Figure 1: growth kinetics of established breast PDXs. The average duration of the 3 first passage was calculated for each PDX and used as an estimate of their growth rate. Time is presented in days. Each bar represents a PDX. Color of the bar indicates the molecular subtype to which the PDX was assigned. As shown in Figure 2 red=Bas-L, light blue=Lum-B, pink=Lum-C, orange=m-Apo.
Figure 2: Molecular classification of PDX and tumors of origin are highly concordant. Identical assignment were found in 87% of the cases with at least 3/5 identical classifications for each PDX. Divergences were restricted to the luminal subtypes. Transcriptome PDX and corresponding tumor of origin were classified using the Guedj, Hu, PAM 50, Sorlie and Neve breast cancer classifiers and assignments were indicated by a color code. Red: Basal or Bas-L, Orange: M-Apo (ER-/HER2+), Pink: Lum-C (ER+/PR-), light blue: Lum-B (ER+/PR+/HER2-), dark blue: Luminal A. ER, PR status determined by IHC is presented in red for ER-, in green for ER+. HER2 status was noted negative in green when scored <2+, positive in red score = 3+. TP53 mutation status determined by DNA sequencing, mutated in red, wt in green.
Figure 3: Breast tumors of origin and corresponding PDXs show elevated similarity at the genomic level. CNC profiles of all PDXs (* indicates corrected profiles) and breast tumors of origin were determined by array-CGH and convergence tested by clustering analysis. Breast tumors and their cognate PDXs were systematically clustered together (pairs, triplet or quadriplet according to profile availability).
Figure 4: CNC profiles of different grafts from the same primary tumor remain remarkably stable.

A: Graft tree indicating the position of the analyzed PDXs which have been highlighted by a color code.

B: Histograms showing the fraction of overlapping events in the tumor of origin and the corresponding PDX which are identified by color codes indicated in the graft tree.

C: whole genome CNC profiles, gains are shown in blue, losses in red, chromosomes by alternating light blue and white bars. Samples are identified as shown in the tree.
Figure 5: quantification of CNC specifically found in primary tumors (A) or specific of the PDX (B). Events specific to the tumors give an insight on tumor heteroclonality, whereas those found only in the PDX represent events acquired *de novo* during propagation in the mice. A: Each bar represents the percentage of the genome interrogated by the array involved in primary tumor specific CNC. B: the fraction of the genome corresponding to CNC occurring *de novo* in the PDX. CGH profiles of the PDXs and cognate primary tumors were analyzed after correction for the contamination by normal stromal cells.
Figure 6: Expression differences between primary tumors that give rise to PDX (Take) and those that did not (No Take). We analyzed 3 expression signatures with known prognostic impact and two further signatures (VEGF, IL8). The tumor set was stratified according to molecular subgroups to verify whether these differences were not due to differences in composition of the Take and the No Take groups. GGI identifies the molecular grade signature (ref), Wound healing (ref) and proliferation signature (ref).