Epidemic Modeling of the Onset of Social Activities (EMOSA Models): Applications to Adolescent Religious Involvement

Joe Rodgers,

Andriy Koval, and Oleksandra Hararuk

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Acknowledgements

- EMOSA models have been in development for almost 25 years, as a collaborative effort shared with my colleague **David Rowe**, University of Arizona – who died in February, 2002

- Other collaborators – Grad students **Sylvia Meseck-Bushey, Maury Buster, Amber Johnson**

- Collaborator on today's work: **Andriy Koval** - current OU Quant grad student, **Oleksandra Hararuk** – OU ecological modeling lab, **William Beasley** - OU Health Sciences Center, Department of Pediatrics

Mathematical/Statistical Models

- Mathematical/statistical models purport to represent the behavioral processes that are being modeled
- Models have exactly two features
  - They match reality (fit)
  - They are simpler than that reality (parsimony)
- Some models are designed as broad representations, portable models – e.g., regression, SEM, MLM
- Some models are built to match a particular version of reality
- EMOSA models are an example of the latter

Introduction

Epidemic

Models

Onset

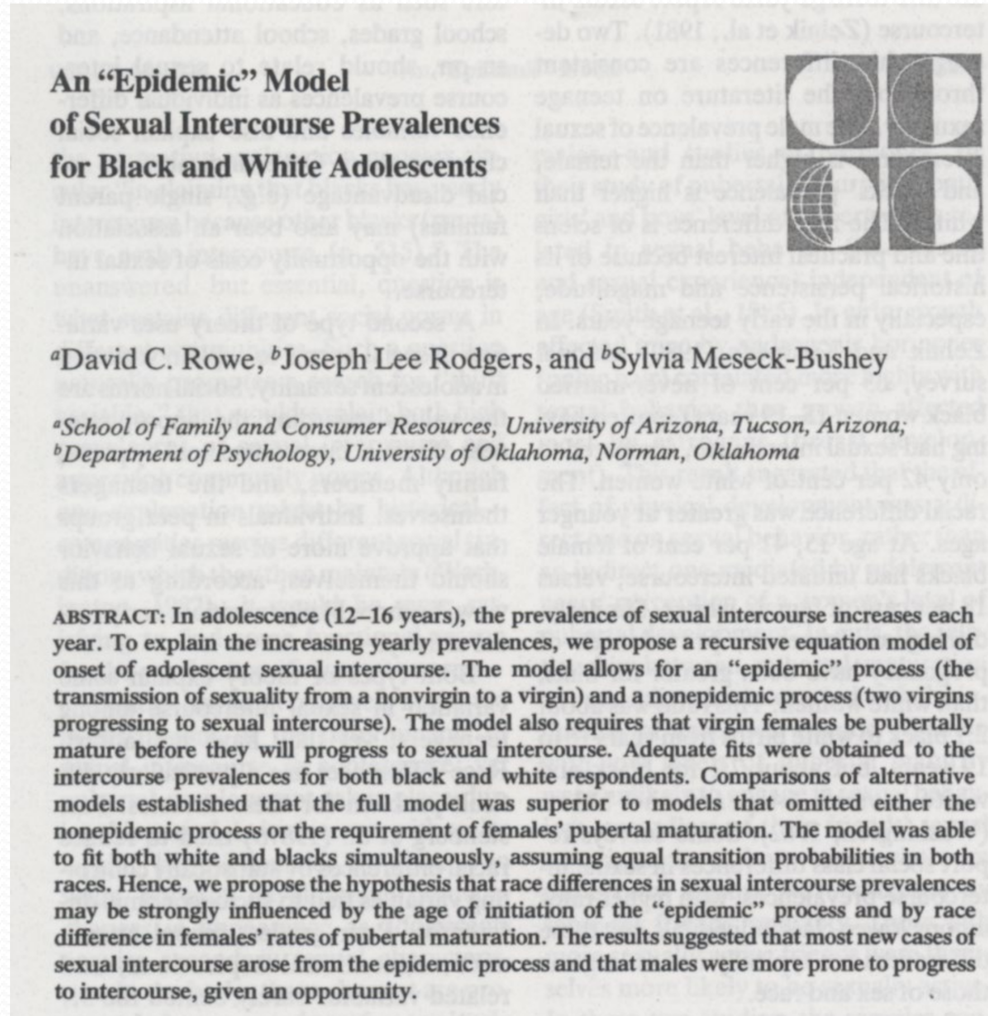
Social

Activity

- These models have been borrowed from the field of Epidemiology
- They originated from May-Anderson models, which describe the spread bacteria, viruses, etc. through **biological contagion**
- But our application involves **social contagion**

