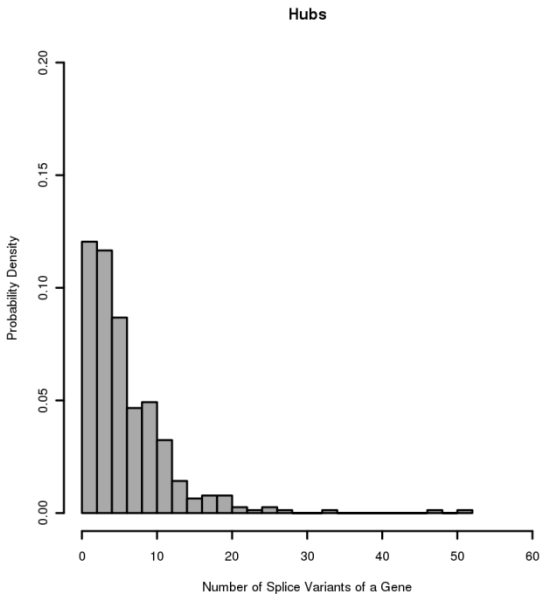

Figure S5 (b)

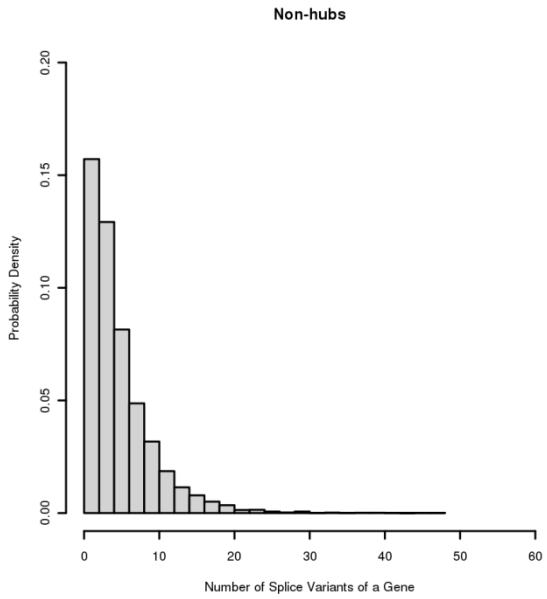

Figure S5 (c)

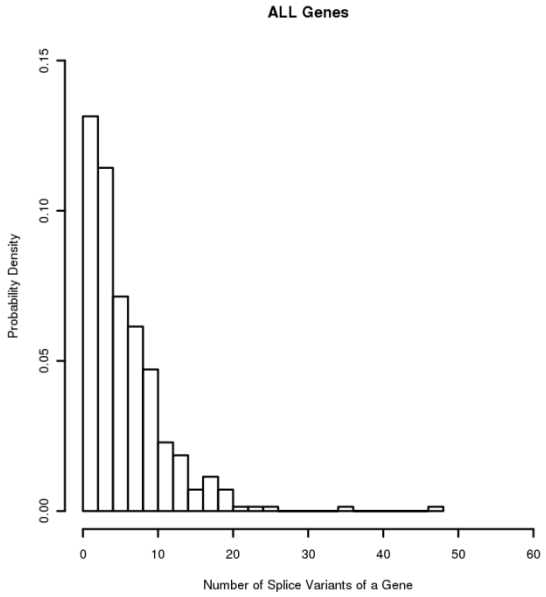

Figure S6 (a)

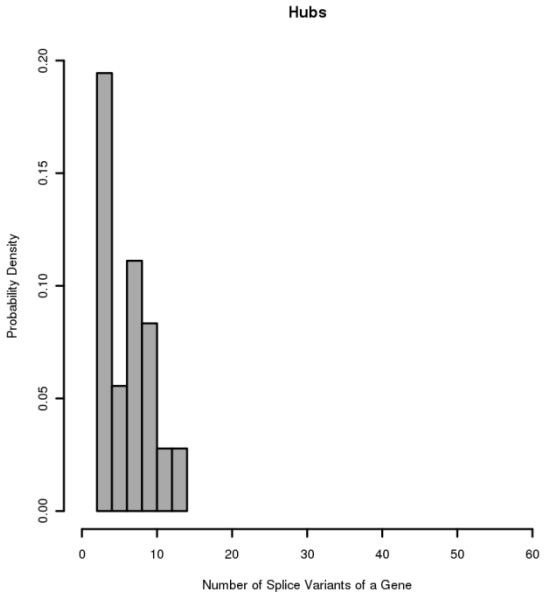

Figure S6 (b)

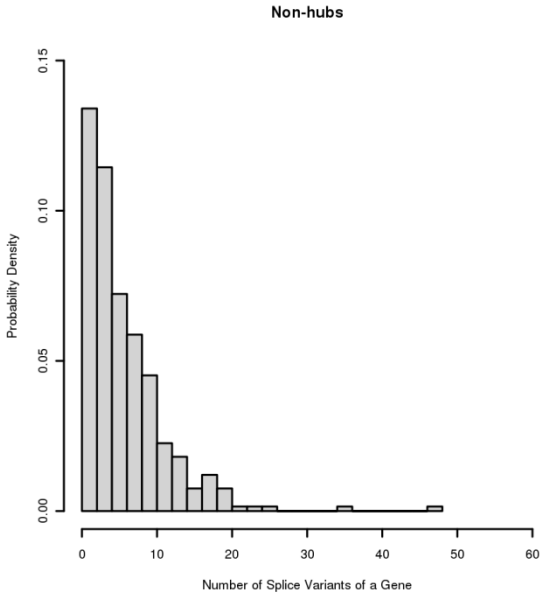

Figure S6 (c)

