

**Mistaken Matchings:
Imperfect Compatibility Information in Kidney Exchanges**

A thesis presented

by

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To

Applied Mathematics

in partial fulfillment of the honors requirements

for the degree of

Bachelor of Arts

Harvard College

Cambridge, Massachusetts

April 1, 2010

Abstract

The preferred treatment for patients with kidney disease is live-donor kidney transplantation. The prospective donors offering a kidney to a sick patient may not be compatible with the patient however; this creates an opportunity for kidney exchange, where incompatible pairs “swap” donors. Kidney exchanges benefit from scale but conducting real compatibility tests between all members of a large exchange pool is not feasible, so operating exchanges make swap allocations using virtual compatibility information and conduct real compatibility tests only at the time of transplantation. Unfortunately, virtual compatibility tests often result in false positives so allocated swaps cannot always be completed. This paper examines the effects of erroneous virtual compatibilities on exchange efficiency, incentives, and policy regarding nondirected altruistic donation. It concludes by considering a feasible and equitable method of erroneous-compatibility mitigation.

Acknowledgements: This paper benefited from the help and advice of many people. Particular thanks go to Alvin Roth and Itai Ashlagi, who introduced me to kidney exchange and whose ideas and criticism were crucial to the development of my analytical methodology. My simulations would not have been possible without the help of Michael Rees and Jonathan Kopke of the Alliance for Paired Donation, who provided an anonymized dataset as well as a first-hand understanding of the mechanics and problems of kidney exchange. Finally, thanks to Susan Saidman for explaining the immunology relevant to kidney exchange, and to M. Utku Ünver for our discussion regarding the importance of the problem I examine and the feasibility of implementing partial solutions.

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1 Introduction

Currently 84,109 Americans with end stage renal disease (referred to herein as **kidney disease**) are on the waiting list to receive a deceased donor’s kidney. In 2009, 4,476 wait-listed patients died waiting for a transplant and another 1,941 were removed from the waiting list because they were too sick to receive a transplant.¹ The preferred treatment for kidney disease is live-donor kidney transplantation ([4]): a healthy person has two kidneys and removing one is relatively safe,² so a friend or relative of a sick patient may offer to give the latter a kidney. Unfortunately the would-be donor’s blood is sometimes incompatible with that of the patient so a transplant is not possible. Such a patient and donor comprise an **incompatible pair**.

A particular incompatibility between the donor and patient of one incompatible pair does not necessarily preclude that patient from receiving a kidney from a different donor however, nor that donor from giving to a different patient. This means an opportunity for exchange arises: the first incompatible pair can “swap” donors with an appropriate second pair in the same situation, allowing both patients to receive transplants when originally neither could. This is **pairwise kidney exchange** and I depict 2-way and 3-way **cycle** exchanges in Figure 1. Only incompatible pairs are allowed to participate in kidney exchanges,³ so all of the pairs p_i in Figure 1 are incompatible, with a pair whose constituent patient is of blood type A and constituent donor is of type B denoted $A-B$; the patient and donor making up p_2 are blood type compatible but they still make up an incompatible pairing because of a **tissue-type** incompatibility.⁴ A directed line points in the direction of donation while an

¹From OPTN data (<http://www.optn.org/latestData/rptData.asp>) as of March 28, 2010; 2009 is the most recent year for which full data is available.

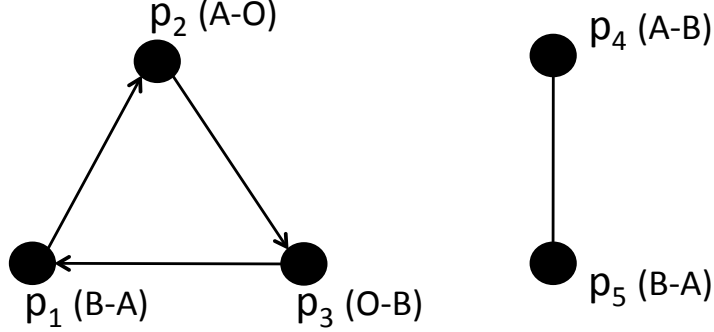
²Segev et al. [21] find the surgical mortality rate from live kidney donation to be approximately 3.1 per 10,000 donors.

³Though Roth et al. [16] find there would be significant benefits to allowing compatible pairs to participate.

⁴Incompatibilities arise because of incompatible blood types or because the potential recipient has antibodies to the potential donor’s blood antigens; I elaborate on this in Section 1.2.

undirected line means a first pair both gives to and receives a kidney from a second pair.

Figure 1: Pairwise kidney exchange



The theoretical and empirical benefits of paired kidney exchange are documented in the economics and medical literature (see [5],[6],[8],[13]). Simulation based estimates find that up to 60% of incompatible pairs may be matched through 2- and 3-way cycle exchanges like those in Figure 1 ([13]) and large-scale multi-hospital kidney exchanges have arisen to coordinate the swaps. The Alliance for Paired Donation (APD) is the most extensive exchange in operation with hospital partnerships in 27 states. Though the numbers of transplants resulting from paired exchange are still small, they have recently shown rapid increases from none in the year 2000 to 74 in 2006 and 300 in 2009. The evidence suggests that up to 1000 patients per year could benefit from a national exchange program in the United States ([20]).

The biotechnology required for kidney exchange has not caught up with the logistics however. Because of tissue-type incompatibilities, serum from a patient and lymphocytes from a donor must be physically mixed to definitively determine compatibility before a transplant can take place. Each such test requires a non-trivial quantity of blood so it is not feasible to exhaustively determine all compatibilities in an exchange with hundreds of participants. Even coordinating subsets of the test domain can be prohibitively complicated because incompatible pairs may be located at multiple hospitals across the U. S. and there is currently no centralized national

testing center. In lieu of using real compatibility data then, exchanges make allocation decisions based on computer-based **virtual compatibility** tests, which are then verified by real, physical compatibility tests just before transplantation.

The problem, and the motivation of this paper, is that virtual compatibility testing is highly inaccurate – the APD reports that certain types of allocations have an 80% chance of not being completable because of false virtual compatibilities. In other words, exchanges like those illustrated in Figure 1 are allocated by the kidney exchange mechanism on the basis of virtual compatibilities when, for example, in reality p_2 cannot receive p_1 ’s kidney or p_4 cannot give a kidney to p_5 . I call an incorrect virtual compatibility a **failure**. Failures have cascading effects on exchange outcomes and dynamics that have not been addressed in the literature. I outline the effects of failures that I consider in the next section.

1.1 Effects of false virtual compatibilities

The lack of accurate compatibility information is costly because it means patients in kidney exchanges may have to wait significant periods of time before being able to successfully participate in a paired exchange, and waiting is harmful to patients for several reasons. First, waiting is strongly associated with both decreased patient survival and decreased graft survival following kidney transplantation ([10]). Second, sometimes patients waiting for a kidney exchange receive a deceased-donor organ in the meantime. These are of lower quality than live kidneys ([19]), and giving one to a patient trying to participate in a kidney exchange means that wait-listed patients without incompatible paired-donors are forced to wait longer ([6]). Finally, the patients for whom virtual compatibilities are the least accurate are usually the most sick, and making these patients wait increases the likelihood that they will receive a blood transfusion or other medical care that increases their antibody load, further decreasing the accuracy of their virtual compatibilities. Segev et al. [18]

examine wait times within exchanges but their model does not allow for failures. I use simulation to compare the number of patients transplanted and their waiting times under the status quo of imperfect compatibility information versus the perfect compatibility information ideal in Section 2.⁵

In addition to affecting the number and wait times of patients taking part in paired exchange, imperfect virtual compatibility information also impacts incentives within an exchange. Kidney exchanges are partnerships of many hospitals, each participating for the good of its patients. Roth et al. [15] and Ashlagi [1] examine how even with perfect compatibility information, hospital incentives are not necessarily aligned with those of a match-maximizing exchange because a hospital with pairs that it can match internally may do so instead of revealing such pairs to the exchange at the risk of some of these pairs being left unmatched. Ashlagi [1] identifies ways of mitigating problematic incentives like this one, such as implementing an exchange mechanism that guarantees each hospital at least the same number of matches as the hospital can arrange on its own.

Ensuring that individual hospitals are incentivized to report their pairs truthfully is even more complicated when compatibility information is not perfect because matches are not equivalent to transplants. Different kinds of matches have different probabilities of success, and a hospital may want to try to make internal matches that it believes to be completable with high probability instead of allowing the exchange to make matches that may have a lower probability of being completable. A hospital might even decide to do all of its own internal testing first so that it knows with certainty whether or not it can complete internal matches before deciding which patients to report to a central exchange. This leads to less efficient matches and could even lead to a kidney exchange breaking down. I consider this problem in Section 3.

⁵This paper is focused on patient outcomes, but it should be noted that the costs born by medical providers due to delayed transplants are not trivial – most patients participating in kidney exchanges are on dialysis and, according to McFarlane et al. [9], the costs of conventional dialysis are on the order of \$70,000 a year.

Inaccurate compatibility information also affects policy decisions regarding the efficient allocation of longer chains of transplants called Non-Simultaneous Extended Altruistic-Donor (NEAD) chains, which are started when a nondirected altruistic donor⁶ donates a kidney to the patient of a first incompatible pair, whose donor donates to a second pair, and so on. Such chains of transplants can include more pairs than the cycle transplants described earlier because chain transplants need not be simultaneous. While cycle transplants must take place all at once because otherwise a donor could refuse to donate after his associated patient received a kidney (thus leaving another patient in the same cycle both without a transplant and without an associated donor), in a NEAD chain no pair gives up a kidney before it receives a kidney. This means chains have no pre-determined length since, in theory, additional pairs can always be added on, but it also means they can be prematurely terminated if a donor whose associated patient has received a kidney refuses to extend the chain; this is called a **renege**.

Reneges can only occur in a series of non-simultaneous transplants, so one way to reduce the possibility of reneges is to conduct chains as a sequence of shorter simultaneous segments bridged by non-simultaneous transplants. Donors connecting simultaneous segments are called **bridge donors**, and because all transplants resulting from a chain except those involving bridge donors take place simultaneously, only bridge donors have an opportunity to renege. Chains can still be unexpectedly broken through a bridge donor renege however, and this means there is an incentive for the exchange to stop the chain early – the mechanism can cut the chain short by only allowing a final pair to participate if the pair’s donor donates to the waiting list while his patient simultaneously receives the previous donor’s kidney. The mechanism can

⁶Technically all donors participating in kidney exchanges must be altruistic since non-altruistic donation is prohibited by federal law. The convention in the literature however has been to refer specifically to donors without associated patients as “altruistic donors,” although sometimes they are referred to more precisely as “nondirected donors.” Donors who are members of incompatible pairs are simply “donors.”

thus eliminate the possibility of a renege completely by only allocating one simultaneous segment that ends with a donation to the wait list; this is called Domino Paired Donation (DPD). The tradeoff the mechanism faces in deciding whether to allocate a NEAD chain or a DPD is between a chain of potentially arbitrary length (only potentially because there could be a renege) versus a shorter simultaneous “domino” of transplants that finishes with a donation to the waiting list, and has no chance of renege.

Previous analyses of this design have not allowed for failures. This matters because extended chains are robust to failure: even if an allocated simultaneous chain segment is unexpectedly shortened by a failure, a bridge donor remains, and he can extend the chain in later months (as long as he does not renege). A single simultaneous segment ending with a donation to the waiting list is similarly cut short by a failure, but it has no opportunity to grow. In Section 4, I use simulation to examine how nondirected donors should be allocated in the context of failures.

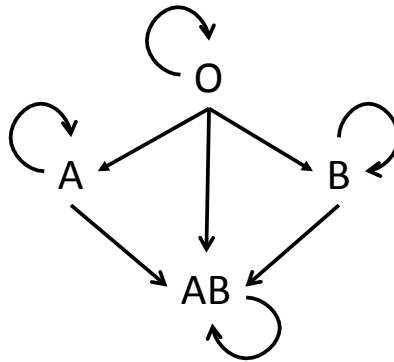
The APD has recently identified inaccurate compatibility information as a serious problem and is interested in implementable solutions. In Section 5, I use simulation to examine one possible solution where a few real compatibility tests are conducted before the exchange chooses matchings. The final matching is conducted over only known compatibilities and thus has no failures. I find that implementing such a policy is not equivalent to having perfect information, but that it increases the long run number of transplants and reduces average wait times among the transplanted.