- Past applications, starting in 1989

- Smoking
- Drinking
- Sexual Activity



An "Epidemic" Model of Sexual Intercourse Prevalences for Black and White Adolescents

[a]David C. Rowe, [b]Joseph Lee Rodgers, and [b]Sylvia Meseck-Bushey

[a]*School of Family and Consumer Resources, University of Arizona, Tucson, Arizona;*
[b]*Department of Psychology, University of Oklahoma, Norman, Oklahoma*

ABSTRACT: In adolescence (12–16 years), the prevalence of sexual intercourse increases each year. To explain the increasing yearly prevalences, we propose a recursive equation model of onset of adolescent sexual intercourse. The model allows for an "epidemic" process (the transmission of sexuality from a nonvirgin to a virgin) and a nonepidemic process (two virgins progressing to sexual intercourse). The model also requires that virgin females be pubertally mature before they will progress to sexual intercourse. Adequate fits were obtained to the intercourse prevalences for both black and white respondents. Comparisons of alternative models established that the full model was superior to models that omitted either the nonepidemic process or the requirement of females' pubertal maturation. The model was able to fit both white and blacks simultaneously, assuming equal transition probabilities in both races. Hence, we propose the hypothesis that race differences in sexual intercourse prevalences may be strongly influenced by the age of initiation of the "epidemic" process and by race difference in females' rates of pubertal maturation. The results suggested that most new cases of sexual intercourse arose from the epidemic process and that males were more prone to progress to intercourse, given an opportunity.

Overview

- EMOSA Sexuality Models
  - Simple two-sex EMOSA sexuality model
  - Inter-cohort contagion model
  - Developmental EMOSA sexual development model
  - EMOSA sexuality/pregnancy/STD model

- EMOSA Smoking and Drinking Models
  - Simple one-sex EMOSA smoking/drinking model
  - Stagewise EMOSA smoking/drinking model
  - The mathematics of social contagion
  - New data - the Oklahoma Smoking/Drinking Survey
  - New empirical results

A Simple EMOSA Sexuality Model

Rowe, Rodgers, & Meseck-Bushey, 1989, Social Biology:

$$P_{t+1} = P_t + T\,(P_t)\,(1-P_t) + k\,(1-P_t)\,(1-P_t)$$

Carry-Over Epidemic Non-epidemic

P_t is prevalence at time t

T is the epidemic transmission parameter

k is the non-epidemic transmission parameter

Figure 1, Rodgers, Rowe, & Buster, 1998, Developmental Psychology

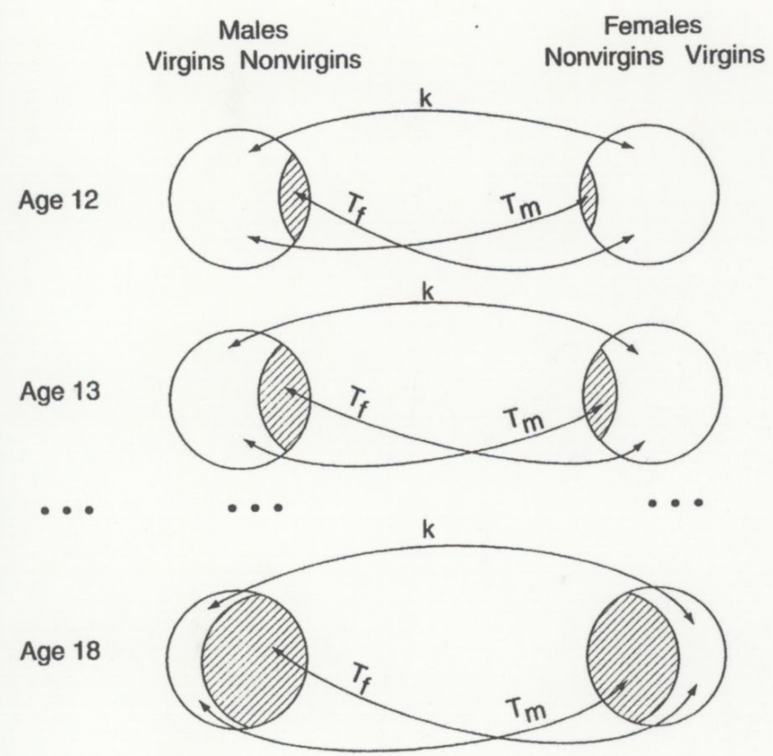
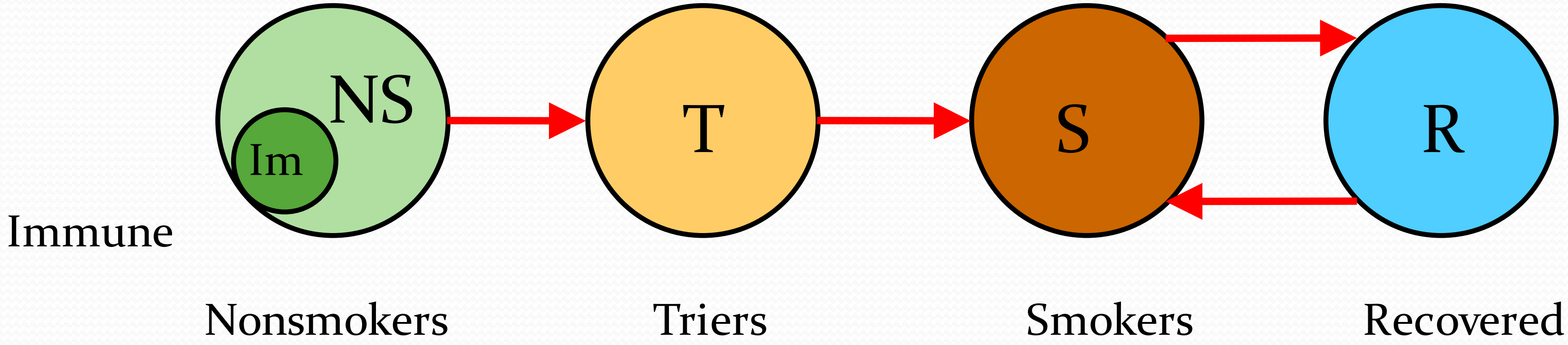


