Chapter Seven: Ecology of disease transmission in multi-host systems

(Chapter reference: Caron, A., de Garine-Wichatitsky, M., Morand, S. (Resubmitted after major revisions to *Ecology Letters*) Ecology of emerging disease transmission in multi-host systems)
Introduction

Predicting the next panzootie or pandemic requires the investigation of multi-host systems (Cleaveland et al. 2001, Taylor et al. 2001). SARS, Ebola, HPAI H5N1 have jumped the species barrier ultimately reaching the human species (Song et al. 2005, Webster et al. 2007, Leroy et al. 2009). Additionally, these diseases all involve wild and domestic hosts. While most significant parasites from domestic animals and humans have probably been described, there is still a large number of unidentified parasites of wild hosts which may translate into emerging diseases for human or domestic species (Hudson et al. 2006). The increased connectivity between ecosystems, artificially created by people and animal movements and human encroachment in pristine areas have resulted in new types of contacts between hosts and pathogens which were very unlikely under natural conditions. Parasites can use these opportunities to spill-over to new hosts at the wildlife/domestic/human interface. This question has attracted recent attention (Jones et al. 2008) but the scientific community struggles to predict which parasite will emerge and where (Dobson and Foufopoulos 2001, Woolhouse 2008). Here, we develop a conceptual and operational framework to identify transmission pathways at this complex interface. We adopt a multidisciplinary approach, integrating recent advances in community ecology, molecular epidemiology, evolutionary biology and social network analysis, shifting the research focus from the host or the pathogen to the transmission process per se.

Critical advances in ecology and epidemiology

Community ecology aims at understanding the rules governing species assemblage in communities (Poulin 2007a). Factors influencing parasite species composition differ between host infracommunities (individual level), component communities (population level) and
parasite fauna (species level) (Guégan et al. 2005, Poulin 2007b). We focus here on component community in host populations at the ecosystem level without making any difference between populations from different species. This level of analysis is necessary to follow transmission pathways between hosts’ populations. A component community is influenced by several factors: a) hosts characteristics all influencing the diversity, quantity and exposure of the hosts’ populations to parasites (body size, home range and activity); b) phylogenetic and geographic distance between hosts’ populations; c) biotic and abiotic factors influencing host species richness and composition (e.g. fragmentation of the landscape, climate). The recent developments in parasite community ecology (summarized in Thomas et al. 2005, Collinge and Ray 2006, Poulin 2007b) provide an analytical approach to compare parasite communities between hosts’ populations (Table 7.1). Most of the studies on parasite community ecology do not focus on transmission processes per se albeit these processes are at the core of the phenomenon observed. Therefore, little information has been produced on the dynamics and the temporal dimension of these parasite communities at the ecosystem level (Pedersen and Fenton 2007).

Recent developments in molecular techniques and subsequent availability of genetic information for both hosts and parasites have opened new perspectives to understand host-parasite relationships (Grenfell et al. 2004, Fricke et al. 2009, Haagmans et al. 2009). A dynamic dimension was added to molecular techniques when they were integrated with evolutionary biology (Galvani 2003). Holmes (2007a) emphasised the “research boulevards” ahead of us: co-infection interactions, intra- and inter-host viral evolutionary changes and genome wide interactions. The HPAI H5N1 case is illustrative of the power of these new technologies at hand: the diversity of strains has been used to infer geographical spread of the pathogen across the globe (Cattoli et al. 2009, McHardy and Adams 2009). Phylogenetic dynamics (phylodynamics) are increasingly integrated into quantitative epidemiology by
using concepts borrowed from evolutionary biology (Johnson and Stinchcombe 2007). As the rate of evolution is usually higher for parasites compared to their hosts, parasite genomic is relevant to trace back recent relationship between a host and its parasites (Gonzalez et al. 2007a), but also between populations of hosts (Poss et al. 2002, Biek et al. 2006, Chessa et al. 2009). This type of inference is increasingly used to explore transmission processes for specific pathogens (Heeney et al. 2006, Biek et al. 2007, Gilbert et al. 2007) (Table 7.1).