1.2 Biological background

I briefly digress into biological terminology. A **blood type** refers to the presence of an inherited blood antigen. Types A and B refer respectively to blood containing A or B antigens. Type AB refers to blood containing both and type O has neither. A patient can only accept a kidney from a donor whose blood has the same or fewer

antigens than he has himself. A patient of type A cannot accept a kidney from a donor of type AB, for example. Type AB is sometimes referred to as the universal recipient and type O the universal donor. As mentioned earlier, a pair of type *A-B* means the patient has blood type A and the donor has blood type B. A patient and donor are said to be **ABO compatible** if they meet the blood type compatibility criteria depicted in Figure 2, where a patient of the blood type at the head of an arrow is ABO compatible with a donor of the type at the arrow's tail. ABO compatibility is easy to determine and is not the source of imperfect compatibility information.

Figure 2: ABO compatibilities



There is more to compatibility than ABO blood types, however. An individual's blood contains a vector of proteins that make up their Human Leukocyte Antigen (HLA) profile, and a patient is incompatible with a donor if the patient's blood serum contains antibodies to one or more of the donor's HLA antigens. Such a non-blood type incompatibility is called a **positive crossmatch**.⁷ A positive crossmatch is the tissue-type incompatibility from earlier; testing for a positive crossmatch is sometimes called **tissue typing**. A **virtual crossmatch test** is an online comparison of a patient's known antibodies and a donor's known HLA antigens, while a **crossmatch test** is the definitive (physical) test of serum and lymphocyte compatibility. A transplant candidate's **Panel Reactive Antibodies** (PRA) is a percentage measure of a

⁷There are a number of ways a patient can develop these antibodies, including exposure to someone else's HLA proteins through a blood transfusion. Mothers often exhibit a positive crossmatch with the father of their children because of an antigen transfer during childbirth.

patient’s antibody load weighted by the prevalence of corresponding antigens in the general population.

Tissue-type incompatibilities are the reason why kidney exchanges can facilitate transplants among all types of pairs. If no pairs were ever tissue-type incompatible, only pairs of type $A-O$, $B-O$, $A-B$, $B-A$, $AB-O$, $AB-A$, and $AB-B$ would be incompatible, and the only 2- or 3-way cycle exchanges that can be formed using these pairs are 2-cycles with one $A-B$ pair and one $B-A$ pair.

Unfortunately, tissue-type incompatibilities are the source of the erroneous virtual compatibility information, or failures, that motivate this paper. The reason for the incorrect virtual compatibilities is technological: there are thousands of possible antigens and corresponding antibodies, and existing technology for detecting those antigens or antibodies has a high false-negative rate. In other words, current testing methods do not always detect all antigens or antibodies, so the online comparison in a virtual crossmatch test may return that a patient and donor are compatible when that is not the case. Because a virtual crossmatch test that returns negative suggests true compatibility, failures are sometimes called **false negative crossmatches**.⁸

False negative crossmatches are rarely a problem for a patient with few antibodies because even if not all of these antibodies are detected, there is a low probability of a random donor’s blood containing the corresponding antigens. But they are a problem for patients with many antibodies: patients with high PRAs are said to be **highly sensitized** because they have a particularly high probability of crossmatch⁹ with a random donor and are thus highly susceptible to incorrect virtual compatibilities. The Organ Procurement and Transplantation Network (OPTN), a subsidiary of the U. S. government mandated United Network for Organ Sharing (UNOS), categorizes patients into 3 classes by PRA level: **low** PRA patients, who have a PRA equal to or less than 0.10; **medium** PRA patients, who have a PRA greater than 0.10 but less

⁸A “false negative crossmatch” always refers to an erroneous *virtual* compatibility.

⁹Unless I refer specifically to a negative crossmatch, “crossmatch” refers to a positive crossmatch.

than 0.80; and **high** PRA patients, who have a PRA greater than or equal to 0.80.

1.3 The mechanics of kidney exchange

As outlined earlier and depicted in Figure 1, the basic structure of a cycle exchange is a simultaneous kidney exchange among incompatible pairs where each pair receives and donates one kidney. The infrastructure costs of multiple simultaneous kidney removals and kidney transplantations are high – a surgical team and operating room is required for each patient and each donor of a participating incompatible pair – and the logistical coordination of the pairs themselves is difficult, so it is usually only feasible to execute paired exchanges with two or three pairs at a time. Such paired exchanges are respectively called 2- and 3-cycles.

Once a month, the APD allows new pairs to join its exchange pool. It then computes all of the pairwise virtual compatibilities and produces a matching that maximizes the number of pairs allocated to cycle exchanges, with priority given to some pairs with particular characteristics. The monthly population addition and matching allows sufficient time for real crossmatch tests to be conducted between matches so that pairs allocated to failed cycles today can still be part of the next match. UNOS has a proposal for a national exchange where matchings still occur monthly but new pairs are only allowed to join the exchange every 3 months ([22]). The motivation behind delaying population addition is that it might help avoid idiosyncratic matchings made in situations when few transplantable pairs are in the pool, and so result in higher quality matches. There is a cost implicit in delaying population addition however, because pairs seeking to join the exchange at unlucky times are forced to wait. Unver [23] implicitly touches on this policy choice when he derives a dynamically efficient mechanism, but he does not examine the secondary effects of dynamic matching and does not consider failures. I examine these effects in my simulations in the next section.

2 The losses due to false negative crossmatches

Long run kidney exchange behaviour is difficult to model analytically because patient populations are small and dynamic, so asymptotic extrapolations can be misleading. I thus turn to Monte-Carlo simulations to examine the effects of false negative crossmatches. In this section, I develop a clinically rigorous computer model by building on those described in Saidman et al. [17] and Gentry et al. [8], and use this model to estimate the loss in transplants and increase in transplanted patients' wait times due to the error rate of virtual crossmatch testing.

2.1 Simulation methodology

I proceed to explain my base case simulation in detail. Each simulation **instance** is a sequence of twenty-four periods. In each period I generate a population, execute a match-run, determine which matchings result in actual transplants, remove a small percentage of unmatched pairs from the exchange through simulated attrition, and then add the remaining pairs to the next period's exchange pool. I elaborate on this process below.

Population generation is accomplished by sampling with replacement from an anonymized APD dataset that contains patient and donor blood types, patient PRA levels, and the binary results of virtual compatibility tests for 340 real pairs. Patients are sometimes associated with more than one donor (57 patients were registered with two donors in the APD dataset) but I still refer to the grouping as a **pair**. In my base case, thirty incompatible pairs join the exchange pool every period. Table 1 presents summary statistics on the distribution of ABO types and PRA levels in the APD sample and Table 2 provides the approximate distribution of blood types and PRA levels in the general population ([7],[8]) for comparison. These distributions are different at least in part because only incompatible pairs join the exchange.

Table 1: Distribution of blood characteristics in the APD sample

	Blood Type				PRA level		
	A	B	AB	O	Low	Medium	High
% Recipients	24.6	13.3	2.5	59.6	43.2	18.8	37.9
% Donors	44.2	14.1	7.9	33.8			

Table 2: Distribution of blood characteristics in the general population

	Blood Type				PRA level		
	A	B	AB	O	Low	Medium	High
% Gen. Pop.	34	14	4	48	70	20	10

I use the APD’s virtual crossmatch information to determine all possible 2- and 3-way cycle exchanges, treating compatibilities as binary. I then determine which exchanges should be chosen to maximize the number of pairs participating in exchanges in the final match, constrained by the fact that a pair can only be part of one exchange that actually takes place. My specification of this optimization problem as a binary integer program is found in Appendix A. I use CPLEX optimization software to solve it, with ties broken randomly in the case of multiple solutions. The generation of possible exchanges and determination of the match concludes a **match-run**. Note that in my base case I do not give priority to cycles in which a donor has already been found to be truly compatible with a recipient, nor do I weight cycles by the PRA levels of their constituent patients, although I consider both of these possibilities later in this paper.

I proceed to simulate the real crossmatch test that occurs after matchings are made but before transplants take place. The probability that a patient of a given PRA level experiences a false negative crossmatch is based on the APD’s empirical rates of false negative crossmatch, which are detailed in Table 3. In the case of a crossmatch, the implicated pairs are listed as incompatible for the remainder of the simulation instance. Resampling from the APD sample is particularly advantageous for accurately simulating the effects of false negative crossmatches because I use

both the results of the APD’s virtual compatibility tests and the APD’s empirical distribution of false negative crossmatches in that same sample. In some tests I conduct sensitivity analysis on an **exogenous failure rate** which affects all patients and is not conditional on a patient’s PRA. This parameter, which has a value of 0 unless otherwise specified, captures the probability that extraneous factors other than a false negative crossmatch, such as someone suddenly getting sick or a patient receiving a blood transfusion, affect allocated exchanges.

Table 3: Distribution of false negative crossmatches at different PRA levels

PRA level	False Negative Crossmatch Rate
0.75 – 1.00	0.5
0.50 – 0.75	0.35
0.25 – 0.50	0.2
0 – 0.25	0.05

Cycles whose constituent pairs all pass the crossmatch tests are considered successfully transplanted and these pairs are removed from the kidney exchange pool. Remaining pairs are subject to a 2% rate of attrition, which is consistent with only approximately 10% of pairs surviving for 10 years without a match ([2],[7]). The next period begins with population generation.

I keep a number of factors consistent to mitigate extraneous variance when examining the effects of different policies within the same simulation. Though each policy’s population of pairs is kept separate throughout a simulation instance, the groups of pairs joining each pool at the beginning of a period are identical. I determine all true (as opposed to virtual) compatibilities when pairs are added so that real crossmatch testing between the same pairs in the population pools of different policies always returns the same result. Reported simulation results are the averages of 200 instances.

2.2 Simulation results

Table 4 compares the average number of transplants completed and the average wait times for transplanted patients for four simulated policies. Imperfect information means there are failures, as in the base case explained above, while perfect compatibility information means the exchange makes matches based on true compatibilities so all allocated matches result in transplants. Policies with perfect information are denoted **PI**. The **APD** policy is the APD’s current status quo where patients join the exchange every month, and the **UNOS** policy is the UNOS recommendation where pairs join the exchange every 3 months. In both cases (and all policies simulated in this paper), match-runs still occur on a monthly basis. For notational clarity, I refer to simulated policies in *italics*, so that *APD* refers to a specific simulated policy, while APD refers to the real Alliance for Paired Donation.

The **ratio of total TX¹⁰ to APD** is the ratio of the average of the total number of transplants completed in a given policy compared to the average of the total number of transplants completed in the *APD* policy with imperfect information. If the ratio is greater than 1, then the average number of transplants completed in a given policy was greater than the average number of transplants completed in the *APD* status quo. **Average wait (periods)** is the mean number of periods that patients who received transplants had to wait between the period they joined the exchange and the period in which they received a transplant. This statistic does not take into account the wait times, nor attrition (possibly death) of patients not receiving transplants. In keeping with the distribution in Table 3, “high PRA” refers to patients with a PRA greater than 0.75, and this convention is held throughout the remainder of this paper.

P-values for paired t-tests on the numbers of transplants completed under each policy compared to the numbers of transplants completed under *APD* are all less than 0.0001. This means that the increase in the average number of transplanted

¹⁰“TX” is an abbreviation for “transplants.”

Table 4: Perfect versus imperfect information

	PRA	Imperfect information		Perfect information	
		<i>APD</i>	<i>UNOS</i>	<i>APD-PI</i>	<i>UNOS-PI</i>
Ratio of total TX to <i>APD</i>	all	1.00	1.01	1.07	1.10
	high	1.00	1.02	1.13	1.20
Average wait (periods)	all	4.11	5.14	3.72	4.29
	high	5.05	6.11	4.41	4.94

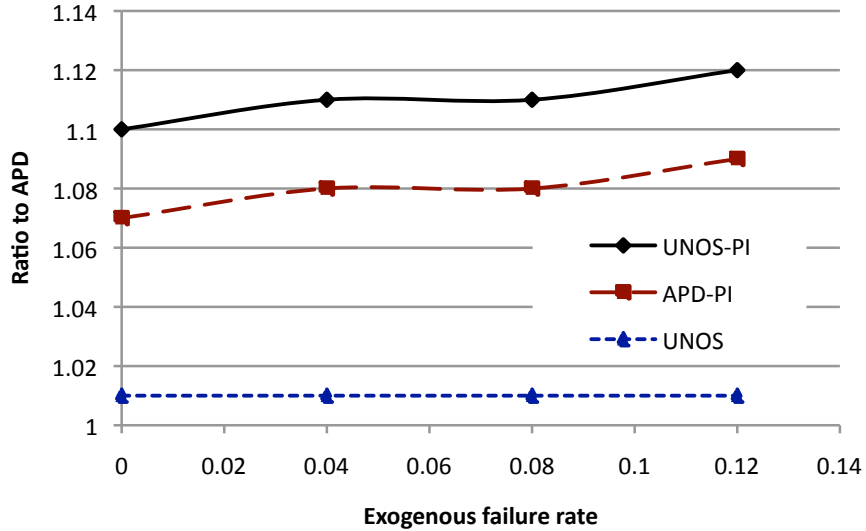
under the policies except *APD* over the average number of transplanted under *APD* is statistically significant. Comparing *APD* to *APD-PI* and *UNOS* to *UNOS-PI*, we see that perfect compatibility information leads to more transplants and shorter wait times for the transplanted. The return to having perfect information in terms of increased transplants is larger for high PRA patients than for the “all” PRA group.