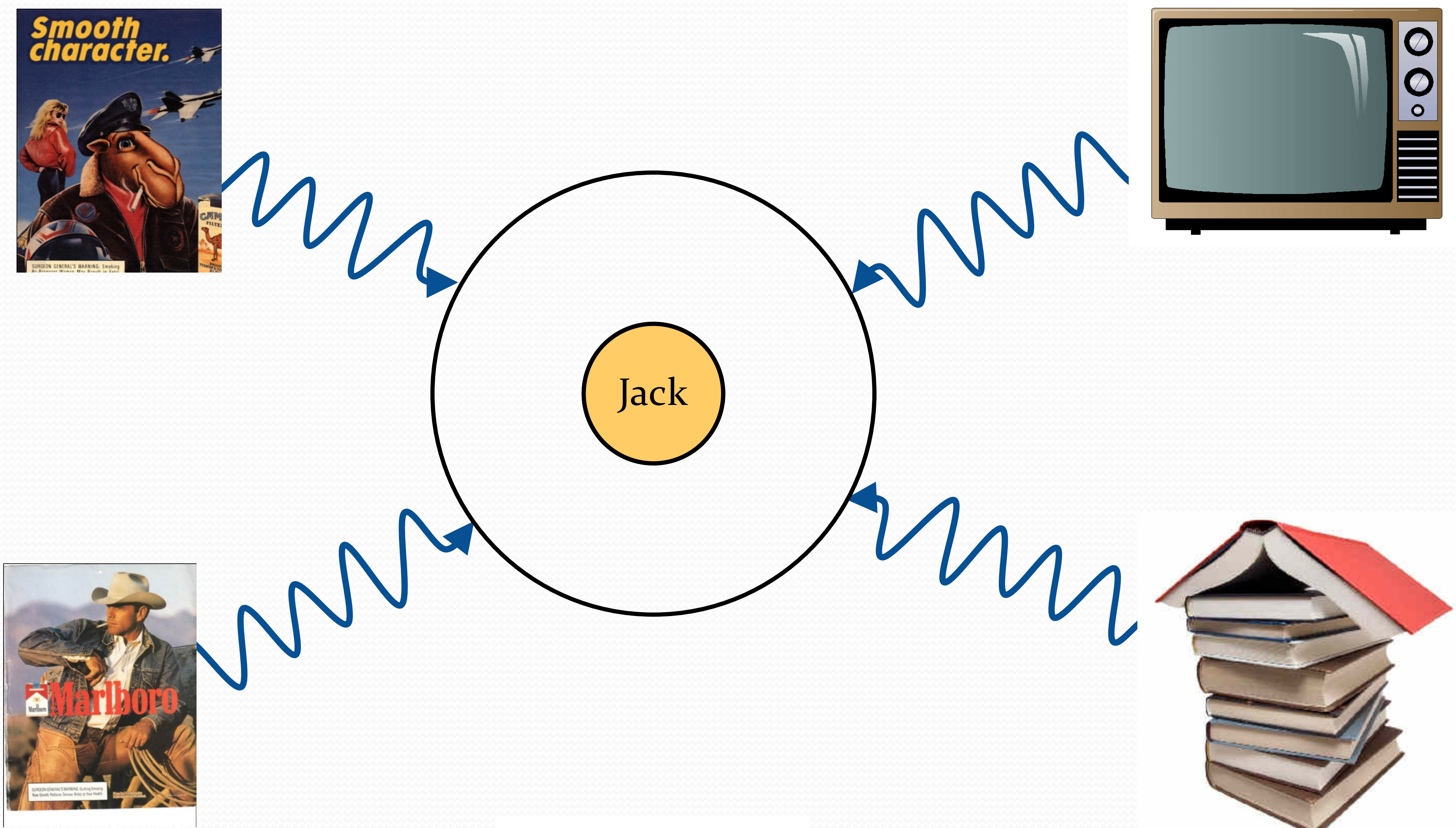
Figure 1. A schematic of the EMOSA sexuality model, with shaded regions indicating the predicted proportion of nonvirgin males in the left circles and the predicted proportion of nonvirgin females in the right circles. EMOSA = epidemic models of the onset of social activities; k = nonepidemic sexuality transition rate; T = epidemic sexuality transition rate; f = female; m = male.

EMOSA Smoking and Drinking Models

- These work the same basic way that the sexuality models work, except that they're single-sex models
- Same types of filters, mixing adjustments, etc.
- More focus on developmental stage models
- Social congation versus General diffusion becomes important in the smoking/drinking models