Application of network analysis to epidemiological data has attracted recent interest (Bansal et al. 2007, Heath et al. 2008). Networks represent contacts (edges) between host individuals or populations (nodes) and the parameters classically computed to characterise the network have an epidemiological significance (Luke and Harris 2007). The network properties inform on the diversity of contacts between host populations and epidemiological inference can be made (Luke and Harris 2007). Once the heterogeneity of contacts between hosts is estimated, the network provides a framework to investigate parasite spread in a defined system. In a specific context, this allows the identification of key nodes for surveillance and control (Waret-Szkuta et al. 2010).

In this paper, we hypothesise that an interdisciplinary research framework using community ecology, molecular epidemiology and network analysis can provide new insights in the understanding of disease transmission in multi-hosts systems. The application of this operational framework should contribute to the identification of the most likely pathways for future parasite emergence.
Table 7.1: Properties of epidemiological interactions between two host populations and references (illustrative, not exhaustive) for methods potentially needed for epidemiological interaction networks.

<table>
<thead>
<tr>
<th>Property</th>
<th>Network representation</th>
<th>Estimation</th>
<th>References using relevant methodology</th>
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</table>
- At population level (Richomme et al. 2006, Dent et al. 2008, Waret-Szkuta et al. 2010)  
- At community level (Caron et al. 2010)                                                                 |
|                          |                        | Contact rate between host populations           | - Comparing species richness (Boyle et al. 1990, Poulin 2003, 2010)  
- Comparing parasite abundance (Krasnov et al. 2005, Munoz et al. 2006, Poulin et al. 2008)  
| Direction                 | Arrow on edge (uni- or bidirectional) | Phylogenetic analysis for one or more parasites |                                                                                                          |
Conceptual and operational framework

At the ecosystem level, the proposed framework focuses on one target population, as defined by Haydon et al. (2002) (e.g. human, livestock or endangered wild populations) which represents the host population at risk from disease emergence. The identification of all hosts’ populations interacting with the target species potentially representing a source of parasites is a crucial step. However to date, the lack of framework for this selection process has often resulted in empirical decision-making. Parasite spill-over between two host populations is more frequent when they are phylogenetically closely related (Nunn et al. 2003). However, epidemiological investigations of recent emerging infectious diseases (EID) have demonstrated that parasite spill-over can involve distantly, related species: rodents and bats represent more than half of mammal species and have been involved in recent EIDs affecting humans (Gonzalez et al. 2007a, Klein and Calisher 2007, Leroy et al. 2009). In fact, it appears that all species interacting, even individually with the target species are relevant candidates as source of EID in a given ecosystem. In addition, disease emergence in a new species often result from complex processes, with several different species involved in the maintenance, the amplification and/or the spread of the parasite. Epidemiologists are thus confronted with an array of (sometimes loosely) interacting species, and belonging to diverse taxonomic groups, which may play a functional role in the transmission of pathogens to the target species. In Box 7.1, we present the concept of “epidemiological functional groups” (EFGs) to structure and standardise this selection process. We draw a parallel with the approaches adopted by community ecologists to assign species to functional groups and elaborate on the concept of EFGs to which hosts’ species could be assigned according to their potential role in the transmission of diseases to a target species.
**Box 7.1: Epidemiological Functional Groups**

Functional ecology focuses on the functions that species play in a community (Calow 1987) (e.g. savanna’s herbivores, ground-dwelling invertebrates) and functional groups of species are defined to address key process-oriented ecological questions (Simberloff and Dayan 1991). We adopt a similar approach with host communities, proposing to allocate the species coexisting in a given ecosystem into epidemiological functional groups (EFG) according to their specific life-history traits and the role they play in the transmission of a parasite, or a group of parasites, in this ecosystem.

The approach first requires a clear identification of the parasite, or group of parasites, at stake (e.g. RNA virus, *Mycobacterium* bacteria) and its mode of transmission between hosts (direct contact, vector-borne or through the environment). All (known) species potentially interacting with the target species (e.g. human, livestock, endangered wildlife) are then allocated to groups defined according to their potential role in the transmission processes: reservoir (primary) host, link (or spreader) between reservoir and target, amplifier host, and incidental (dead-end) hosts.