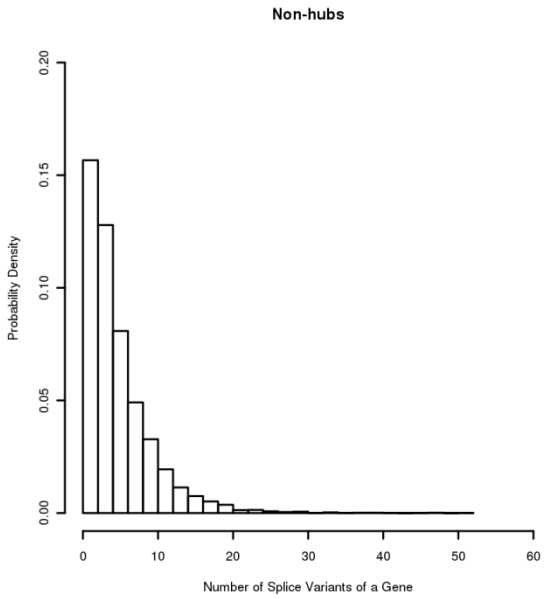

Figure S7 (a)

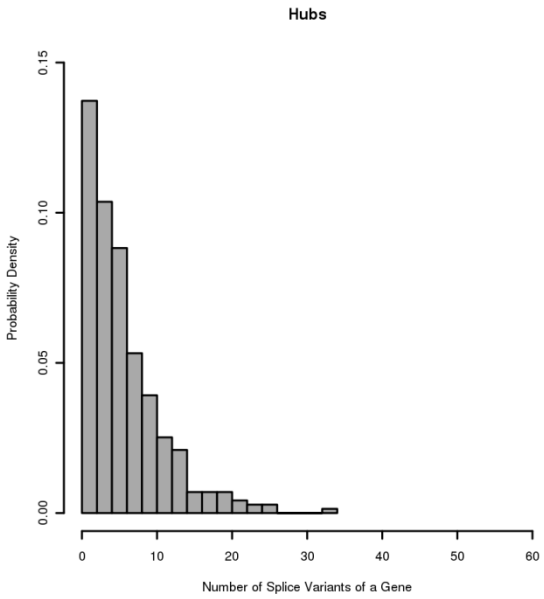

Figure S7 (b)

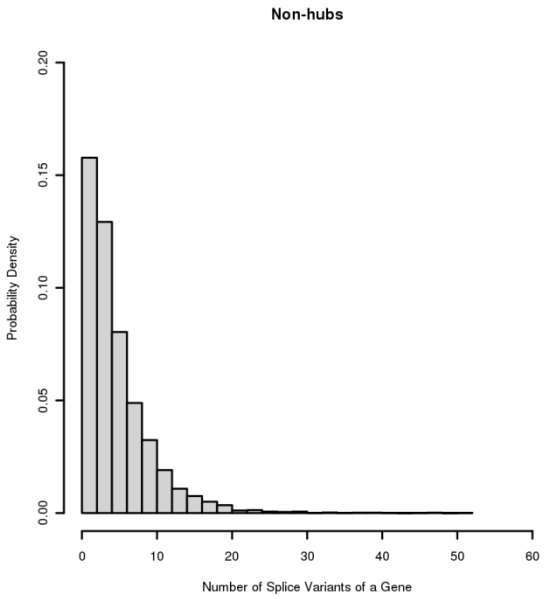

Figure S7 (c)

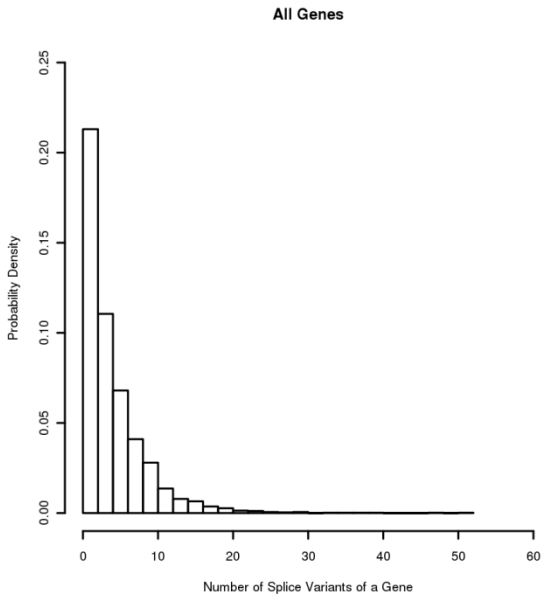

Figure S8 (a)