Considering only the policies with imperfect information, *UNOS* yields marginally more transplants than *APD*, but increases average wait times among the transplanted considerably. We see a stronger tradeoff under the policies with complete information, as *UNOS-PI* has a larger average number of transplants than *APD-PI*, but also has higher average wait times among the transplanted. The high PRA group in particular benefits from more transplants in *UNOS-PI* over *APD-PI*; this makes sense because these patients are generally difficult to match so they are susceptible to idiosyncratic matches. That the difference between the policies with imperfect information is not the same as the difference between the policies with perfect information suggests that perfect information is not a good guide for making policy decisions about constraining when pairs can join the exchange. I return to the tradeoff between forcing patients to wait to join the exchange and letting them join every month below.

Varying the number of pairs added to the exchange every period between 20 and 40 changes the statistics presented in Table 4 by less than 1% and does not affect significance levels. Recall that exogenous factors such as sudden patient illness can also cause failures; I present sensitivity analysis on an exogenous failure rate in

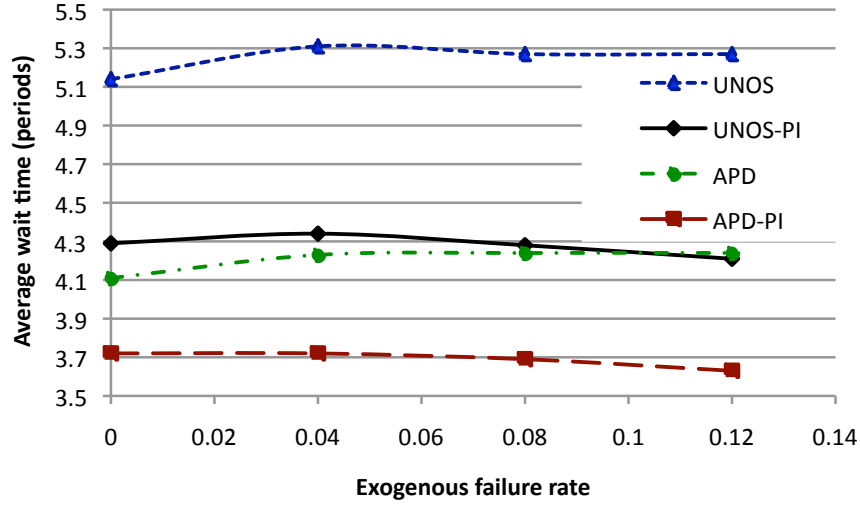
Figures 3 and 4. We see in Figure 3 that an exogenous failure rate increases the benefits of perfect information with respect to the total number of transplants under both policies. This is intuitive since a higher failure rate means there should be more to be gained by having perfect compatibility information. However, wait times, depicted in Figure 4, change negligibly with the exogenous failure rate. This suggests a higher failure rate means that fewer patients receive transplants and consequently that more patients never receive transplants. My results from Table 4 are robust with respect to an exogenous failure rate.

Figure 3: Effect of exogenous failure rate on total transplants



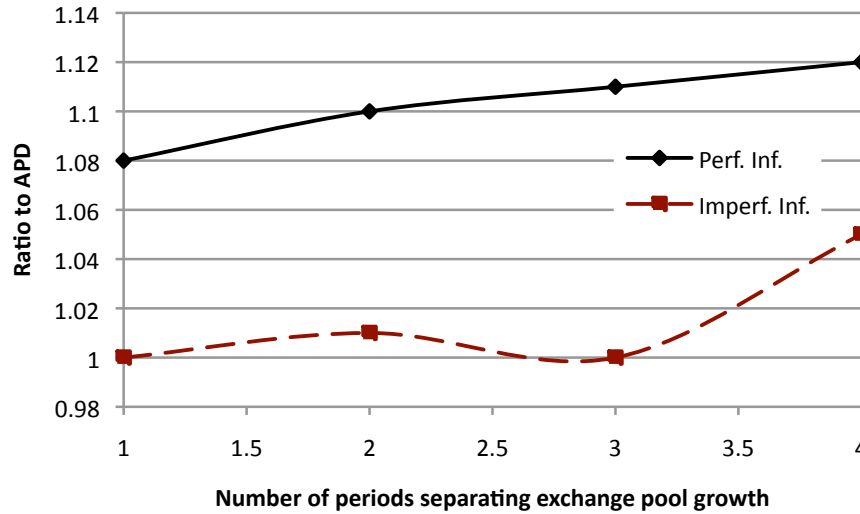
My final simulation in this section returns to the balance between wait times among the transplanted and exchange efficiency. Figures 5 and 6 present the results of a simulation on the effects of a mechanism that only allows pairs to join the exchange every 1, 2, 3, or 4 periods, under both perfect and imperfect compatibility information. Match-runs are still conducted every period. Note that the four policies presented in Table 4 are all contained within this simulation – allowing pairs to join every third period with perfect compatibility information is equivalent to the *UNOS-PI* policy, for example. In the first period I add 120 pairs (instead of the 30 pairs in my base case) to each policy’s population pool so that each policy’s first match-run

Figure 4: Effect of exogenous failure rate on waiting times



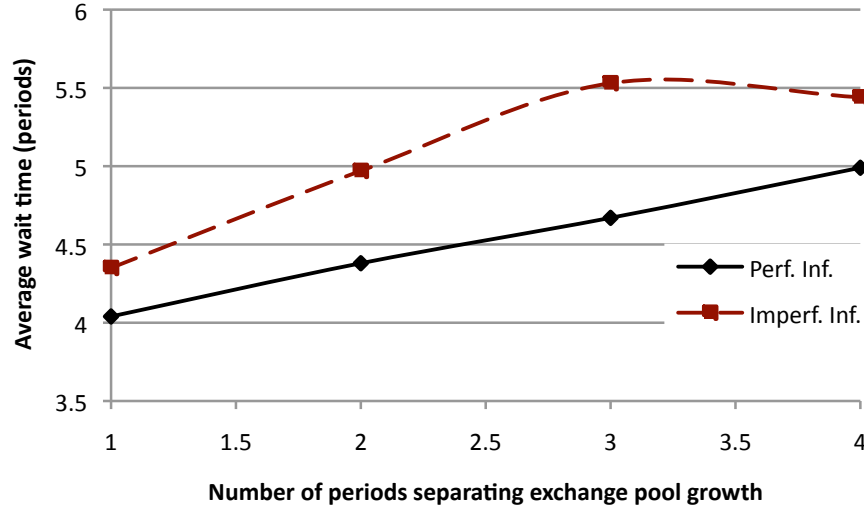
is executed over the same pool. This means the exact statistics presented in these graphs are slightly different from those resulting from the same policies in Table 4.

Figure 5: Effect of forced waiting on total transplants



We see from Figure 5 that forcing pairs to wait longer increases efficiency under both perfect and imperfect information, confirming earlier results. Figure 6 shows that making pairs wait 3 or 4 periods instead of 1 to join the exchange increases the average wait among the transplanted – under imperfect information, the average

Figure 6: Effect of forced waiting on waiting times



transplanted patient's wait is a period longer when pairs join every third period instead of every period. This is also similar to earlier results, but notice that the trends for the policies under imperfect information are slightly different from those for the policies under perfect information. This demonstrates that it is indeed important to take failures into account when examining the effects of different kidney exchange policies.

The results presented in this section show that imperfect information is worse than perfect information both in terms of numbers of patients transplanted and the transplanted patients' wait times. Drawing a conclusion regarding how often an exchange should allow pairs to join is not straightforward, however. There is an important tradeoff between decreasing average wait times for the transplanted and increasing the number of transplants completed. The wait times I present can be misleading because they do not capture the waiting (or death) of patients not transplanted. Although there is an attrition rate in my simulations, the numbers of transplants I present are not equivalent to patient outcomes and in particular they do not take into account how a transplanted patient's outcome decreases with waiting. Extreme mechanisms are not necessarily appealing: a mechanism seeking to minimize wait

times for the transplanted might only match patients who joined the exchange in a given period, for example. Further work to capture the costs of waiting among both the transplanted and untransplanted is required.

3 Incentive implications of failures

The case for improving the accuracy of virtual compatibility information is not merely justified by simulation results on numbers of transplants and patient wait times. As noted by Roth et al. [15] and Ashlagi [1], it is important to take hospital incentives into consideration when designing an exchange mechanism or else a hospital that can match pairs on its own may do so instead of revealing its pairs to the exchange at the risk of fewer pairs being matched. This would likely be suboptimal for the exchange since a larger pool of pairs can only lead to higher quality matches.¹¹ Ashlagi [1] develops an incentive compatible kidney exchange mechanism in an effort to avoid just this situation, but he does not take false negative crossmatches into account. In the following section, I outline how these false negative crossmatches can undermine the alignment of incentives required for an efficient kidney exchange.

3.1 A kidney exchange game

I proceed to construct a generalized game of a single period kidney exchange where hospitals are players that choose which pairs they report to a centralized exchange.

I start by developing a mechanism for making kidney exchange allocations on a single population. Let A be a set of incompatible pairs. A **mechanism** $\mathcal{M}(\cdot)$ is responsible for executing match-runs on a set of incompatible pairs based on virtual compatibilities.¹² Recall that executing a match-run means first computing the set

¹¹Recall that the potential benefits of conducting match-runs over a larger population pool motivated making patients wait to join the exchange in the *UNOS* policy in Section 2, and that this policy facilitated more transplants than the *APD* status quo.

¹²This was accomplished by solving an integer program in the simulations in Section 2.

of all possible 2- and 3-way cycle exchanges, and then choosing the subset of these cycles that maximizes the number of pairs matched, constrained by the requirement that no two cycles in the chosen subset can contain the same pair. Formally, \mathcal{M} is a function that takes as input a set of incompatible pairs and returns the set of cycles that maximizes the number of disjoint matches involving 2- and 3-way exchanges. Ties are broken randomly. I call E_A the set of cycle exchanges returned by executing the mechanism on the set A , so that

$$\mathcal{M}(A) = E_A. \quad (1)$$

E_A is said to constitute a **matching**. I sometimes write a cycle exchange belonging to a matching as an ordered tuple of pairs, so that the original cycle exchanges denoted in Figure 1 would be respectively written (p_1, p_2, p_3) and (p_4, p_5) .