All species allocated to a given EFG therefore play similar roles in transmission pathways or epidemiological interactions in a particular ecosystem. To a certain extent species are thus allocated to EFG independently from taxonomic considerations and mostly based on ecological considerations as they share (at least temporarily) some resources with target and reservoir species. The example below illustrates how species can be allocated to EFG, and how, even with incomplete or inconclusive epidemiological data, this approach can help identifying key species for an identified transmission pathways.

Leroy et al. (2005) have explored the transmission pathways of Ebola virus in Central Africa. Fruit-eating vertebrates congregate on fruiting trees, a seasonal and discrete resource
in rainforest. This gathering is a potential explanation for Ebola transmission through bat saliva left on half-eaten fruits, dropped on the forest floor and subsequently eaten by great apes, monkeys or duikers. Human beings are thought to get infected when they eat or manipulate these animals. Gonzalez et al (2007b) further provided serological and molecular evidence of Ebola infection of a number of wild and domestic hosts in Central Africa. In this case, host species could be allocated to the following EFGs: fruit-eating bats reservoir, fruit-eating links (e.g. wild primates, some antelopes and livestock such as pigs), fruit-eating dead-ends (e.g. shrew, rodents or birds which are not hunted and consumed by human) and, non fruit-eating animals (e.g. wild and domestic carnivores).

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The parasite emergence that one wants to predict or control is the result of the transmission of a parasite from a reservoir or intermediate host to the target species. The transmission pathway used by this parasite will depend on host mobility resulting in contacts between hosts: direct contact (e.g.: transmission through aerosol or physical contact) or indirect contact (e.g.: through a shared habitat or via a food resource). Not all contacts will result in parasite transmission and there is a limited number of transmission pathways between two host populations which depend on the frequency and intensity of contacts between hosts’ populations. Epidemiological interactions at the ecosystem level can be presented via networks with edges representing the sum of transmission pathways between two nodes (or host populations). Such a network provides hypothetical pathways for future pathogen spill-over in this ecosystem. Two types of data can be used to build epidemiological interaction network: data on host ecology and data on pathogen co-occurrence in host populations.

Ecological data has already been used to estimate host contacts using telemetry, counts or direct observations (Morgan et al. 2004) (see Table 7.1). The main weakness of these techniques is that they underestimate contacts between hosts, as only a fraction of hosts’ populations can be equipped or observed. In addition, the detection of host contacts does not necessarily imply the transmission of parasites (Real and Biek 2007) and the conclusive determination of infecting contacts is often difficult without an experimental and controlled design. Furthermore, few studies have focused on the contacts at the wildlife/domestic interface.

We suggest another approach based on the comparison of parasite component communities shared by sympatric hosts’ populations, which can be considered as an indicator of past host contacts successful in transmission events. In other words, the shared parasites indicate the extent of the epidemiological interactions which have occurred between two
hosts’ populations. At the ecosystem level, the pairwise shared community of parasites between populations can be used to build networks of epidemiological interactions between hosts’ populations. The assumption we make is that successful transmission pathways used by some parasites in the past could also be used by other pathogens, especially if they share the same mode of transmission. In addition, the more parasites with different modes of transmission shared by two host populations, the higher the probability of the epidemiological network to have identified a future transmission pathway.

Interaction networks have been used for public and animal health studies (Bansal et al. 2007, Dent et al. 2008, Heath et al. 2008, Waret-Szkuta et al. 2010). Classically the nodes represent host populations and the edges represent the epidemiological interactions (see for a definition Chapter Three - Caron et al. 2010) between populations. Parasite component communities define the properties of each node and the shared parasites between host populations determine the two main properties of edges: their intensity and their direction (uni- or bidirectional) (see Table 7.1) (and see an example in Box 7.2).