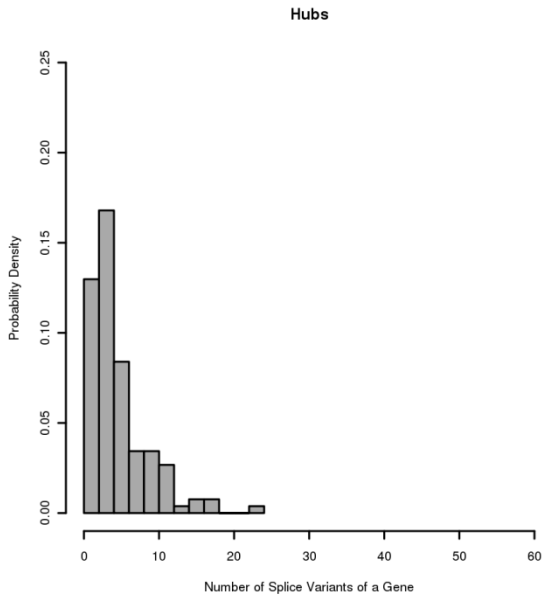

Figure S8 (b)

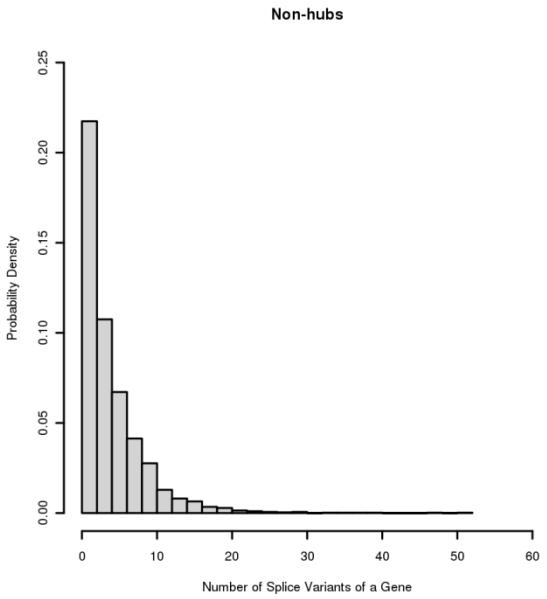

Figure S8 (c)

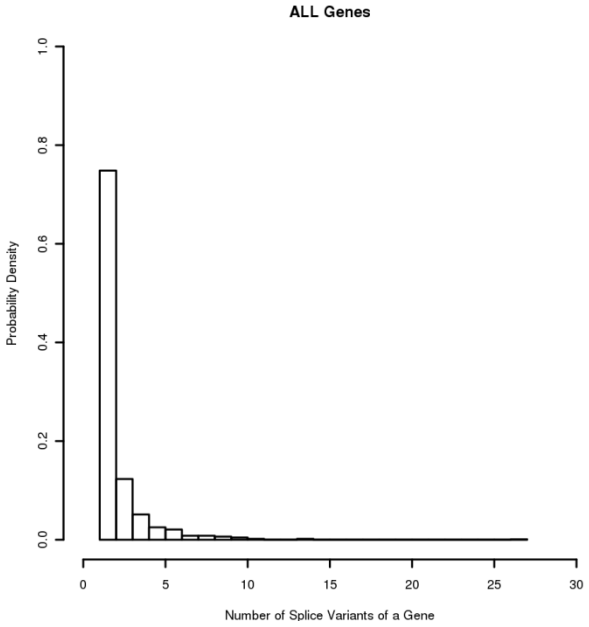

Figure S9 (a)

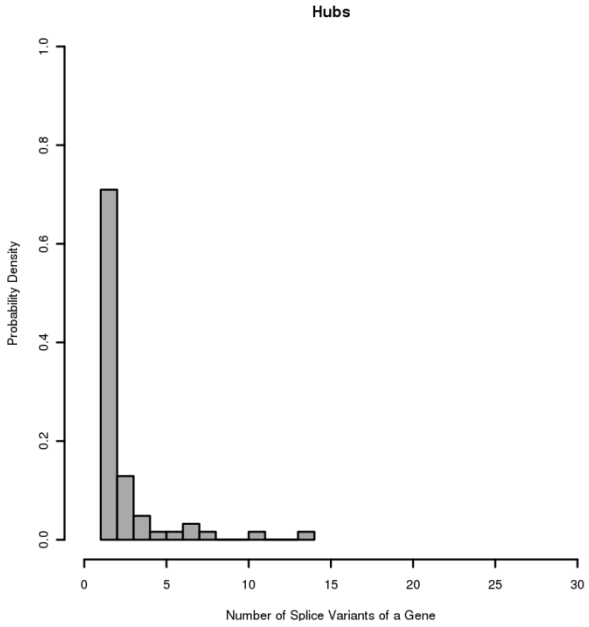

Figure S9 (b)