I now extend this mechanism to a context in which there are many hospitals. Let H_i denote the set of incompatible pairs belonging to **hospital** i . Each hospital i strategically chooses to report a subset of its pairs $\sigma_i \subseteq H_i$ to an impartial entity called a **kidney exchange**. A kidney exchange **pool** P is the set of all pairs reported by the n hospitals participating in the kidney exchange, so that

$$P = \{\sigma_1, \dots, \sigma_n\} = \bigcup_i \sigma_i. \quad (2)$$

The kidney exchange authority takes the pool P and executes the mechanism on it, returning $\mathcal{M}(P) = E_P$. Hospitals also conduct their own match-runs on unreported pairs,

$$\mathcal{M}(H_i \setminus \sigma_i) = E_{H_i \setminus \sigma_i}. \quad (3)$$

Let m_i denote the total number of hospital i 's pairs matched, and notice that

$$m_i = |E_P \cap H_i| + |E_{H_i \setminus \sigma_i}|. \quad (4)$$

\mathcal{M} makes matchings based on virtual compatibilities which are not necessarily accurate. Let $p \in [0, 1]$ be the probability that a given virtual compatibility is erroneous, and call p the **failure rate**. Because there is a positive failure rate, it is necessary to conduct real crossmatch tests on all allocated exchanges before transplants can go forward. These crossmatch tests are only conducted once, so no secondary match-run over the pool of failed and previously unmatched pairs can take place.¹³ Formally, let $\mathcal{F}(\cdot)$ be a function that represents the process of real crossmatch testing. If A is a set of pairs, then \mathcal{F} takes as input the mechanism's matching on A , and outputs F_A , the set exchanges that can proceed to transplantation:

$$\mathcal{F}(\mathcal{M}(A)) = \mathcal{F}(E_A) = F_A. \quad (5)$$

Call T_i the set of patients at the i th hospital that receive transplants, and note that

$$T_i = \mathcal{F}(E_P) \cap H_i + \mathcal{F}(E_{H_i \setminus \sigma_i}) = F_P \cap H_i + F_{H_i \setminus \sigma_i}. \quad (6)$$

It must be that the number of a particular hospital's pairs matched is at least equal to the number of that hospital's pairs receiving transplants, so

$$m_i \geq |T_i| \quad \forall i. \quad (7)$$

This concludes the development of my notation.

The literature generally assumes that all hospitals report truthfully, but I am interested in situations in which some hospital conceals at least one pair, or formally in situations where there exists at least one hospital j such that $H_j \setminus \sigma_j \neq \emptyset$. In order to isolate incentives to conceal, I construct a worst case scenario where:

¹³This is how the APD's kidney exchange works – pairs allocated in cycles that fail cannot be matched until the next month's match-run because the coordination of required crossmatch tests takes too much time.

Assumption 1. *All hospitals have complete population information. That is, each hospital is aware of all pairs belonging to all hospitals, $\bigcup_i H_i$, and the respective pairwise virtual compatibilities of all these pairs before hospitals choose which pairs to report to the kidney exchange.*

Assumption 1 is not likely to be precisely true in real exchanges, but hospitals do know at least the approximate distribution of other pairs in the exchange as well as the approximate probabilities of external matchings. So that computing hospital strategies is straightforward, I additionally assume that:

Assumption 2. *Each hospital seeks to maximize the **expected number** of its own patients transplanted.*

Assumption 2 means the i th hospital's problem of choosing which pairs to reveal is equivalent to deciding which subset $\sigma_i \subseteq H_i$ to report in order to maximize the expected number of hospital i 's pairs transplanted. By Assumption 1, the i th hospital is aware of the strategy sets of other hospitals, so hospital i 's expected transplants are conditional on the realization of what other hospitals strategically choose to report, which I denote $\hat{\sigma}_j$ for all $j \neq i$. Recall that there is an exogenous probability p that each virtual compatibility is erroneous. This means formally,

$$\sigma_i = \arg \max_{\sigma'_i: \sigma'_i \subseteq H_i} \mathbb{E} \left[|T_i| : p, \sigma'_i, \hat{\sigma}_j, \forall j \neq i \right] \quad (8)$$

where \mathbb{E} is the expectation operator. Note that if $p = 0$, then each hospital's expected value-maximization problem is a match-maximization problem because all matches result in transplants if there are no failures. For clarity, I summarize the progression of my kidney exchange game in 4 steps:

1. **Strategizing.** Each of the n hospitals chooses which pairs it will report to the kidney exchange, σ_i for $i \in \{1, \dots, n\}$, to maximize the expected number of i 's

pairs transplanted given the virtual compatibilities of all pairs at all hospitals and a probability of failure p .

2. **Matching.** Each hospital conducts an internal match-run on pairs not reported to the kidney exchange, returning $E_{H_i \setminus \sigma_i} = \mathcal{M}(H_i \setminus \sigma_i)$ for the i th hospital, and the kidney exchange conducts a match-run on the pool of pairs reported from all hospitals $P = \bigcup_i \sigma_i$, returning $E_P = \mathcal{M}(P)$. The number of the i th hospital's pairs matched is m_i .
3. **Crossmatching.** The required real crossmatch tests are conducted on all cycles of matched pairs, returning $F_{H_i \setminus \sigma_i} = \mathcal{F}(H_i \setminus \sigma_i)$ for pairs matched inside the i th hospital and $F_P = \mathcal{F}(E_P)$ for the kidney exchange. No secondary or “repair” matches are considered over the failed or unmatched pairs.
4. **Transplantation.** The set of incompatible pairs from the i th hospital participating in paired exchanges is finally realized as T_i .

I introduce metrics for evaluating efficiency in the context of this game and then apply the game's framework to three specific examples in the next section.

3.2 Winners and losers

A match is said to be efficient if it results in the largest possible number of transplants on any set of pairs A . I formalize this. A match E_A^* is **efficient** if, for any other match E on A such that $E_A^* \neq E$,

$$|\mathcal{F}(E_A^*)| \geq |\mathcal{F}(E)|. \quad (9)$$

Efficient matches are straightforward to compute in the absence of failures since if $p = 0$ then every allocated matching is equivalent to a transplant, so the efficient match over a set of pairs A is simply $E_A^* = \mathcal{M}(A) = E_A$ by the specification of \mathcal{M} as a match-maximizing mechanism. This is not true when $p > 0$ however, because

when it is not necessarily true that $\mathcal{F}(E_A) = E_A$, there may exist some match E'_A that results in more transplants than E_A . Equivalently, E_A is not efficient if there exists E'_A such that $|\mathcal{F}(E'_A)| > |\mathcal{F}(E_A)|$. If we know which virtual compatibilities are erroneous before the match, thus having perfect compatibility information, we can find the efficient match, E_A^* , by solving

$$E_A^* = \arg \max_{E: E \subseteq \mathcal{S}(A)} |\mathcal{F}(E)|, \quad (10)$$

where $\mathcal{S}(A)$ is the set of all possible 2- and 3-way cycle exchanges on the set of pairs A . This is precisely the efficient match that I simulated in Section 2.1; I compared this efficient match, which can only be computed under perfect information, to the match produced by \mathcal{M} with imperfect information and found that $|\mathcal{F}(E_A^*)| > |\mathcal{F}(E_A)|$ with statistical significance.

Efficiency is more complicated in the context of a kidney exchange made up of many hospitals that may not report their pairs truthfully. When $p = 0$, the efficient match is just $\mathcal{M}(\bigcup_i H_i)$. If some hospital j conceals a pair however that would have been a part of the efficient match, so that $(H_j \setminus \sigma_j) \cap (\mathcal{M}(\bigcup_i H_i)) \neq \emptyset$, then the match produced by $\mathcal{M}(\bigcup_i \sigma_i) = \mathcal{M}(P)$ may not be efficient ([1]). The efficiency loss can be even more pronounced when $p > 0$ because hospitals may have an additional incentive to conceal: the presence of a failure rate means a hospital is concerned with the number of transplants expected to result from the match, and the expected number of transplants depends on the size of the match's constituent cycles.

I formalize the incentive to conceal. When $p = 0$, a mechanism \mathcal{M} is not **strategyproof** if there exists a pool of reported pairs $P = \bigcup_i \sigma_i$ and a hospital j such that when hospital j honestly reports $\sigma_j = H_j$, and the mechanism allocates $E_P = \mathcal{M}(P)$, hospital j receives fewer matches than if j reports some $\sigma'_j \subset H_j$ and matches concealed pairs internally. In other words, a mechanism is not strategyproof if there

exists a hospital j and some $\sigma'_j \subset H_j$ such that

$$|\mathcal{M}(P') \cap H_j| + |\mathcal{M}(H_j \setminus \sigma'_j)| > |\mathcal{M}(P) \cap H_j| \quad (11)$$

where $P' = \{\sigma'_j, \bigcup_{i \neq j} \sigma_i\}$ and $P = \{H_j, \bigcup_{i \neq j} \sigma_i\}$.

Hospitals are concerned with expected transplants instead of just matches when there is a positive probability of failure $p > 0$. My definition of strategyproofness extends naturally to such a context, so that a mechanism is not strategyproof if there exists a hospital j and some $\sigma'_j \subset H_j$ such that

$$\mathbb{E}[|\mathcal{F}(\mathcal{M}(P')) \cap H_j| + |\mathcal{F}(\mathcal{M}(H_j \setminus \sigma'_j))|] > \mathbb{E}[|\mathcal{F}(\mathcal{M}(P)) \cap H_j|] \quad (12)$$

where again $P' = \{\sigma'_j, \bigcup_{i \neq j} \sigma_i\}$ and $P = \{H_j, \bigcup_{i \neq j} \sigma_i\}$. Notice that this is a generalized notion of strategyproofness because it collapses to the first when there are no failures: if $p = 0$, then $E_P = \mathbb{E}[\mathcal{F}(E_P)]$. I illustrate how strategyproofness is difficult to ensure in the following examples.

Example 1 (Failures in expectation). *Consider an exchange made up of two hospitals, $A = \{a_1, a_2, a_3\}$ and $B = \{b_1\}$, with virtual compatibilities depicted in Figure 7. An undirected line $x_i - x_j$ means x_i can donate to x_j and vice versa, while a directed line $x_i \rightarrow x_j$ means x_i can donate to x_j but not the reverse. I use dashed lines to illustrate that these compatibilities are not true compatibilities so failures may occur.*

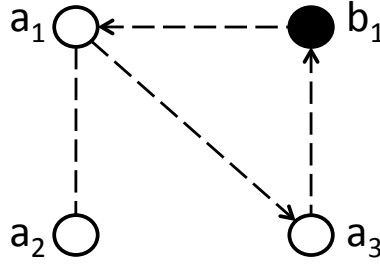
Hospitals A and B are faced with the decision of which pairs to reveal. Assume first that B reveals b_1 so $\sigma_B = \{b_1\}$. Then A has to decide how to specify $\sigma_A \subseteq \{a_1, a_2, a_3\}$. If A reveals all of its nodes so that $\sigma_A = A$, then the kidney exchange pool is $P = A \cup B$ and the mechanism \mathcal{M} will maximize matchings by allocating the 3-cycle $\mathcal{M}(P) = E_P = (a_1, a_3, b_1)$. This makes the exchange's expected number of transplants $\mathbb{E}[|\mathcal{F}((a_1, a_3, b_1))|] = 3 \cdot (1 - p)^3$, and in particular A 's expected number of transplants is $\mathbb{E}[|T_A| : \sigma_A = A, \sigma_B = B] = 2 \cdot (1 - p)^3$. Hospitals A and B are

reporting all their respective pairs so there are no internal matches.

Now consider what happens if A conceals all its nodes so that $\sigma_A = \emptyset$. The kidney exchange pool contains only B 's pair so $P = \{b_1\}$ and the resulting match is empty $\mathcal{M}(P) = \emptyset$. Hospital A executes an internal match-run on its concealed pairs however, and $\mathcal{M}(A \setminus \sigma_A) = \mathcal{M}(A) = E_A = (a_1, a_2)$. This gives A an expected number of transplants $\mathbb{E}[|T_A| : \sigma_A = \emptyset, \sigma_B = B] = \mathbb{E}[|\mathcal{F}((a_1, a_2))|] = 2 \cdot (1 - p)^2$.

It is immediate that $2(1 - p)^2 > 2(1 - p)^3$ for any positive failure rate p . Thus A is better off concealing, as opposed to revealing, all its pairs in the case when B reveals b_1 . Symmetric analysis yields that it is weakly dominant for A to conceal all its pairs for all other possibilities of σ_A, σ_B . This means \mathcal{M} is not strategyproof.¹⁴

Figure 7



Example 1 shows a particular case of misaligned hospital and exchange incentives. Ashlagi [1] tries to align hospital incentives in the absence of failures by designing individually rational mechanisms. Without failures, a mechanism is individually rational if each hospital is guaranteed as many matchings in the final allocation as it could match internally among the set of pairs it reports.¹⁵ Formally, a mechanism \mathcal{M} is **individually rational** (IR) when $p = 0$ if executing \mathcal{M} over a pool P of all reported pairs returns an allocation such that for every hospital i with $i \in \{1, \dots, n\}$, at least as many of hospital i 's pairs are matched as hospital i can match on its own

¹⁴This is a single period model so I do not consider the dynamics of inter-temporal strategies.