The intensity of the edges can be estimated using direct estimation of host contacts or similarities of component communities (Poulin 2003, Vinarski et al. 2007, Krasnov et al. 2009, Poulin 2010). Community ecology studies have investigated the decay of similarity between parasite communities with phylogenetic or geographic distance (Poulin 2007b). Phylogenetic distance between host populations and sampling effort need to be controlled for, and appropriate methods are currently developed (see references in Table 7.1). By design, the geographic distance between host populations is accounted for as they belong to the same ecosystem but spatially explicit epidemiological networks have also been designed (Poulin 2007b). Qualitative and quantitative methods have been developed to measure component community similarity: the Jaccard Index (Jaccard 1912) is a simple presence/absence index; the Sorensen (Vinarski et al. 2007) and Morisita-Horn index (Horn 1966) are quantitative
indices using proportion of different parasites or abundance (i.e., prevalence data in epidemiology). The diagnostic methods used for parasite detection are also of importance as they do not all detect the same indicator of parasite presence (e.g. antibodies, antigens). If most available studies have used direct observation of macroparasites during post-mortem inspection which is assumed to be both sensitive and specific, for most microparasites however, direct observation is not an option and specific detection techniques need to be applied. The index values calculated by comparing pairs of parasite component communities (nodes) characterise each edge of the network (Table 7.1).

The direction of the interaction between two hosts’ populations indicating which host population is at the origin of the parasite transmission, cannot be measured with the community ecology approach presented above. A priori, epidemiological interactions are bidirectional, as direct contact between two hosts can potentially result in parasite transmission both ways. However, transmission related to host contacts can be asymmetric: when a reservoir host transmits a parasite to a naive population or when the differential use of a habitat translates into indirect parasite transmission. The concepts and tools of population genetics and parasite genomics may help at tracking back the direction of transmission. Phylogenetic trees have appeared in epidemiological literature and successions of outbreaks can be followed based on variations in parasite genomes (Table 7.1). Most of these tools have been applied to parasite species of economic or public health importance such as HIV (Heeney et al. 2006), foot-and-mouth disease (Cottam et al. 2008a) or tuberculosis (Michel et al. 2008). Information on these parasites can be added in edges of the interaction network and inform the network on transmission pathways of neglected parasites sharing an EI.
**Box 7.2: Epidemiological Interaction Network for 14 rodent species and the human species.**

In this example, the human species is our target species and we explore the Epidemiological Interactions (EIs) between the human species and several rodent species present in particular ecosystems of Southeast Asia represented by different habitats (dry and irrigated agricultural areas, forests, and villages).

From the literature (Chaisiri et al. 2010, Herbreteau et al. unpublished), we obtained presence-absence data on 14 rodent species. Information about 34 macroparasite species and 8 microparasite species were collected for these 14 rodent species and susceptibility to these parasites for the human species were taken from the available literature (Table 7.2). A 42 parasites*15 hosts matrix was built (and filled with “1” or “0” for occurrence of infection and absence respectively in each host species. This matrix was used to calculate the Jaccard Index (=number of parasite species present in both host populations/sum of parasite species present in each host populations) displayed in Table 7.3. The Jaccard index value varies therefore between “0” for no parasite species shared and “1” for all parasite species shared.

We used the Jaccard index as a proxy of EIs between each host population and built the corresponding EI network (Figure 7.1).
Table 7.2: Host and parasite species used

<table>
<thead>
<tr>
<th>Target sp.</th>
<th>Homo sapiens</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rodent sp.</td>
<td>Bandicota indicata (Bi), Bandicota savilei (Bs), Verylmys bowersi (Bb), Leopoldamys edwardsi (Le), Maxomys surifer (Ms), Mus caroli (Mc), Niviventer fulvescens (Nf), Rattus andamanensis (Ran), Rattus argentiventer (Rar), Rattus exulans (Re), Rattus losea (Rl), Rattus norvegicus (Rn), Rattus tanezumi (Rta), Rattus tiomanicus (Rti)</td>
</tr>
<tr>
<td>Microparasite sp.</td>
<td>Leptospira, scrub typhus, Bartonella, hanta virus, herpes virus, LCM virus, Trypanosoma, rabies virus.</td>
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</tbody>
</table>

Table 7.3: Matrix of Jaccard index values between 15 host species for a shared community of 42 pathogens. “ID” represents the host species name abbreviation (Hs = Homo sapiens). ‘Nb Patho” indicates the number of pathogens detected in each host species.