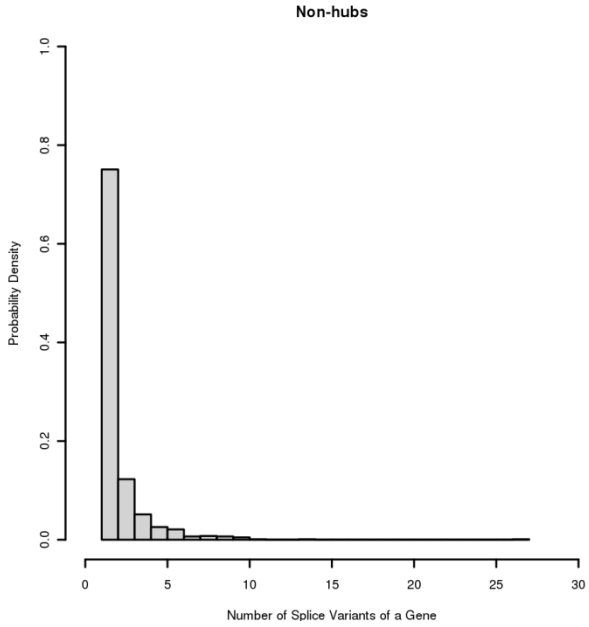

Figure S9 (c)

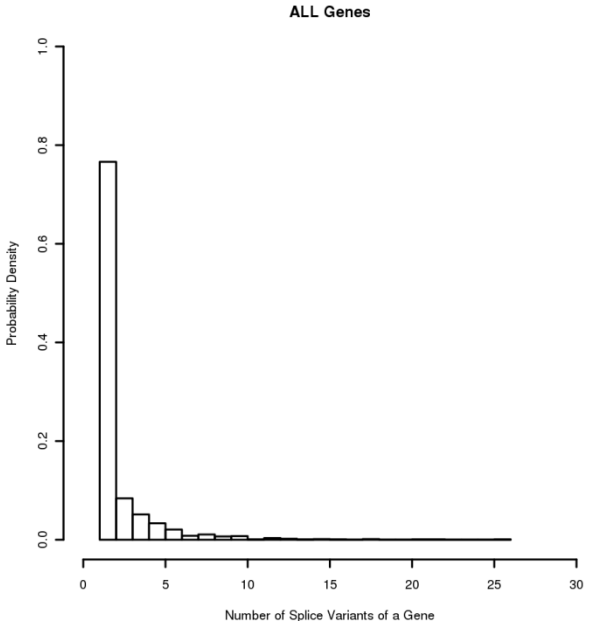

Figure S10 (a)

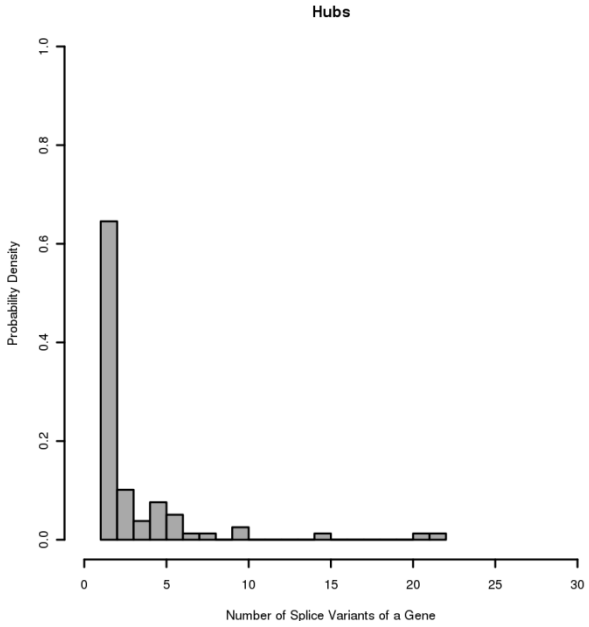

Figure S10 (b)

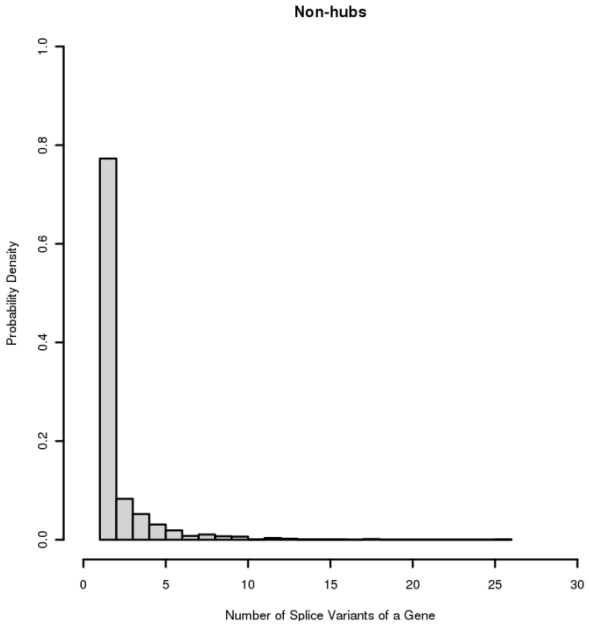

Figure S10 (c)