¹⁵While I assume that all hospitals are aware of all other hospitals' pairs $\bigcup_i H_i$ when they strategize, the exchange authority is only aware of the pairs reported by hospitals $\bigcup_i \sigma_i$, so it can only make guarantees based on that information set.

within its reported set. Equivalently, \mathcal{M} is IR if

$$\left| \mathcal{M} \left(\bigcup_j \sigma_j \right) \cap H_i \right| \geq |\mathcal{M}(\sigma_i)| \quad \forall i. \quad (13)$$

This definition is not compelling in a context in which virtual compatibilities may be erroneous because it is unclear how to quantify the value of matches when matches do not necessarily lead to transplants. In particular, the IR mechanism guarantees the number of matchings and not the expected number of transplants. Thus, a natural extension of the IR mechanism to a context with failures is a mechanism that guarantees each hospital the expected value of transplants the hospital can facilitate on its own reported pairs. I formalize this. Define a mechanism \mathcal{M} to be **individually rational in expectation** (IRE) if executing \mathcal{M} on the set of all reported pairs P returns an allocation such that for every hospital i with $i \in \{1, \dots, n\}$,

$$\mathbb{E} \left[\left| \mathcal{F} \left(\mathcal{M} \left(\bigcup_j \sigma_j \right) \right) \cap H_i \right| \right] \geq \mathbb{E} [|\mathcal{F}(\mathcal{M}(\sigma_i))|]. \quad (14)$$

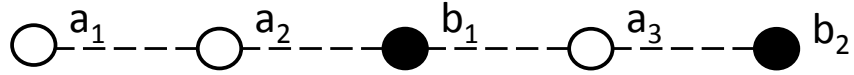
Consider the IRE mechanism in the context of Example 1. A obtains an expected $2(1-p)^2$ transplants by matching its pairs internally, so if A reports all pairs truthfully then the mechanism is forced to choose the matching (a_1, a_2) . It is straightforward to see that hospital A is never incentivized to conceal any of its pairs. This does not mean the IRE mechanism is strategyproof in general, however, as we see in the following example.

Example 2 (IRE strategyproofness counter-example). *Consider an exchange made up of two hospitals, $A = \{a_1, a_2, a_3\}$ and $B = \{b_1, b_2\}$, with virtual compatibilities depicted in Figure 8. The exchange mechanism \mathcal{M} is IRE. It is immediately apparent that B always reports truthfully: B cannot make any internal matches so it cannot receive more transplants in expectation by concealing, no matter what A reports.*

If A reports truthfully so that $\sigma_A = H_A$, then the IRE mechanism knows A can do 2 internal matches, so it has to choose a match such that A has an expected $\mathbb{E}[|T_A|] = 2(1-p)^2$ transplants. There are multiple possible allocations $E = \mathcal{M}(P) = \mathcal{P}(A \cup B)$ that satisfy this constraint. Each of these allocations contains the same number of matches for the exchange – two 2-cycles – so \mathcal{M} picks one randomly. Suppose \mathcal{M} chooses $E = \{(a_2, b_1), (a_3, b_2)\}$. Then A receives precisely an expected $\mathbb{E}[|T_A| : \mathcal{M}(A \cup B) = E] = 2(1-p)^2$ transplants.

Now suppose A conceals a_1, a_2 because it can match them internally. This means the mechanism sees only $P = \{b_1, a_3, b_2\}$ so it need not make any guarantees. The IRE mechanism can thus choose either $E' = \{(b_1, a_3)\}$ or $E'' = \{(a_3, b_2)\}$. Hospital A receives one match in either of E' or E'' , and combining the expected value of this match with the expectations of A 's internal matchings, we see that A receives an expected $\mathbb{E}[|T_A| : \sigma_A = \{a_3\}, \sigma_B = B] = 1(1-p)^2 + 2(1-p)^2 = 3(1-p)^2$. Because $3(1-p)^2 > 2(1-p)^2$ for any $p > 0$, A is better off concealing. This gives us that the IRE mechanism is not strategyproof.

Figure 8



I have extended some of Ashlagi [1]'s methodologies for aligning incentives to a context within which there is imperfect compatibility information, and shown that the IRE mechanism is not necessarily an improvement. I conclude this section with one final example that reveals another negative possibility.

As I described earlier, one of the reasons why the APD conducts only a single match-run every month is because it takes time to coordinate all the crossmatch tests necessary to check whether allocated cycles will proceed to transplantation, and the APD wants failed cycles to be able to take part in the next match-run.

Crossmatch tests need not occur only after the match-run however, because it may be feasible for a hospital to conduct its own internal matching and crossmatching before strategizing. If a hospital first conducted crossmatch tests on its own pairs, it would know their real (internal) pairwise compatibilities before deciding which pairs to report to the exchange. In other words, such a hospital would have no internal failure rate. Assuming all hospitals execute internal crossmatch testing changes the sequence of my kidney exchange game so that there is a new first step:

0. **Internal crossmatching.** A pairwise compatibility is equivalent to a fragment of a 3-cycle, so I call the j th pairwise compatibility among the pairs in the i th hospital $c_{i,j}^{(internal_compat)}$. Each hospital i conducts real crossmatch tests on all such pairwise compatibilities, $\mathcal{F}(c_{i,j}^{(internal_compat)}) \forall j$.

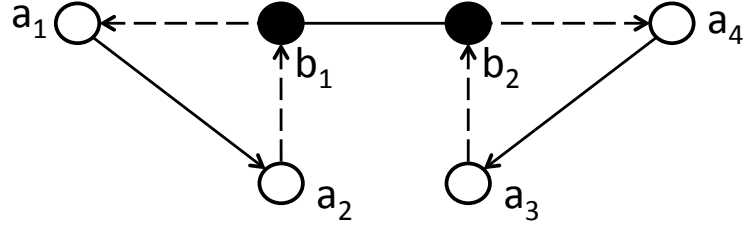
The steps (1) – (4) still proceed normally after step (0), except that now the part of step (3) in which each hospital conducts the crossmatches for its internal matches is redundant. The next example demonstrates how the addition of step (0) can affect efficiency.

Example 3 (Hospitals with internal testing). *Consider an exchange consisting of two hospitals, $A = \{a_1, a_2, a_3, a_4\}$ and $B = \{b_1, b_2\}$, with virtual compatibilities depicted in Figure 9. I make the same assumptions as in Examples 1, 2 except that hospitals conduct crossmatch testing on their own pairs at the beginning of the game, so that each hospital’s true internal compatibilities are known before they choose which pairs to report to the exchange. Solid lines represent true compatibilities with no failure rate and dashed lines represent virtual compatibilities subject to the failure rate p . Assume that \mathcal{M} seeks to maximize the number of pairs matched in exchanges, so \mathcal{M} is not concerned with individual rationality nor individual rationality in expectation.*

Hospital A can do no matching on its own so it always reports $\sigma_A = A$ truthfully. Hospital B must choose whether or not to reveal b_1 and b_2 . Reporting nothing so

that $\sigma_B = \emptyset$ means B matches both pairs (b_1, b_2) internally, which gives B precisely $\mathbb{E}[|\mathcal{F}(b_1, b_2)|] = 2$ transplants. Alternatively, if B reports truthfully, then the match-maximizing \mathcal{M} will pick $E = \mathcal{M}(A \cup B) = \{(a_1, a_2, b_1), (b_2, a_4, a_3)\}$. This allocation gives B an expected $\mathbb{E}[|\mathcal{F}(E) \cap B|] = 2(1 - p)^2$ transplants. Since $p > 0$, and thus $2 > 2(1 - p)^2$, B will conceal both pairs.

Figure 9



The IRE mechanism could be used to choose matches in Example 3. This would prevent B from needing to conceal its pairs because if B reported $\sigma_B = B$ then an IRE mechanism would need to guarantee 2 transplants in expectation to hospital B , so the matching would have to be $\{(b_1, b_2)\}$. This still results in only 2 transplants, when 6 could take place if all pairs were matched in 3-cycles and no failures occurred in the crossmatching phase.

Hospitals see clear benefits from conducting crossmatch tests on their own internal pairs. If hospitals do conduct internal crossmatching then they will always put priority on their own internal exchange cycles because these exchanges have higher expected values than exchanges subject to failures. The APD's mechanism is not IR, nor does it take failures into account; it relies instead on hospitals playing the kidney exchange game truthfully. The results in this section suggest that hospitals can have strong incentives to conceal for the benefit of their own patients. This does not build confidence in the future of large-scale kidney exchange.

3.3 Exchange incentives

I conclude my analysis of the incentive implications of failures with an examination of the incentives of the exchange mechanism itself. If the objective of an exchange is to maximize the total number of transplants, a mechanism that maximizes matchings based on virtual compatibilities is naive because it does not take patient PRAs, and thus associated probabilities of failure, into account. This means a kidney exchange seeking to maximize the number of transplants might not implement a mechanism that merely maximizes the number of pairs matched.

One way an exchange might increase the number of patients receiving transplants is to implement a mechanism that maximizes the sum of **weighted matchings**, where the “weight” of a particular cycle exchange refers to the expected number of transplants it will facilitate. The specifics of the new implied optimization problem are detailed in Appendix B. It is immediately apparent that such a mechanism discriminates by patient PRA, so it is not equitable and some kinds of patients will likely be worse off. The loss in equity may be counteracted by an increase in efficiency however; I return to my simulation framework to examine how such a mechanism compares to the APD’s status quo. Let **weighted** be a simulation policy where the objective of each match-run is to maximize weighted matchings. Table 5 compares the results of *weighted* to *APD* and *APD-PI*, which are the same as before.

Table 5: Weighted matchings

	PRA	Imperfect information		Perfect information
		<i>APD</i>	<i>weighted</i>	<i>APD-PI</i>
Ratio of total TX to <i>APD</i>	all	1.00	1.03	1.07
	high	1.00	0.95	1.13
Average wait (periods)	all	4.11	3.46	3.72
	high	5.01	4.13	4.41

The *weighted* policy yields 3% more transplants overall than the APD’s status quo. However, in discriminating by probability of success, and thus patient PRA

levels, *weighted* decreases the average number of high PRA patients transplanted by 5%. Average wait times for the transplanted among both the all PRA group and high PRA group are better under *weighted* than *APD*; this makes sense because only cycles that have a relatively high probability of success are chosen by the match, and choosing only such cycles should reduce the wait times of patients actually receiving transplants. Comparing the ratio of total TX to *APD* of *weighted* to that of *APD-PI*, we see that having perfect compatibility information still makes a notable difference.¹⁶

Weighted results in lower wait times among transplanted patients (including high PRA) than *APD-PI*. This shows the cost of discriminating by PRA because, along with the fact that considerably fewer high PRA patients receive transplants under *weighted* than under *APD-PI*, it demonstrates how under the *weighted* policy, patients with particular characteristics receive transplants quickly after joining the exchange, while the rest are effectively doomed to never receive transplants. Weighting by probability of success thus acts as a proxy to minimize wait times among the transplanted, and this returns to my discussion in Section 2 of the tradeoff between efficiency and wait times among the transplanted. The *weighted* policy improves both efficiency and wait times among the transplanted overall, at the cost of ignoring a particular group of patients who are difficult to transplant. In summary, this simulation says that a kidney exchange can increase the number of patients receiving transplants while simultaneously reducing the average wait time of a transplanted patient by deliberately transplanting fewer high PRA patients. This is not necessarily surprising but it is an important tradeoff to understand because patient equity is a contentious issue in the kidney exchange literature.¹⁷

¹⁶Note that the *weighted* policy with perfect compatibility information is equivalent to *APD-PI* since under perfect information, the probability of any cycle's success is always 1, so the weights are the sizes of the cycles: $w_i = |c_i| \forall i$.

¹⁷There is a debate for example about the ethics of "list exchange," where a patient in an incompatible pair receives priority on the deceased-donor waiting list after his associated donor donates a kidney to a patient on the deceased-donor waiting list. The problem is O-type patients on the deceased-donor waiting list may be harmed by allowing such exchanges (see [24]).

I have shown that the presence of a failure rate means that there is a balance between efficiency and incentive compatibility in kidney exchange mechanism design. I examine one final policy decision in the context of failures in the next section.

4 NEAD chains versus domino paired donation

Failures play an important role in how altruistic donation fits into the framework of kidney exchange. I proceed by reviewing and elaborating on the policies described earlier for allocating nondirected donors, and then compare them within my simulation framework.

An altruistic **nondirected donor** (NDD) (recall that they are said to be “nondirected” because they are without an associated patient) can donate directly to the deceased-donor waiting list but this means they only facilitate one transplant. As described earlier, another option is for an NDD to catalyze a series of transplants called a Non-Simultaneous Extended Altruistic-Donor (NEAD) **chain**, where the NDD donates a kidney to the patient of a first incompatible pair, whose donor may then donate to a second pair, and so on.