<table>
<thead>
<tr>
<th>Nb Para</th>
<th>Bi</th>
<th>Bs</th>
<th>Bb</th>
<th>Le</th>
<th>Ms</th>
<th>Mc</th>
<th>Nf</th>
<th>Ran</th>
<th>Rar</th>
<th>Re</th>
<th>Ri</th>
<th>Rn</th>
<th>Rta</th>
<th>Rti</th>
<th>Hs</th>
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<tbody>
<tr>
<td>Bi 16</td>
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<tr>
<td>Bs 9</td>
<td>0.47</td>
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<tr>
<td>Bb 7</td>
<td>0.15</td>
<td>0.00</td>
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<tr>
<td>Le 3</td>
<td>0.12</td>
<td>0.09</td>
<td>0.11</td>
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<td></td>
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<tr>
<td>Ms 8</td>
<td>0.20</td>
<td>0.06</td>
<td>0.15</td>
<td>0.22</td>
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<tr>
<td>Mc 2</td>
<td>0.06</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.25</td>
<td></td>
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<tr>
<td>Nf 2</td>
<td>0.06</td>
<td>0.10</td>
<td>0.00</td>
<td>0.00</td>
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<td>0.00</td>
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<td>Ran 5</td>
<td>0.24</td>
<td>0.17</td>
<td>0.09</td>
<td>0.33</td>
<td>0.30</td>
<td>0.40</td>
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<td>0.14</td>
<td>0.00</td>
<td>0.09</td>
<td>0.06</td>
<td>0.12</td>
<td>0.10</td>
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<tr>
<td>Re 18</td>
<td>0.48</td>
<td>0.35</td>
<td>0.00</td>
<td>0.11</td>
<td>0.13</td>
<td>0.05</td>
<td>0.05</td>
<td>0.15</td>
<td>0.30</td>
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<td>0.29</td>
<td>0.00</td>
<td>0.00</td>
<td>0.06</td>
<td>0.00</td>
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<td>Rn 23</td>
<td>0.34</td>
<td>0.23</td>
<td>0.03</td>
<td>0.04</td>
<td>0.07</td>
<td>0.04</td>
<td>0.09</td>
<td>0.08</td>
<td>0.33</td>
<td>0.52</td>
<td>0.19</td>
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<td>Rta 32</td>
<td>0.45</td>
<td>0.24</td>
<td>0.11</td>
<td>0.06</td>
<td>0.14</td>
<td>0.03</td>
<td>0.06</td>
<td>0.09</td>
<td>0.32</td>
<td>0.47</td>
<td>0.17</td>
<td>0.72</td>
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<tr>
<td>Rti 13</td>
<td>0.26</td>
<td>0.05</td>
<td>0.05</td>
<td>0.00</td>
<td>0.17</td>
<td>0.07</td>
<td>0.07</td>
<td>0.06</td>
<td>0.36</td>
<td>0.24</td>
<td>0.10</td>
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<tr>
<td>Hs 15</td>
<td>0.48</td>
<td>0.50</td>
<td>0.05</td>
<td>0.06</td>
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<td>0.06</td>
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<td>0.20</td>
<td>0.41</td>
<td>0.42</td>
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</table>
**Figure 7.1:** Epidemiological Interaction Network for 14 rodent species and the human species in the Southeast Asian ecosystems based on presence-absence data for 34 macroparasite species and 8 microparasite species. Each node represents a host species, the size of the node is proportional to the number of parasite species harbored by the host and the color of the circle represents the habitat in which the host species is mostly found (except for human): red=in human settlements; orange=in rice fields; blue=in modified forest and dry agricultural areas; green=in primary forest. Each edge between two nodes represents the shared parasite community and its width is proportional to the Jaccard index. We placed the human species in the centre of the figure and its edges in red for visual comfort.
The analysis of this network leads to the following observations:

- Interpreting the size of the nodes, the three rodent species with the highest parasite diversity are occurring in human settlements. The three rodent species with the lowest parasite diversity occur in primary or secondary forest and dry agricultural land.