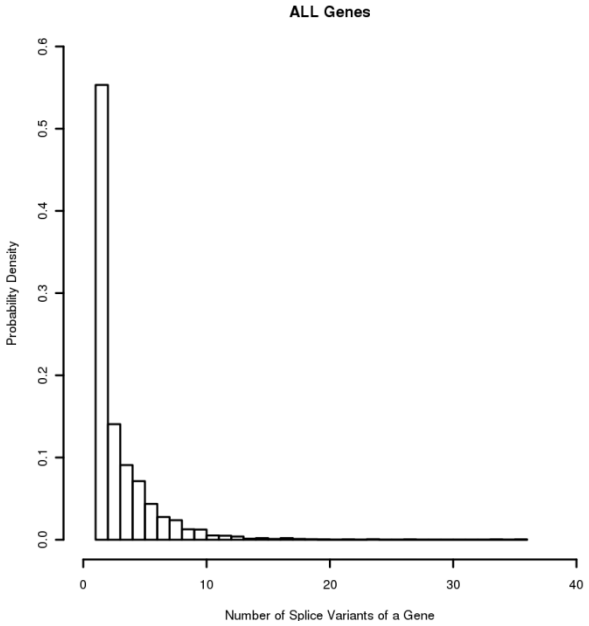

Figure S11 (a)

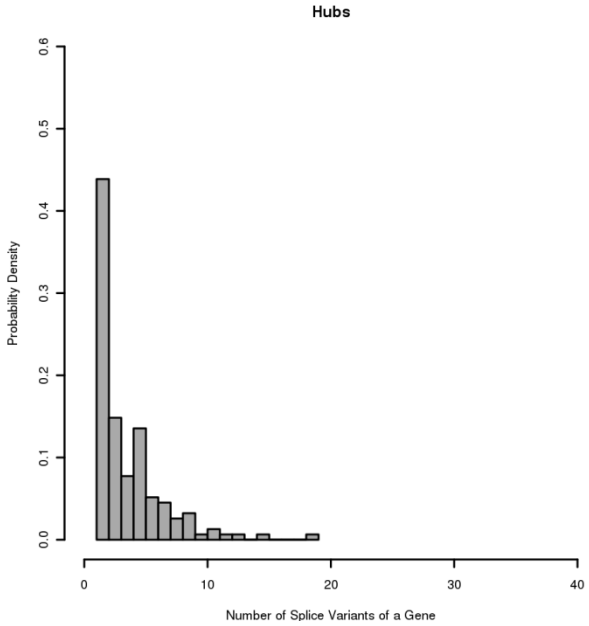

Figure S11 (b)

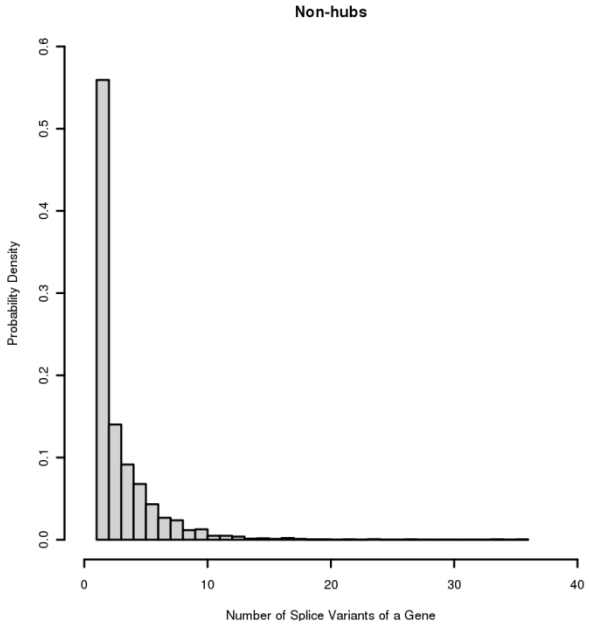

Figure S11 (c)

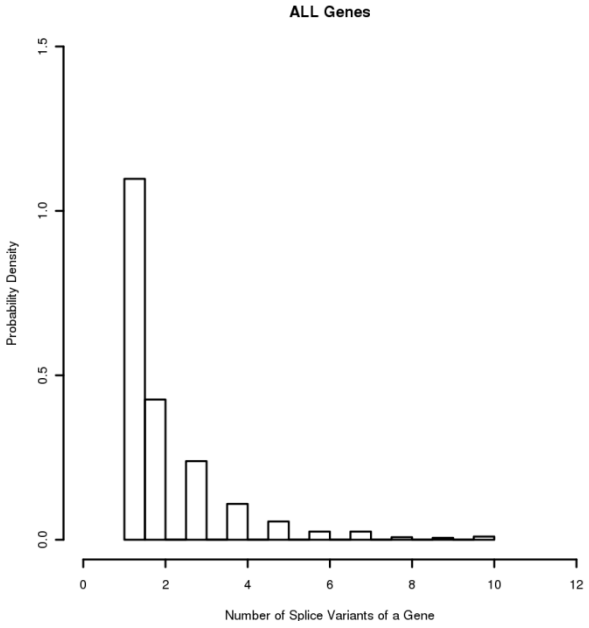

Figure S12 (a)

