INTRODUCTION
A horse without clinical lameness is the aim of all rider. In another way, radiographic status of
the horse has more and more impact on his economic value. Is this trait heritable and which is
the relation between radiographic status and performances?

MATERIAL
A design for the detection of juvenile osteo-articular lesions of young horses has been set up at
the ENVA from 1991 (Denoix et al. 1997, 2000). Horses were provided by 3 national
organisations: the national stud, “la garde républicaine”, one national riding school and one
private stud. They were generally aged 3 (83%) or were examined early in their fourth year and
were from the breed Selle Français. 14 radiographic views were taken on the left and right fore
and hind limbs, for 15 localisations (Table 1). 1050 horses with radiographic findings were
registered with identification number. To carry out the genetic analysis, the file was restricted
to horses born from stallions with at least 3 offspring. Then, the file contained 733 horses from
103 different sires. All registered ancestors of these 103 sires represent 1506 horses which
were included in the analysis.

Radiographic signs were classified with a severity code according to Denoix et al. (1997). The
radiographic findings were classified in 4 grades with coefficient of severity between
parentheses: suspicious and transitional (1), abnormal with unsure clinical significance (2),
abnormal with probable clinical significance (4), abnormal with sure clinical significance (8).
So, for each site, there was a sequence of qualifications which was the set of visible lesions in
the right and left limbs. Then, two measures were used in the analysis:
- A gradual discrete measure. The measure is the sum of coefficients of severity of
  abnormality detected on the X-ray for each localisation.
- A bivariate measure. When at least one abnormality on the localisation was detected, the
  measure was 1, and 0 otherwise (if findings were normal or only doubtful).
A variable which summarises lesions on all localisations was also studied. This variable is the
sum of each measure on each localisation transformed into a normal deviate after estimation of
thresholds. Due to missing data, carpus, hind foot and stifle were discarded from this sum.

METHOD
The measures of lesions are categorical variables (except for the all-body variable). The
underlying variable susceptibility to osteochondrosis was analysed with the following model:

Session 05. Horse breeding Communication No 05-08
\[ y_i = s_j + a_l + b_o + r_q + m_t + n_u + e_{ijqp} + v, \]
where \( i \) is the \( w \)th horse form sire, \( y \) is the underlying performance responsible of lesions, \( s \) is the effect of the sex (male, female, gelding), \( a \) is the age effect (3 years old, 4 years old, 5 years old and more), \( b \) is the breed effect (Selle Français, Anglo-Arab and thoroughbred), \( o \) is the owner effect (4 different owners), \( r \) is the region effect (4 regions), \( m \) is the month of birth (January and February, March, April, May, June and July), \( n \) is the year of examination (1991 to 1993, 1994, 1995, 1996, 1997, 1998), \( u \) is the random sire effect, and \( e \) is the residual effect. Variances between sire effects included relationship matrix. Residual variance was set to 1.

The model used to analyse the influence of lesions on performances in jumping competition was the following : 
\[ y_i = v_j + l_k + p_l + e_{ijl}, \]
where \( y \) is the Log(annual earnings in jumping) corrected for sexe, age and year (estimated from the whole population of horses in jumping), \( v \) is the breeding value estimation before competition (3 years old) for jumping ability (from official breeding evaluation of whole population for jumping), \( l \) is the the score for the lesion in each location, \( p \) is the permanent environmental effect, and \( e \) is the residual error.

RESULTS
The prevalence of radiographic abnormality varied from 1% to 13% according to the localisation (Table 1). These occurrences reached 2% to 26% for suspect findings. But horses with no abnormality on any localisation were only 37% and with no suspect findings they were 15%.

Heritabilities are in Table 1

There were few significant phenotypic correlations between localisations, but all significant ones were positive and moderate. Inside the fore foot, there was low positive correlation between sesamoïde bone and other part of the foot (0.13). It was 0.13 between distal interphalangeal joint and fetlock and 0.28 between distal phalanx and carpus. Inside the hind limb there was a correlation between foot and pastern (0.29) and between hock and fetlock (0.17 for dorsal aspect of fetlock). Between fore limb and hind limb there was a correlation between feet (sesamoïde bone 0.22, distal interphalangeal 0.25), between hind pasterns and sesamoïde bone (0.15), distal interphalangeal joint (0.21) and fore pastern (0.33), between hind fetlock and fore pastern (0.21) and fore fetlock (0.32, 0.18). These results suggest a susceptibility of all pasterns (fore and hind) and all fetlocks (fore and hind).

Effect of lesions was significant (5%) for sesamoïdal bone, distal phalanx of the fore foot, distal interphalangeal joint of the fore foot, hind foot and femoropattelear joint. Effect of the sum of lesions was significant only for the 16% worse horses, .25 phenotypic standard deviation below average.

DISCUSSION
In the literature, prevalence of such diseases on sport horses were reported from german and dutch horses population in studies on heritability. But differences between distribution in populations studied seemed to arise from definition of codification, despite a theoretically equivalent scale with 4 similar gravity codes (majority from the European X Ray commission) in these studies (Willms et al., 1996 ; Winter et al , 1996 ; Van heelsum et al., 1996). For
example, for navicular disease, the first code (sound) had an occurrence equal to 0.23 (Willms et al.), 0.20 (Winter et al.), 0.00 (Van heelsum) and 0.75 in this study. Adding the first two codes gave respectively 0.74, 0.42, 0.27 and 0.84. So, if diseases were comparable and population perhaps also, the scale of measurement had no relation between countries or regions.