- The size of the human species node indicates that we share 15 parasite species with rodent species studied here.

- Interpreting the width of the edges at the network level, there is a higher density of large-width edges on the left of the network, indicating that rodent species in human settlements and rice-fields share a higher proportion of their parasite diversity than rodent species in the remaining habitats.

- Interpreting the width of the edges concerning the human species, Bi and Bs have the highest Jaccard index values (0.48 and 0.5 respectively), followed by Re, Rta and Rn (0.43, 0.42, 0.41 respectively).

- The nodes of Bi, Ran, Rn and Rta have the maximum number of edges (n=14) possible in this network. They all occur in human settlements, rice fields except for Ran occurring in primary forest. The nodes of Bb, Le and Mc have the lowest number of edges in the network (n=9) and they all belong to primary and secondary forest or dry agricultural areas.

- The node of the human species has 13 edges close to the maximum of 14.

This preliminary analysis of the EI network provides more information than a separate analysis of each parasite species and their hosts. The method of calculus of the index needs to be kept in mind: the observation that Bi and Bs have the highest Jaccard index values with the human species is irrelevant as the human species shares more parasite species with Rn and Rta than with Bi and Bs (11, 14, 10, 8 respectively). The difference is due to the high parasite
species richness of Rn and Rta. Other indices can be used to address this kind of issue but no index is perfect. However, this first network can orientate surveillance protocols towards the most interesting host species to be included in order to answer the question at stake: if the question is the probability of emerging infectious diseases in humans from rodent hosts in this ecosystem, the surveillance protocol will target species living in the human settlements (and a ranking can be done on this species) and in the rice fields with maybe Rti being an interesting sentinel species to look at as a bridge between pristine and modified environment. To our knowledge, this species is never mentioned as a potential source of infectious disease or as a potential sentinel for disease surveillance in the literature.

- End of the Box –
The temporal variability of the interaction between two host populations can also be obtained by molecular analysis on specific parasites in the different populations or longitudinal studies designed for detecting parasite seasonal profiles. An interaction network can change drastically between seasons as host contacts will vary with host movements depending on host ecology and resource availability (Brook and McLachlan 2009, Butt et al. 2009). This temporal dimension can be represented by different networks for different seasons.

**Scope and limitations of the approach**

We believe that the definitions and the framework presented in this paper can provide a solid basis to explore the ecology of disease transmission in multi-host systems as it disentangles the complex processes involved in transmission and provide further testable hypotheses. However, they are several limitations which should be kept in mind when interpreting epidemiological interaction networks.

First, host susceptibility to specific parasites is important to consider as it can blur the directionality of epidemiological interactions between two nodes/hosts’ populations. A parasite not shared by two populations could be the results of the host lacking susceptibility for this parasite. Co-evolved host-pathogen interactions result in a more stable network (in time) than recently created interactions. Most wildlife/domestic/human interfaces are the products of recent changes in human activities or behaviours (Daszak et al. 2000, Osofsky et al. 2005) and the newly established epidemiological interactions are possibly in an unstable state in time. Second, inter-parasite ecological interactions within hosts (e.g. direct competition or synergies or indirect through the host immune system) can influence epidemiological interactions networks although this is a poorly explored field of research (but
see Poulin 2005, Jolles et al. 2008, Lagrue and Poulin 2008, Telfer et al. 2010). These ecological interactions can provide another explanation for the lack of detection of a parasite in a susceptible host population: its elimination by direct or indirect competition by another parasite. Third, the performance of the diagnostic tests used need to be assessed: parasite isolation and antibody detection techniques do not give the same information about the past and present history of host-pathogen interactions. Whenever possible, this type of data should be harmonised across parasites.