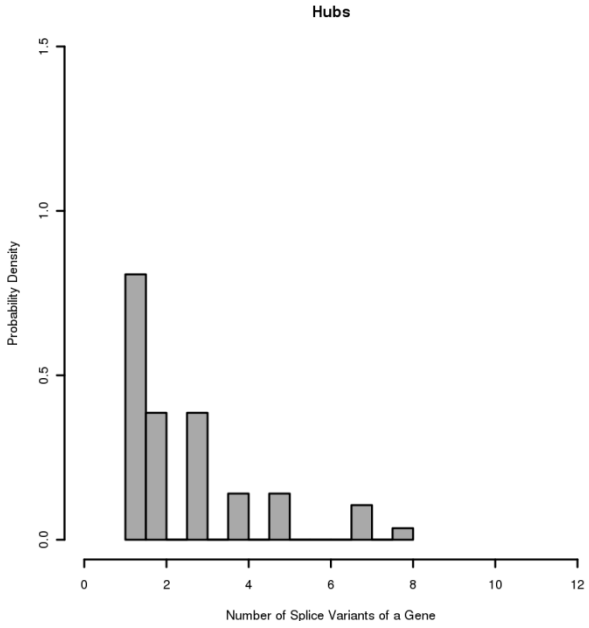

Figure S12 (b)

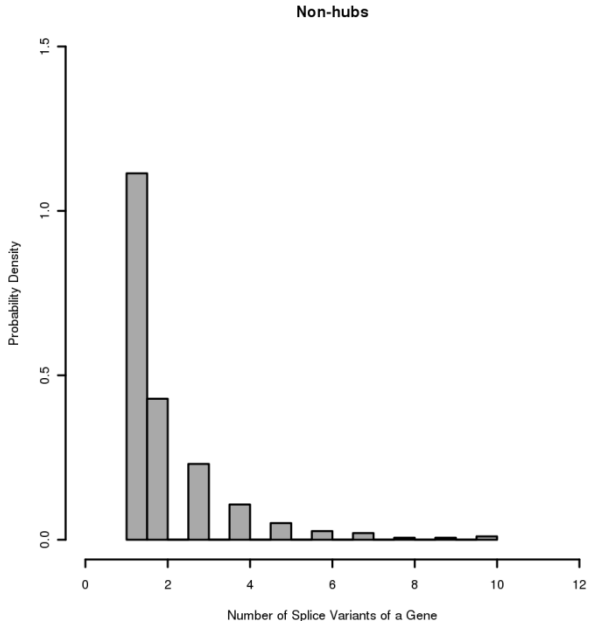

Figure S12 (c)