Table 1. Prevalence and estimates of heritability of osteo-articular lesions of sport horses

<table>
<thead>
<tr>
<th>Localisation</th>
<th>Frequency of abnormality</th>
<th>$h^2$ of gradual measure</th>
<th>$h^2$ of all-or-none measure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sesamoïdal bone</td>
<td>11%</td>
<td>0.50</td>
<td>0.16</td>
</tr>
<tr>
<td>Distal phalanx</td>
<td>7%</td>
<td>0.08</td>
<td>0.15</td>
</tr>
<tr>
<td>Distal interphalangeal joint</td>
<td>12%</td>
<td>0.04</td>
<td>0.00</td>
</tr>
<tr>
<td>Other parts of foot</td>
<td>3%</td>
<td>0.87</td>
<td>0.63</td>
</tr>
<tr>
<td>Proximal interphalangeal joint</td>
<td>4%</td>
<td>0.00</td>
<td>0.07</td>
</tr>
<tr>
<td>Metacarpo-phalangeal joint/Fetlock</td>
<td>11%</td>
<td>0.10</td>
<td>0.21</td>
</tr>
<tr>
<td>Carpus</td>
<td>11%</td>
<td>0.17</td>
<td>0.12</td>
</tr>
<tr>
<td>Hind foot</td>
<td>2%</td>
<td>0.06</td>
<td>0.21</td>
</tr>
<tr>
<td>Proximal interphalangeal joint of the hind limb</td>
<td>5%</td>
<td>0.07</td>
<td>0.00</td>
</tr>
<tr>
<td>Dorsal aspect of the metatarso-phalangeal joint</td>
<td>8%</td>
<td>0.04</td>
<td>0.12</td>
</tr>
<tr>
<td>Plantar aspect of the metatarso-phalangeal joint</td>
<td>11%</td>
<td>0.00</td>
<td>0.01</td>
</tr>
<tr>
<td>Proximal tarsal row</td>
<td>11%</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Distal tarse row</td>
<td>13%</td>
<td>0.02</td>
<td>0.00</td>
</tr>
<tr>
<td>Femoropatellear joint</td>
<td>7%</td>
<td>0.09</td>
<td>0.17</td>
</tr>
<tr>
<td>Femorotibial joint</td>
<td>1%</td>
<td>0.00</td>
<td>0.11</td>
</tr>
</tbody>
</table>

Differences in binary measure and progressive measure for the same localisation may be explained by the difference of sorting between animals according to the measure. In any case these differences reveal that the method of measure of radiological lesions is very important in the interpretation of heritability and may explained differences with bibliography.

In the literature, each location presents different features. Heritability on sesamoïdal bone, or navicular disease in other studies, varied from 0.07 to 0.31 (mean 0.21), on riding horse population (Winter et al., 1994; Willms et al., 1996; Van Heelsum et al., 1996). The lower estimation was obtained with a continuous method applied to categorical data, and so was expected to be underestimated. Our own estimation ranged from 0.16 (5 categories) to 0.50 (all or none trait). The work of Winter et al. was based on the largest population (2182 horses, 237 sires) but as in our work not from a random sample: horses were sold at auctions. The others works used random sample of comparable number of horses than in our work. In conclusion, it seems reasonable to expect a moderate heritability near 0.20 or 0.30 for navicular disease, and perhaps a more drastic measure (all or none trait) might increase heritability as it would reveal a major pathology and not "noises". For osteochondrosis in hock, differences in estimations of heritability seemed to depend on the breed. In standardbred, heritability was reported in several studies (0.24 in Phillipson et al., 1993; 0.52 in Grondhal et al. 1993) but not in riding horses.
Our study on hock seemed to agree with these results (no heritability on distal or proximal tarsal row). For fetlock, estimations of heritability were homogeneous: form 0.09 to 0.24, mean 0.18 for riding and trotter horses. Our results are in agreement: 0.10 to 0.21 for fore fetlock, 0.04 to 0.12 for dorsal aspect of hind fetlock. A low heritability, near 0.15 may be expected. For carpus, it was only studied by Dolvik and Klemetsdal (1994) on Norwegian trotter. Heritability was 0.25, and we found 0.17 to 0.12. A higher heritability (0.65) was obtained by Dolvik and Klemetsdal when considering only bilateral lesions. So, diffuse problem with carpus give moderate heritability but important problem reveal a higher heritable pathology.

Influence of radiographic findings on performance is not straightforward. Very affected horses on all localisations have weaker performances (~25% of standard deviation) but for each localisation there were few major effects: only 5 on 15 localisations were significant and, very often, the effect was not linear. For example, the effect of lesions on the distal interphalangeal joint was positive on performance for scores 1 compared to normal one (+18% of standard deviation), unfavourable for score 2 (~25%) and neutral for scores 3 and more. According to the number of data only large effects may be estimated and then low effect must be validated in the future.

For some localisation, the use of drastic measure (all or none) seemed to give a better interpretation for genetic purpose (sesamoïdal bone, carpus). In others, gradual measures would be better. The choice of measure led to difficulty in comparing the distribution of variables in the different populations, but heritability estimates seemed to converge to the same values. Sesamoïdal bone, fetlock and carpus were sites with moderate heritability. Hock sites in riding horses presented only low heritability.

REFERENCES