Exchanges previously considered in this paper have been restricted by a **simultaneity requirement**: cycle transplants must take place simultaneously or else a donor might renege on his commitment to donate after his associated patient has received a kidney but before he has given up one of his own. A mechanism allowing for such a possibility would probably not be sustainable. As mentioned earlier however, chains need not be simultaneous because no pair in a chain gives away a kidney before receiving a kidney. Thus no incompatible pair is irreparably short-changed in the event that a previous donor in the chain **reneges**, or refuses to extend the chain with his donation – the chain simply stops and pairs after the renege return to the exchange pool. Non-simultaneity means chain transplants can take place over

an extended period of time and can include more incompatible pairs than cycle exchanges,¹⁸ which in turn allows chains to include pairs with characteristics that make it difficult for them to take part in a cycle exchange. If it becomes clear that a chain cannot be continued – there are no incompatible pairs that can receive a transplant from the donor of the last pair on the chain – the chain can be terminated by having the last donor donate to the deceased-donor waiting list.¹⁹

There is always a possibility that a donor in a non-simultaneous chain of transplants will renege however, and a renege prematurely terminates the chain and forfeits potential future chain links. As described in Section 1.1, the mechanism can always stop a chain early by requiring the terminal pair’s donor to give a kidney to the waiting list while his associated patient simultaneously receives a kidney. This means that, at the very least, a renege means the loss of a donation to the waiting list, so a renege means an efficiency loss of at least one transplant. In order to help minimize renegees, chains are typically conducted as a series of simultaneous segments joined together by the non-simultaneous donation of **bridge donors**. All transplants except those facilitated by bridge donors take place simultaneously, so bridge donors are the only donors with an opportunity to renege. Although chains can offer advantages, the possibility of a bridge donor renege means they can result in fewer transplants than simultaneous exchange. The following example illustrates this possibility.

Example 4 (Chain renegeing). *Consider an exchange composed of one hospital with the compatibility graph in Figure 10. All crossmatch testing has been completed so there is no possibility of failure. Let \mathcal{M} be a match-maximizing mechanism that considers 2- and 3-way cycle exchanges, as well as chains of any length.*

\mathcal{M} will choose the 6-pair chain beginning with the NDD and passing through $p_1, p_2, p_3, p_4, p_5, p_6$. Because only segments of length 2 and 3 can be completed si-

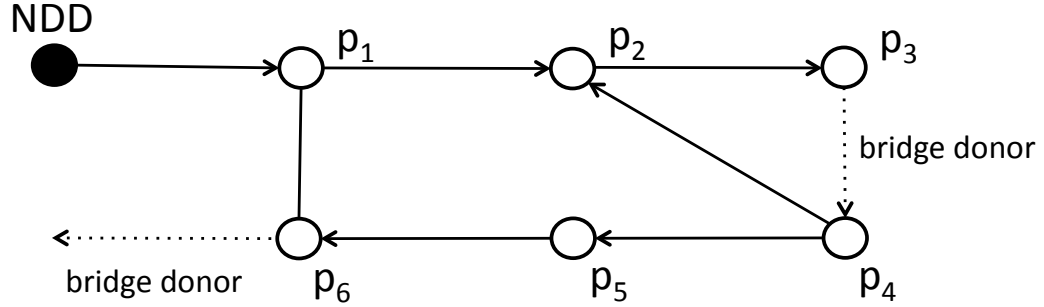
¹⁸Rees et al. [12] report a chain of 10 transplants taking place over 8 months.

¹⁹Given the length of the deceased-donor waiting list, the literature generally assumes that there is always at least one patient waiting who is compatible with a given donor.

multaneously, \mathcal{M} breaks this chain into two segments, p_1, p_2, p_3 and p_4, p_5, p_6 , which are joined by p_3 's donor, who is thus a bridge donor. p_6 's donor is also a bridge donor, and is asked to wait for the possibility of extending the chain in future periods.

If the NDD is not included, \mathcal{M} instead chooses 2 cycle exchanges: (p_1, p_6) and (p_2, p_3, p_4) . Ex ante, the chain will result in 6 transplants conditional on p_3 not reneging, while the cycles result in 5 transplants with certainty. Worst case ex post analysis of the chain shows that if p_3 's donor reneges, then only 3 transplants will take place. This means the chain could result in only $\frac{3}{5}$ as many transplants as the cycles. Further, if p_3 's donor reneges, there are no remaining cycles that could later be matched in a repair solution.

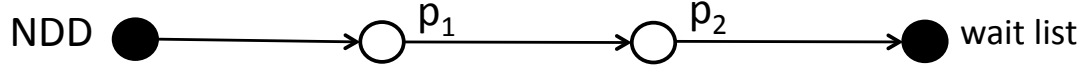
Figure 10: A NEAD chain



That lives are at stake in transplantation makes mitigating risk attractive, and this might mean that allowing any opportunities for reneging is not desirable. The incentive to eliminate reneges has made **Domino Paired Donation** (DPD) popular, a policy where chains are terminated by a donation to the waiting list after just one simultaneous segment.²⁰ Figure 11 shows the structure of a DPD. Gentry et al. [7] assert through simulation that a mechanism allocating DPDs of up to 2 pairs and a final donation to the wait list (as in Figure 11) as well as 2- and 3-cycles produces more transplants in the long run than a mechanism allocating chains and 2- and 3-cycles when the probability of reneging is as high as 5%.

²⁰DPD was first considered as a way to allocate NDDs in Montgomery et al. [11].

Figure 11: A DPD



Gentry et al. [7]’s simulations do not consider failures however, and this is important because failures affect how DPDs compare to longer chains. Recall that one failure in a cycle means the entire cycle must be abandoned. Chains are comparatively robust to failure because when there is a failure in a chain, only the planned transplants after the point of failure cannot be carried out and, unless there is also a renege, a bridge donor remains so the chain can still grow in future periods. A DPD is likewise unlikely to be completely abandoned because of a failure, but a failure will mean it is shorter than is merely required for simultaneity, so in practice a DPD may be very short indeed. In summary, even if failures make chain segments short in any individual period, over time chains can grow to include many segments, and this means they can become much longer than DPDs. I proceed to compare the long run benefits of chains versus DPDs in the context of failures.

4.1 An expanded simulation model

Chains are inter-temporal by design and hard to model analytically, so I proceed with analysis through simulation. I first expand on my earlier simulation model to include chains and DPDs. The APD dataset contains anonymized virtual compatibility information for 32 NDDs. In my base case, one NDD drawn randomly with replacement from this sample joins the kidney exchange every period. Adding one NDD to the exchange every period is realistic given that the APD had 32 NDDs in its first 30 months of operation.

It is not possible to optimize across periods and often the chain chosen to maximize matchings within a given period ends with a bridge donor of a blood type that makes it difficult to extend the chain in later periods, so I do not allow the match mechanism

to choose chains that end in bridge donors of type AB.^{21,22} Bridge donors are subject to a **renege rate**, which I set to 1% in my base case.²³

Gentry et al. [7] only allowed intra-period chain segments to consist of 3 transplants so that all the transplants within a segment could be completed simultaneously. Increasing the maximum chain length adds exponential complexity to the matching problem, so my simulations only consider chains that consist of up to 4 pairs in a given period. Not considering chains longer than 4 pairs means my simulation results are biased towards DPD because longer intra-period chains are allowed in the APD.

I call the policy in which I allow chains to consist of up to 3 pairs in a given period **S-NEAD** and I call the policy in which chains may consist of up to 4 pairs in a given period **L-NEAD**. Simulating both of these policies gives me a sense of how much even longer chains might have to offer. Since the 4 transplants allocated in a chain of length 4 cannot take place simultaneously with current infrastructure, I break such chains into two segments of length 2 joined together by a **quasi-bridge donor**. The quasi-bridge donor is subject to an **inter-renege rate** that I set to half the normal renege rate, or 0.5%.²⁴ My **DPD** policy allows DPDs to consist of up to 2 pairs from the exchange pool, with the final pair donating to the deceased-donor waiting list. A DPD can thus maximally consist of 3 transplants, so all DPD transplants take place simultaneously. I still allow 2- and 3-way cycle exchanges in all policies.

²¹These still may occur if an AB donor is embedded inside a chain segment and they exhibit a false negative crossmatch with the patient allocated to receive their kidney.

²²The APD does not allocate chains with AB bridge donors because they did so once and, as of December 2009, the bridge donor had been waiting to donate for 22 months.

²³As of December 2009 the APD had never experienced a renege in over 30 months of operation with 15 bridge donors.

²⁴The APD similarly breaks long chains up into multiple simultaneous segments that are bridged over consecutive days.

4.2 Simulation results²⁵

I first attempt to reproduce Gentry et al. [7]’s result that a policy allocating chains consisting of up to 3 pairs in a given period results in fewer transplants than a policy allocating DPDs when the renege rate is higher than 5%. Since the principle difference between my model and theirs is that I include failures, I fix my failure rate at 0 for all PRA levels in this simulation. I depict my results in Figures 12 and 13. Figure 12 shows the sensitivity of ratio of the average of the total number of transplants completed under *L-NEAD* and *S-NEAD* policies compared to the average of the total number of transplants completed under *DPD* to the renege rate. This ratio is calculated the same way as before. Figure 13 is from the same simulation but shows the proportion of simulation instances in which the *L-NEAD* and *S-NEAD* policies respectively produced more transplants than *DPD*. This graph lets us visualize the statistical significance of simulation results by depicting how often more patients received transplants under the chain policies than under *DPD*.²⁶

We see in Figure 12 that *S-NEAD* is very close to *DPD* at a renege rate of 5% and is worse than *DPD* at higher renege rates. This is confirmed in Figure 13 because the percentage of instances in which *S-NEAD* yielded more transplants than *DPD* is just above 50% for a renege rate of 5%, and this percentage decreases at high renege rates. Examining *L-NEAD*, we see from Figure 12 that *L-NEAD* is approximately 3% better than *S-NEAD* at all renege rates and that *L-NEAD* only becomes comparable to *DPD* at high renege rates. This result is mirrored by the significance levels in Figure 13. The relationship shown here between *S-NEAD* and *DPD* is very similar to Gentry et al. [7]’s findings. I do not consider these graphs to accurately depict how a real exchange would operate because this simulation ignores the effects of false negative

²⁵The simulation results presented in this section are part of a working paper entitled “Nonsimultaneous Chains and Dominos in paired Kidney Exchange – Revisited” with Itai Ashlagi, Alvin Roth, and Michael Rees.

²⁶For example, if *L-NEAD* produced more transplants than *DPD* in 180 of the 200 simulation instances, then the proportion of better instances would be 0.9.