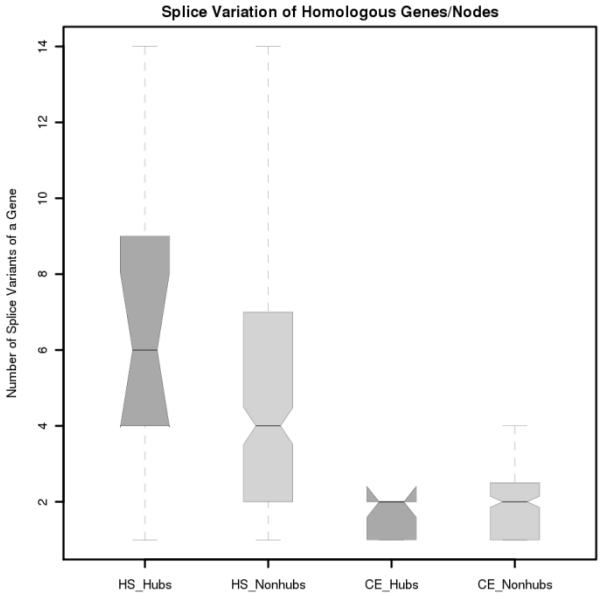

Figure S13 A

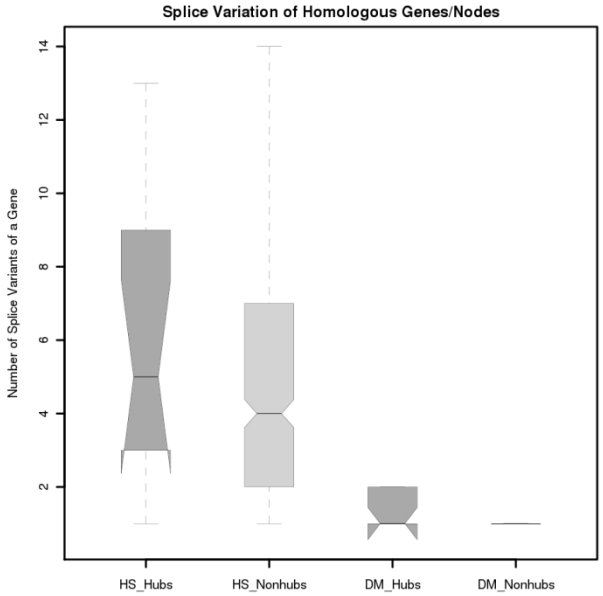

Figure S13 B

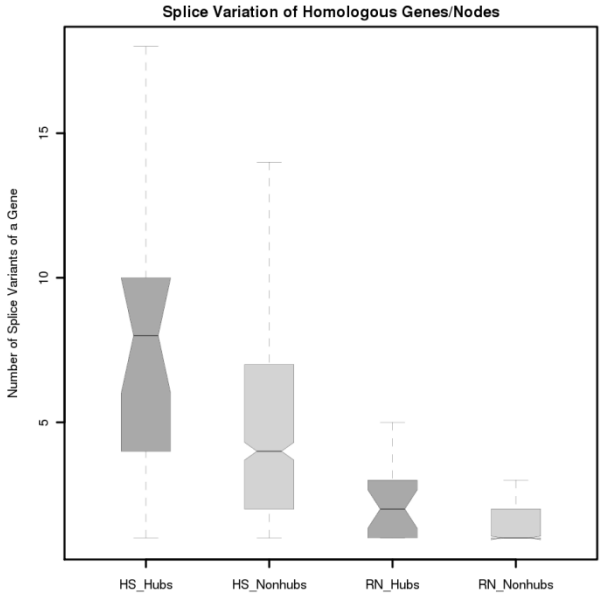

Figure S13 C

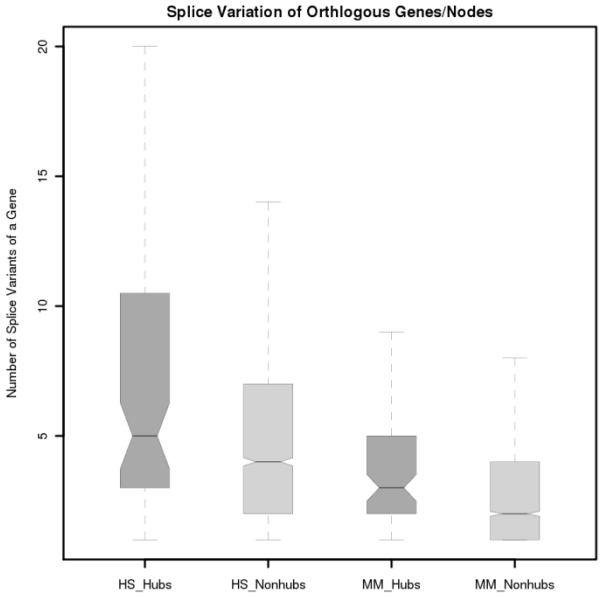

Figure S14 A

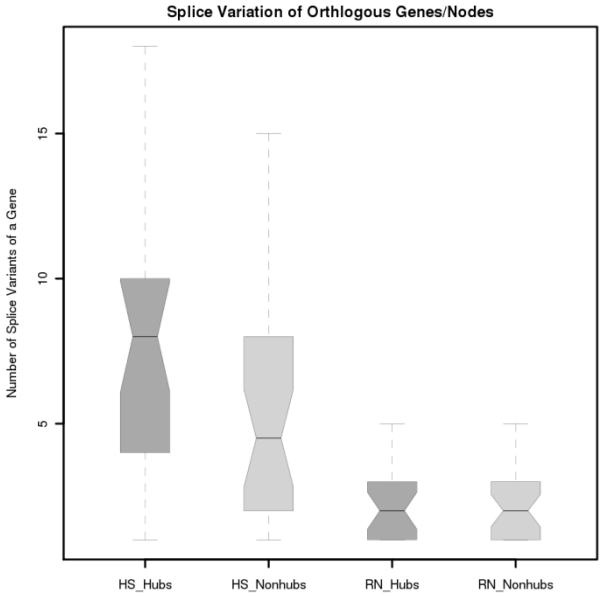

Figure S14 B

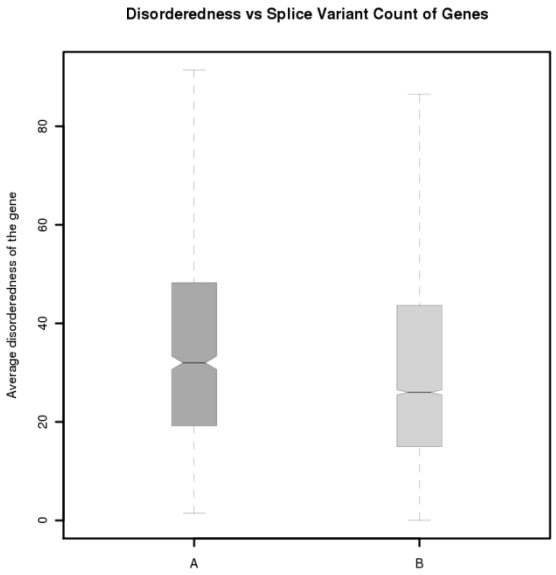

Figure S15 A