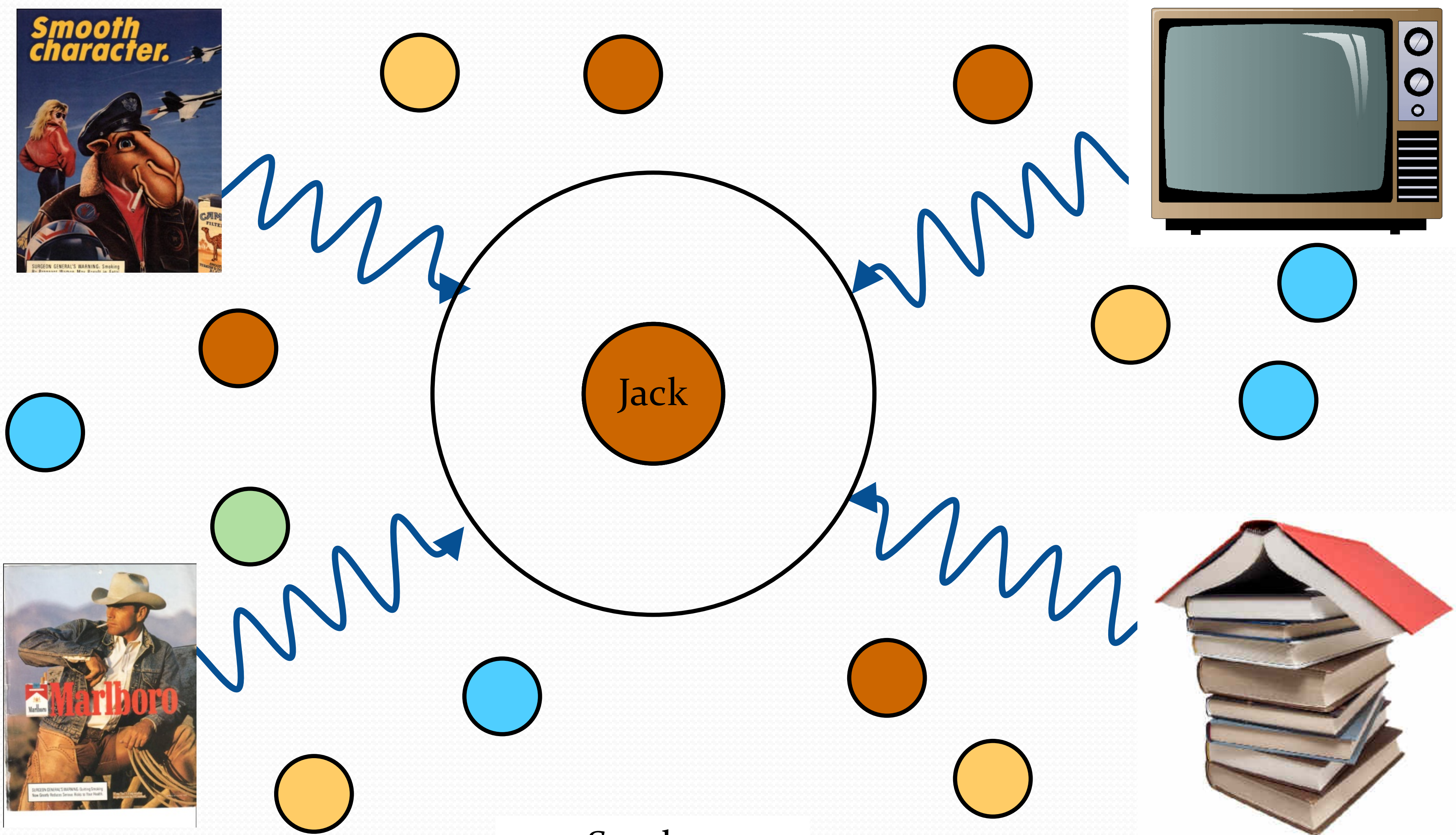


Contagion vs. Diffusion

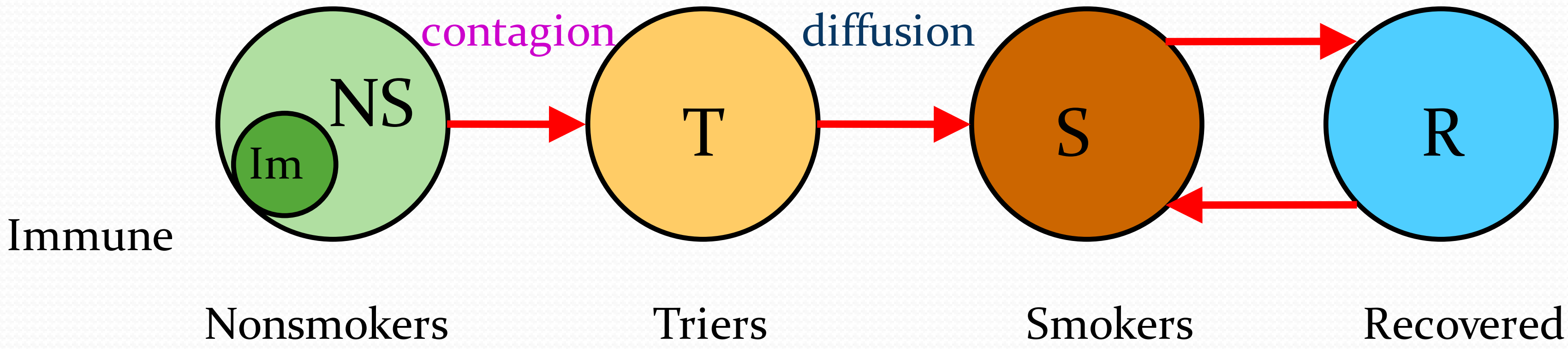


Trier

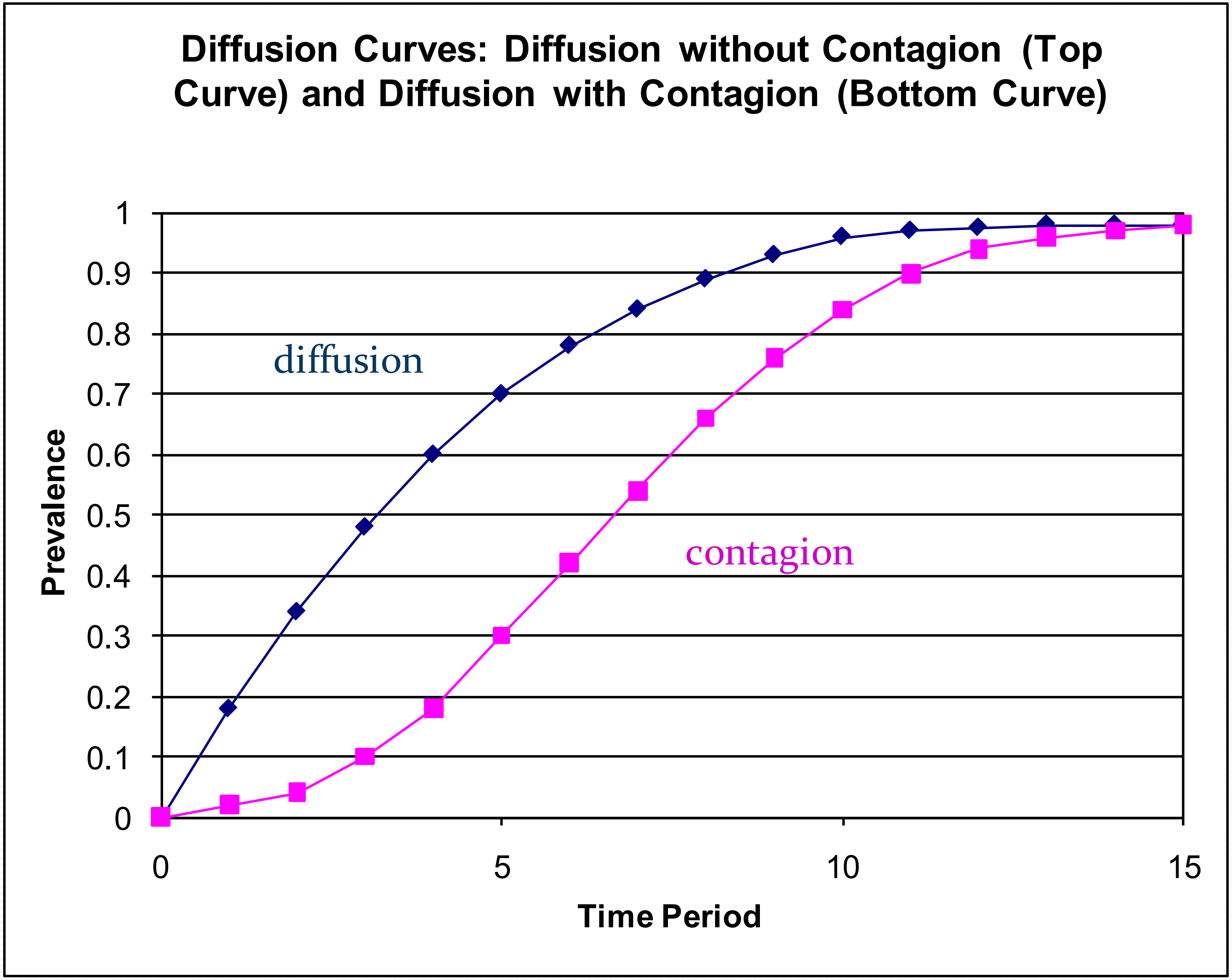
Contagion vs. Diffusion