Figure 12: Gentry et al. [7] reproduction: effect of renege rate on ratio to *DPD*

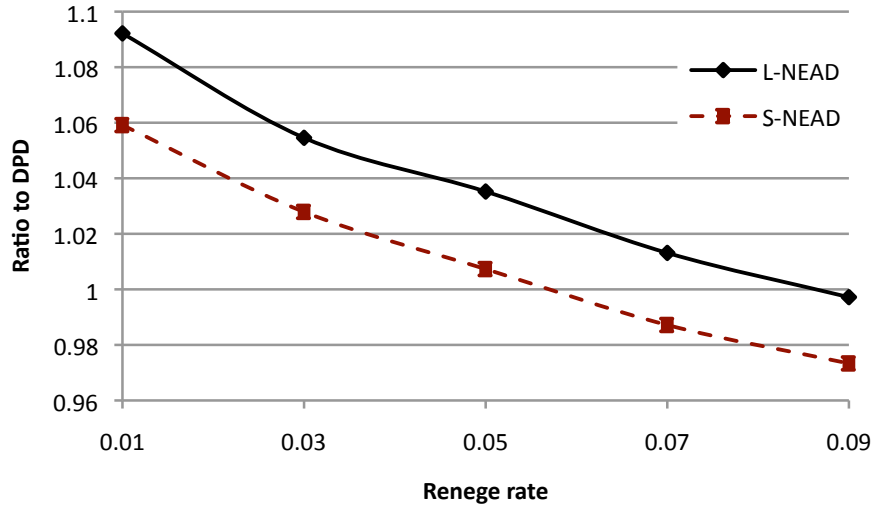
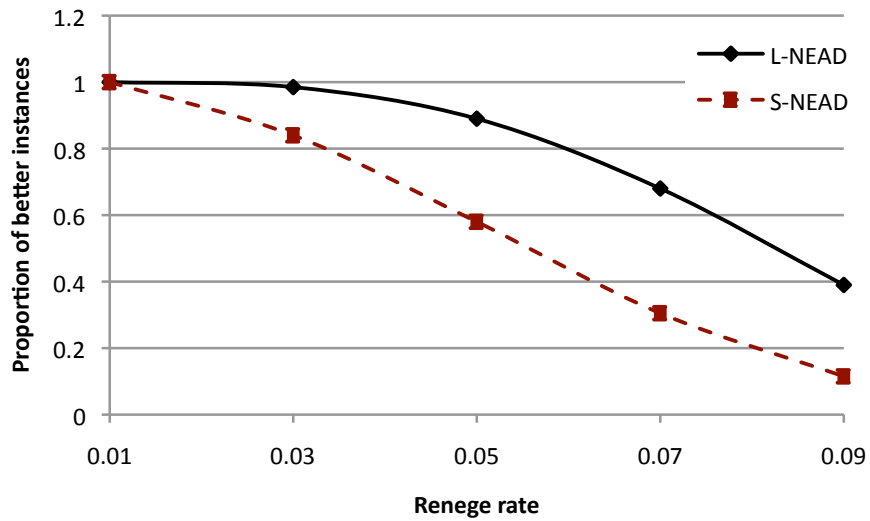


Figure 13: Gentry et al. [7] reproduction: significance of renege rate



crossmatches. The graphs do show encouraging results however, because we see that even without failure rates, *L-NEAD* has a convincing advantage over *S-NEAD* and, more importantly, over *DPD*.

I now return to my base case that includes failure rates. The addition of chains and DPDs to my original simulation model adds a number of new parameters. The following 6 graphs show sensitivity analysis on the renege rate, inter-renege rate,

exogenous failure rate, and the number of pairs added to the exchange every period.

Figures 14 and 15 depict sensitivity analysis on renege rates and inter-renege rates. We see in Figure 14 that *S-NEAD* and *L-NEAD* are better than *DPD* for low renege rates and that they are still at least at least 9% better than *DPD* at a renege rate of 5%. This suggests that although the renege rate has an important effect on the total number of transplants completed in the chain policies, the effect of the renege rate when failures are taken into account is smaller than without failures.

Figure 14: Effect of renege rate on ratio to *DPD*

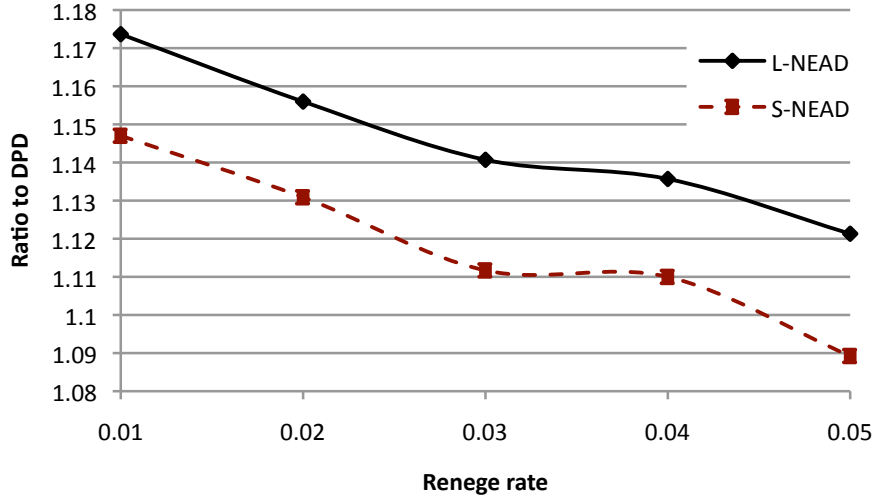
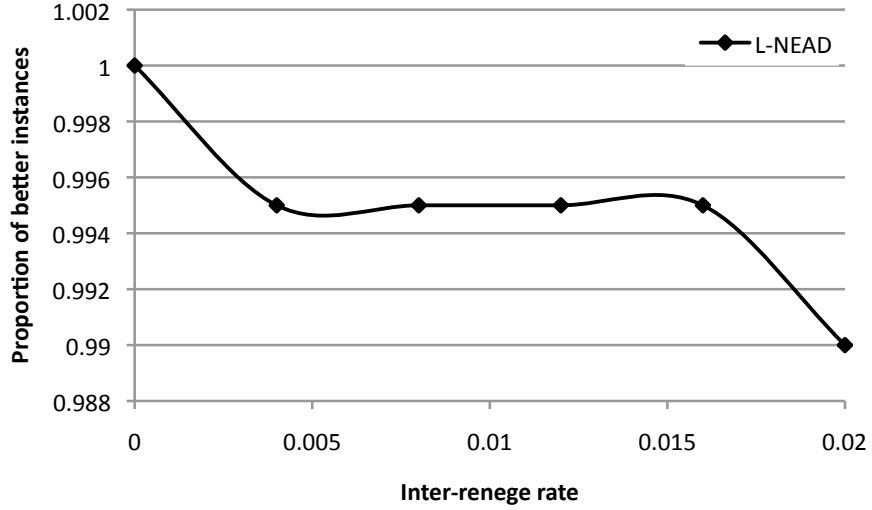


Figure 15 shows the proportion of instances in which *L-NEAD* was better than *DPD* for different inter-renege rates. (*S-NEAD* is not shown because *S-NEAD* has no quasi-bridge donors and thus no inter-renege rate.) The normal renege rate is set at 2% in this simulation so that it is never exceeded by the inter-renege rate. Changing the inter-renege rate has only a negligible effect; we see that even with an inter-renege rate of 2%, *L-NEAD* was better than *DPD* in 99% of instances. I thus do not depict the ratio of transplants under *L-NEAD* compared to those under *DPD* while changing inter-renege rate because this ratio shows little change.

Figures 16 and 17 examine the effects of the exogenous failure rate on how *L-NEAD* and *S-NEAD* compare to *DPD*. Figure 16 depicts how an exogenous failure

Figure 15: Significance of inter-renege rate, comparing L -NEAD to DPD



rate affects the ratio of total transplants under S -NEAD and L -NEAD versus DPD . We see that the exogenous failure rate appears to help L -NEAD and S -NEAD as compared to DPD . This makes sense: the robustness to failure of chains is more pronounced at higher failure rates. Figure 17 shows how the simulation instances compare – for all exogenous failure rates tested, both chain policies were better than DPD in at least 99% of all simulation instances.

Figure 16: Effect of exogenous failure rate on ratio to DPD

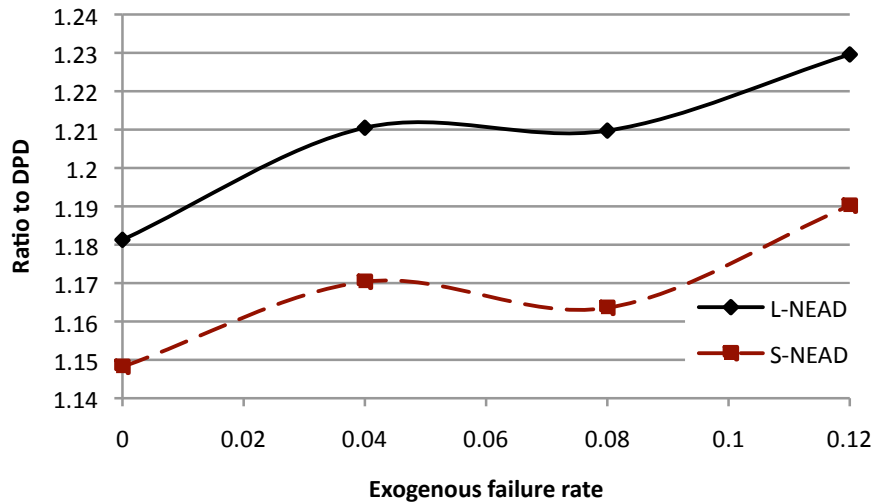
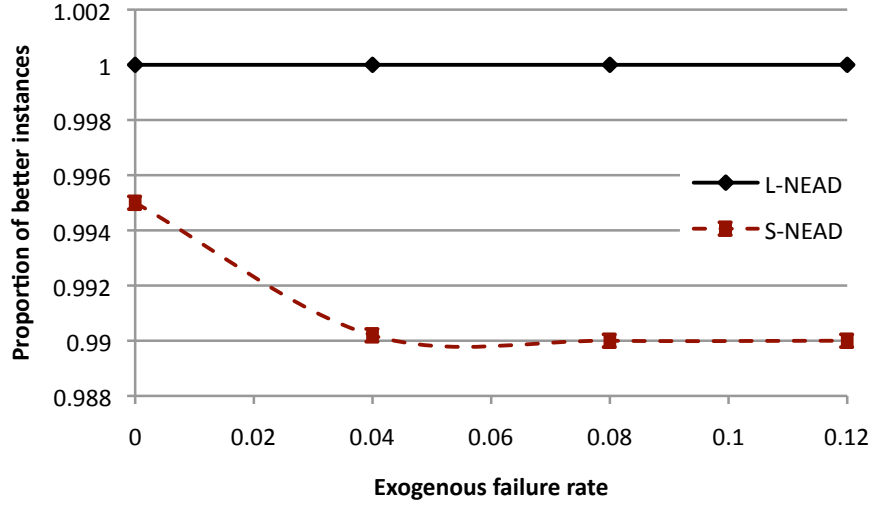


Figure 17: Significance of exogenous failure rate as compared to *DPD*



My last graphs in this section, Figures 18 and 19, depict sensitivity analysis on the number of pairs added to the exchange every period. Figure 18 shows that the chain policies are better than *DPD* for wide ranges of population growth. That chains offer less improvement over *DPD* in terms of total transplants as exchange population size increases is not surprising because an increase in the pool size corresponds to a decrease in the ratio of the number of NDDs to the number of pairs – in a large population, the vast majority of transplants will take place as result of cycle exchanges so the relative significance of comparing the chain policies with *DPD* is reduced.

Figure 19 is different from my other simulations because it compares the chain policies to a policy in which NDDs give directly to the waiting list so that there are no chains or *DPDs* and the exchange only allocates transplant cycles. I call this new policy **cycles-only**, and run this simulation to examine the importance of chains in facilitating transplants in general. The pattern here is similar to the one Figure 18, supporting my previous reasoning regarding why the benefits of chains over *DPD* are smaller in large populations. We see however that both chain policies offer large increases in numbers of transplants over *cycles-only*.