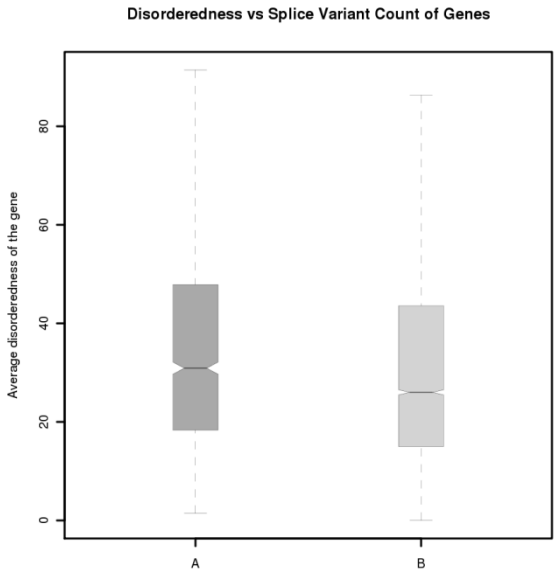

Figure S15 B

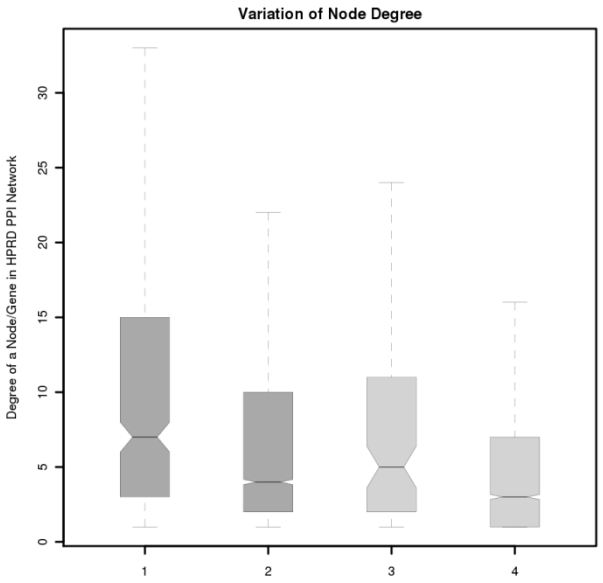

Figure S16 A

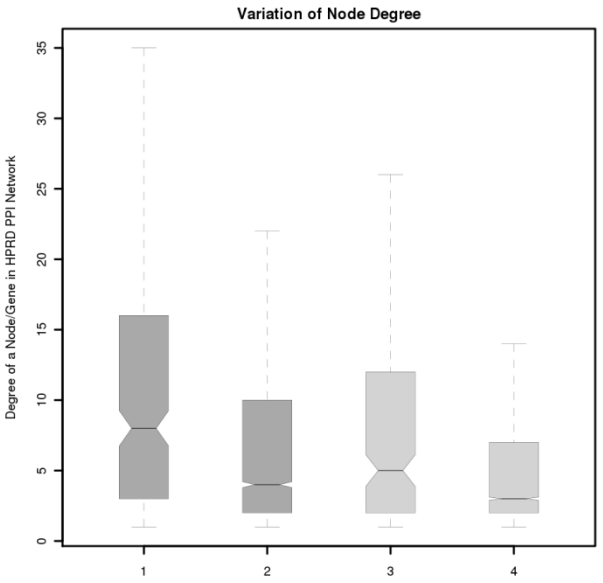

Figure S16 B

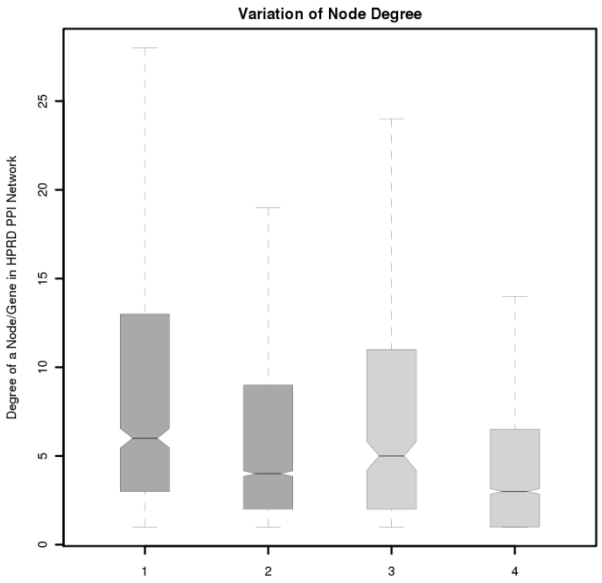

Figure S16 C

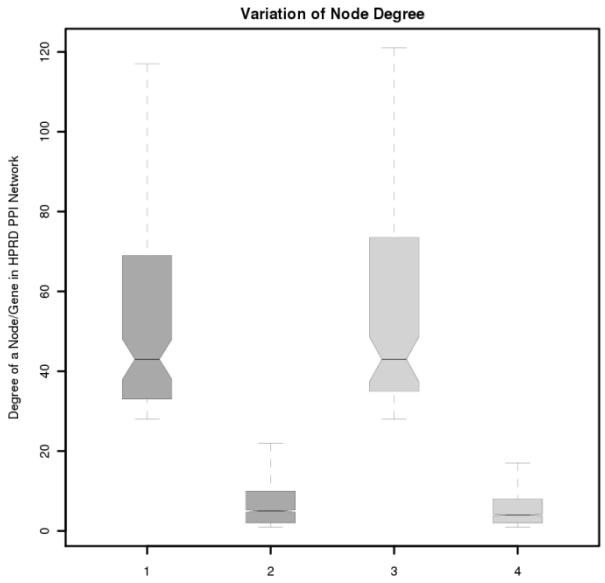
Variation of Node Degree
Degree of a Node/Gene in HPRD PPI Network
0
20
40
60
80
100
120
1
2
3
4

Figure S16 D