Fourth, the variability in transmission modes across parasites in relation with EFGs will have a high impact on the network. Therefore the choice of the parasite species under study and the definition of EFG relevant to the transmission mode of this parasite will be crucial. This choice can be guided by knowledge of the parasite suspected to emerge and the life history traits of potential hosts in the ecosystem. RNA viruses are good candidates due to their implication in recent emergence (Cleaveland et al. 2007, Holmes and Grenfell 2009). If no a priori is made about the future emerging parasite, we suggest after building the global epidemiological interaction network, to provide subsets of this epidemiological interaction network based on transmission modes. The comparison of these networks can help identifying particular properties related to specific transmission modes. For a more holistic approach, the parasite choice should be oriented towards species representative of the different transmission modes in the ecosystem.

**EIDs at the wildlife/domestic/human Interface**

The common context of the wild/domestic interfaces from an ecological perspective is: a) a multi-host system, increasing in complexity as wildlife diversity increases; b) a multi-parasite system, increasing in complexity as wildlife diversity increases; c) the type of
interface (e.g. fence, area of contact), in expansion worldwide and creating a mosaic of contrasted natural and human-modified habitats. EIDs have recently captured the attention of media and scientific community (Cleaveland et al. 2007, Alexander and McNutt 2010). The recent steep increase in the power of technical (molecular) tools and the multiplication of emergence events in a globalised and changing world have increased the perceptions of EIDs as a threat for animal and public health. Several reviews have identified potential “hotspots” for parasite emergence (Jones et al. 2008, Woolhouse 2008) and underlined the linkages between human, domestic and wild parasites (Cleaveland et al. 2001, Taylor et al. 2001, Jones et al. 2008). Multi-steps processes have been presented to offer a mechanistic framework for emergence events (Woolhouse et al. 2005, Childs et al. 2007, Wolfe et al. 2007, Lloyd-Smith et al. 2009) sensu stricto. The emerging pathogen is detected in a target species with a variable time-lag between the inter-species transmission and the detection. This time-lag associated with ecological traits of the parasite and host will determine the severity of the outbreak. For instance, the time-lag for AIDS has taken probably several decades and maybe centuries from the first human case to the recognition of the disease at the beginning of the 80’s (Heeney et al. 2006, Holmes 2007b); for Ebola, the time-lag has often been short with massive localised human deaths (Leroy et al. 2009); finally for SARS both detection and spread have been quick (Rota et al. 2003). A smaller time-lag between interspecies spill-over and detection can save lives and limit the socio-economical impact of EID outbreak (Childs and Gordon 2009). From a scientific, ethical and economical point of view, research, surveillance, prevention and control should focus on EID hotspots in order to anticipate and prevent epizootics or epidemics potentially leading to panzootics and pandemics.

We believe that the epidemiological interaction networks can provide the basis for reducing the time lag between actual spill-over of pathogens and detection in EID hotspots. We propose a framework for the selection of hosts’ populations allocated to EFGs which
should be monitored in priority in a given hotspot. By identifying and quantifying epidemiological interactions between hosts’ populations, a risk can be attributed to each transmission pathways. Epidemiological interaction networks generate testable predictions of future parasite emergence, with direct implication for surveillance and control in a resource-limited environment. From a practical point of view, EID hotspots are usually located in remote areas of developing countries, economically poorly developed. Using already available sanitary information (e.g. from governmental veterinary services, NGOs) can provide the data to start building a network helpful in identifying gaps of knowledge or key hosts’ populations or parasites.

The EI network framework that we present here could achieve two objectives: increasing theoretical knowledge on the ecology of disease transmission and on multi-host multi-pathogen interactions and providing a tool for EID early detection. A crucial question in the ecology of disease transmission will be to determine if EFGs share common properties (see Box 7.1). Are there common transmission processes for parasites with different modes of transmission? And do hosts species play similar functional epidemiological roles for different parasites? If transmission processes in a given ecosystem share generic properties, these findings will have important consequences on animal and human health surveillance and control as resources could be more efficiently targeted for priority host populations and transmission chains.
Literature Cited