We have seen in this section that the inclusion of failures makes the chain policies

Figure 18: Effect of population growth on ratio to *DPD*

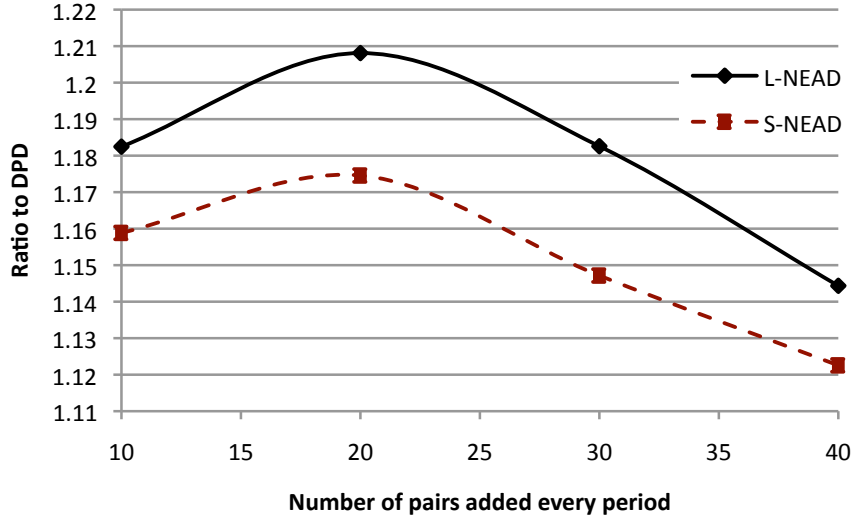
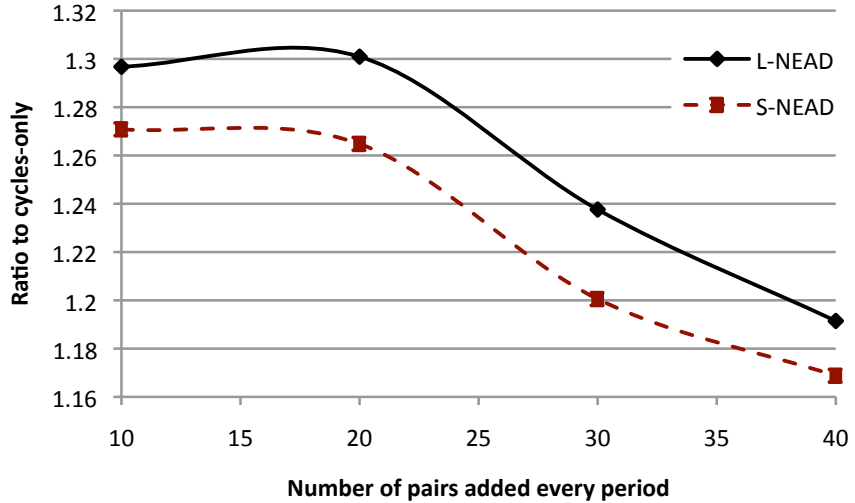


Figure 19: Effect of population growth on ratio to *cycles-only*



compare more favourably to *DPD* than suggested in Gentry et al. [7]. We have also seen that optimizing over chains of length 4 instead of length 3 makes a considerable difference in terms of total patients transplanted. That even longer chains are considered in practice suggests that the benefits of chains over *DPD* shown in my simulations are understated. The inclusion of failures in my simulation suggests that the policy decision between allocating chains versus *DPDs* is straightforward, but

it remains clear from my earlier analyses that failures are detrimental to exchange participants. I continue in the next section by considering a way of mitigating the effects of failures.

5 A market designer’s response

Completing exhaustive crossmatch testing before the match-run is not possible because every crossmatch test requires a sample of each donor’s blood to be sent to each potential recipient’s hospital, and there are simply too many possible matches for this to be feasible. The resources needed for one as opposed to several crossmatch tests do not scale linearly however. A single crossmatch test requires a certain amount of blood from the potential participants on both sides of a transplant, but three or four crossmatch tests can be performed with that same sample. This means that although comprehensive pre-match crossmatch testing is not feasible, if an exchange used a centralized crossmatch testing centre, it might be possible to execute the relevant crossmatch tests for some small subset of all possible matches. These crossmatch tests would all be conducted simultaneously, and then it would be possible to execute the match-run using only real (as opposed to virtual) compatibilities, so there would be no failure rate. Since the APD believes that implementing a centralized pre-match crossmatch testing centre is feasible, I proceed to examine which pre-match tests should be run and the magnitude of the benefits of such testing.

5.1 Pre-match crossmatch test selection

Determining which real crossmatch tests to conduct before the match is challenging because we seek to choose the appropriate subset of all possible tests (based on virtual compatibilities) so that ex post, after the tests are simultaneously completed, we have the required real compatibility information to allow the match-run to choose

the efficient match. A naive way to choose pre-match crossmatch tests is to optimize repeatedly over all possible exchanges using the APD’s match-maximization criterion, assuming before consecutive optimizations that previously chosen cycles resulted in failures. After executing several such optimizations to some testing **depth**, the exchange would have a list of cycles and implied crossmatch tests to run before conducting the true match-run.

I assume that optimizing to a depth of 4 is realistic given that about 4 crossmatch tests can be run with a single blood sample, and proceed to simulate two variations of this testing policy. In **Cycle-Failure Depth 4** (CFD4), I optimize 4 times and assume each time that all previously chosen cycles failed. This means cycles chosen by the match in consecutive optimizations have not been chosen before. In **Pairwise-Failure Depth 4** (PFD4), I also optimize 4 times but this time I assume that previously chosen cycles failed because each pairwise compatibility inside each chosen cycle resulted in a failure. Thus, cycles containing pairwise allocations that were previously chosen are never part of subsequent pre-match optimizations in *PFD4*. This differs substantially from *CFD4*, where I made no assumptions about which pairs caused previously chosen cycles to fail. Consider the following example for clarity.

Example 5 (*CFD4* vs *PFD4*). *Assume there is an exchange composed of 3 pairs, $P = \{p_1, p_2, p_3\}$, and virtual compatibility information suggests each pair can participate in a 2-way exchange with any other pair. This means that there are 3 possible 2-cycles – (p_1, p_2) , (p_1, p_3) , and (p_2, p_3) – and 2 possible 3-cycles – (p_1, p_2, p_3) and (p_1, p_3, p_2) . Recall that the mechanism under both *CFD4* and *PFD4* maximizes matches, so both policies choose the 3-cycles, one after the other, over the first two optimizations. In the third optimization, *CFD4* might choose the 2-cycle (p_1, p_2) , while *PFD4* would not because *PFD4* would have assumed (p_1, p_2, p_3) failed and in particular that all pairwise exchanges were no longer possible. Indeed, *PFD4* would terminate after two*

pre-match optimizations while CFD_4 would continue to allocate each 2-cycle in turn (until reaching a depth of 4) before turning the results of the pre-match optimizations over to the centralized testing centre.

That PFD_4 ignores all previously chosen pairwise allocations means it should lead to a more diverse set of pre-match tests than CFD_4 , since CFD_4 might choose similar allocations repeatedly. The policies should produce different results and give some indication as to whether testing should be more pessimistic, assuming that all pairwise compatibilities fail, or more optimistic, assuming that only specific cycles fail.

5.2 Simulation results

Table 6 details my simulation results. APD and $APD-PI$ are the same as before. It is visually clear that the results of CFD_4 and PFD_4 are not very different from one another, and indeed the difference in total transplants is not statistically significant. The p-values on paired t-tests of the total number of transplants completed under the pre-match testing policies compared to the total number of transplants completed under the APD status quo are less than 0.0001 however, so these pre-match testing policies offer a statistically significant 5 or 6% average improvement over APD . Notice in particular that, on average, both pre-match testing policies increase the number of high PRA patients transplanted by 9%. This is an encouraging result because it shows that the pre-match testing policies are effective in helping those who are most prone to failure. Both pre-match testing policies also reduce average wait times for the transplanted.

The similarity of the results for CFD_4 and PFD_4 suggests that most of the pre-match tests conducted by both policies are redundant however. Indeed, sensitivity analysis on testing depth for both pre-match policies shows that increasing depth from 1 to 5 affects the average total number of transplants by no more than 1% while more

Table 6: Pre-match testing

	PRA	Imperfect information			Perfect information
		APD	CFD_4	PFD_4	$APD-PI$
Ratio of total TX to APD	all	1.00	1.05	1.06	1.07
	high	1.00	1.09	1.09	1.13
Average wait (periods)	all	4.11	3.94	3.93	3.72
	high	5.05	4.73	4.72	4.41

than doubling the number of pre-match tests conducted. Further sensitivity analysis on modifying both policies so that only previously chosen cycles or pairwise exchanges involving one or more high PRA patients are disallowed after each optimization (instead of simply assuming all previously chosen cycles or pairwise exchanges fail) has a similarly negligible effect. This is good because it shows that the effectiveness of pre-match testing in increasing the total number of transplants is robust, but also bad because it is clear these policies are far from optimal in selecting pre-match tests.

The pre-match testing I examine is equitable in that it does not give priority to one patient over another. It is true that more high PRA patients will fail the pre-match crossmatch tests and thus that fewer high PRA patients will be part of cycles in the final match-run, but this is also true in the APD status quo, and nevertheless on average 9% more high PRA patients receive transplants with my pre-match testing policies than without. Comparing the results of the pre-match testing policies with those of the *weighted* policy from Table 5, we see that pre-match testing does better on average in the total number of transplants but not in the wait times among the transplanted; this reflects the tradeoff between the wait times of transplanted patients and the number of patients transplanted. Although pre-match testing need not be equitable, my simulations show equitable pre-match testing to facilitate more transplants in the long run than maximizing weighted matchings. This suggests that pre-match testing is an attractive method of improving efficiency.

6 Discussion

This paper examines the effects of erroneous virtual compatibility tests within kidney exchanges. I explore the inefficiencies caused in terms of lost transplants and increased wait times among the transplanted, finding through simulation that patients, and highly sensitized patients in particular, are seriously harmed by an exchange’s inability to accurately allocate transplantable matches. Balancing the wait times of patients receiving transplants while ensuring that as many patients as possible are transplanted is an ethical concern beyond the scope of this paper.

I examine how failures affect hospital participation within multi-hospital kidney exchanges, finding that failures have perverse effects on a hospital’s incentives to truthfully report its pairs. These effects are difficult to mitigate because even mechanisms that come close to aligning hospital-exchange incentives produce fewer transplants than might take place under the match-maximizing mechanism. I further find that exchanges are not immune to incentives because an exchange can benefit in terms of total transplants by choosing only allocations with low probabilities of failure, but that doing so is particularly harmful to highly sensitized patients. Based on these results, I find that failures pose a significant problem for kidney exchange and need to be addressed.

I additionally examine how failures affect the optimal matching of nondirected donors, looking in particular at how NEAD chains compare to DPDs. I find that exchanges should encourage chains over DPDs because although the non-simultaneity property of chains puts them at risk of bridge donor renege, it also makes them robust to failure. This ability of a chain to grow in the future regardless of failure today outweighs the detriment caused by the possibility of a renege. I confirm this result through sensitivity analysis on relevant parameters.

Since patients would clearly benefit if there were fewer failures, I investigate pre-match testing policy. I find that naive pre-match crossmatch testing increases the

total number of transplants and decreases wait times among the transplanted significantly, but that most tests conducted are redundant. This suggests there is a high marginal gain to improved compatibility information given current rates of false negative crossmatch. That pre-match testing makes a difference is encouraging, and indeed the APD is planning to implement a procedure where some pre-match crossmatch testing would occur at the APD's headquarters in Toledo, OH. Pre-match testing is also beneficial because it means the final match-run can choose matches known to be transplantable, and this should make individual rationality more easily attainable.

The problem of choosing relevant pre-match tests relates to a general class of labour market matching problems. Consider the problem an employer faces when choosing which job candidates to interview, given that the employer can only interview a certain number of candidates and that some candidates might turn down job offers. (The employer might even be looking to assemble a team of employees with complementary skills; such a complementary team is analogous to the “cycles” of complementary pairs that I examine.) Matching problems like these have no closed form solutions but market design can help to reduce the mis-matches, or “failures.” The American Economic Association for example has recently implemented a signaling mechanism where job market candidates can “signal” to two potential employers whom they believe to be a particularly good fit. This reduces information asymmetry and improves match quality ([3]). The pre-match testing I describe is a step in that direction.

Appendix

A. The match-maximization problem

Denote the set of all pairs in the exchange population pool as P and let $\mathcal{S}(P)$ be the set of all possible 2- and 3-cycle exchanges induced by the virtual compatibilities of those pairs. Choosing the disjoint cycles that maximize the number of matches is equivalent to solving a binary integer program, whose objective is

$$\max_{x_i} \sum_{i=1}^{|\mathcal{S}(P)|} |c_i| \cdot x_i, \quad (15)$$

where x_i represents choosing the i th cycle c_i to be part of the match and $|c_i|$ denotes the size of c_i . Notice that $|c_i| \in \{2, 3\} \forall i$ since I consider only 2- and 3-cycles. This objective is subject to the constraints

$$\sum_{i: \text{pair } j \in c_i} x_i \leq 1 \quad \forall \text{ pairs } j, \quad (16)$$

$$x_i \in \{0, 1\} \quad \forall c_i. \quad (17)$$

which ensure respectively that a pair can only be part of one cycle in the solution and that the x_i are binary. This problem is NP-Complete ([14]), but my simulations are on a small enough scale that it is tractable.

B. The weighted match-maximization problem

The objective of the original binary integer program (specified in Equation 15) used to determine matchings becomes

$$\max_{x_i} \sum_{i=1}^{|\mathcal{S}(P)|} w_i \cdot x_i \quad (18)$$

where w_i is a particular cycle c_i 's expected value in transplants (the probability that there is no failure multiplied by magnitude). Again, $\mathcal{S}(P)$ is the set of all possible 2- and 3-cycles on the set of pairs P and x_i is the binary decision variable that denotes whether the i th cycle is chosen to be part of the match. The original constraints in Equations 16 and 17 are unchanged.

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