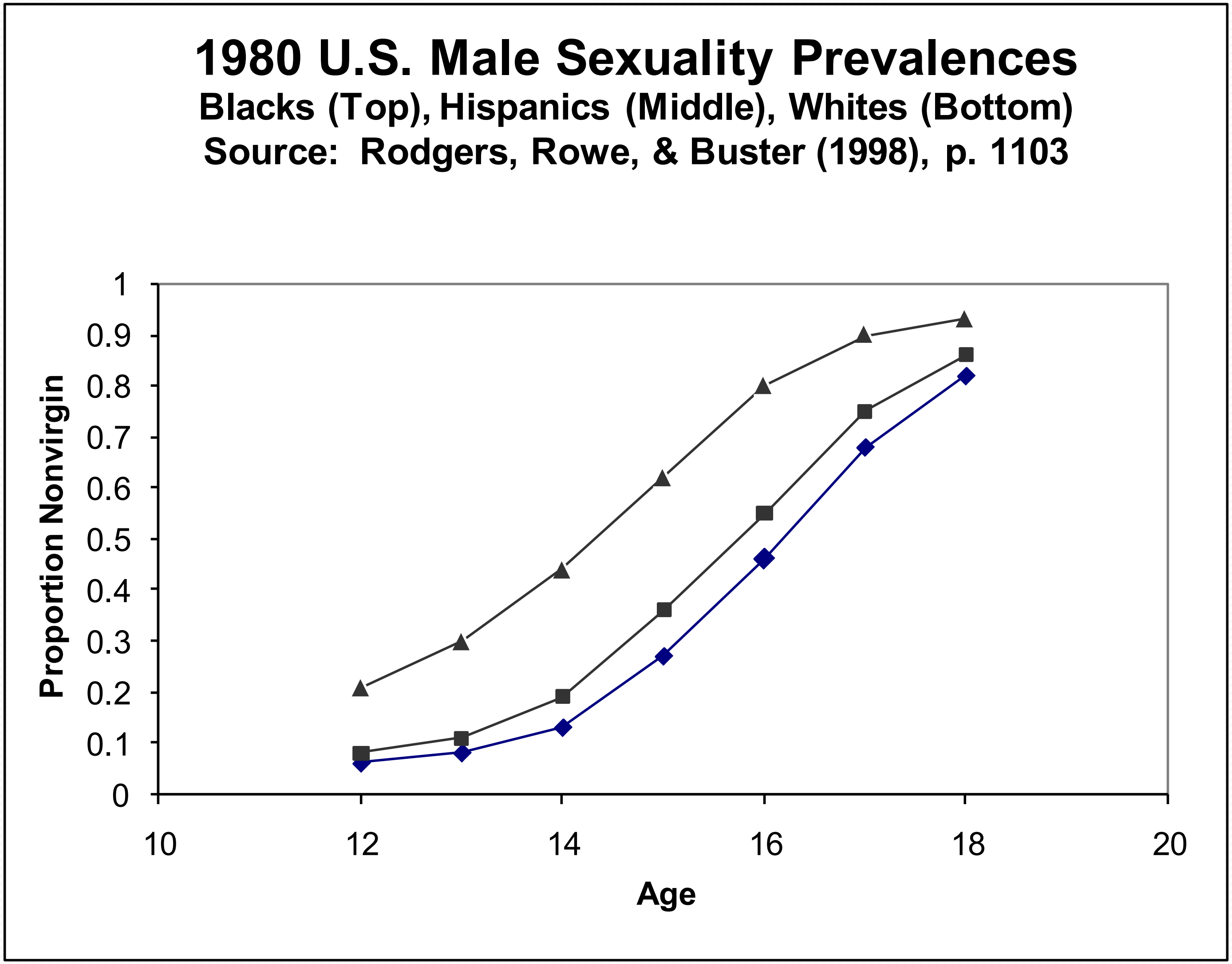
- Work by David Rowe (Rowe et al, 1994) showed that <u>onset</u> of smoking is all about social contagion
- But <u>transition to regular smoking</u> is all about general diffusion
- In other words, kids start smoking because their friends encourage them to; they continue smoking because of the presence of a “smoking culture” in their families, on tv, in magazines, etc.



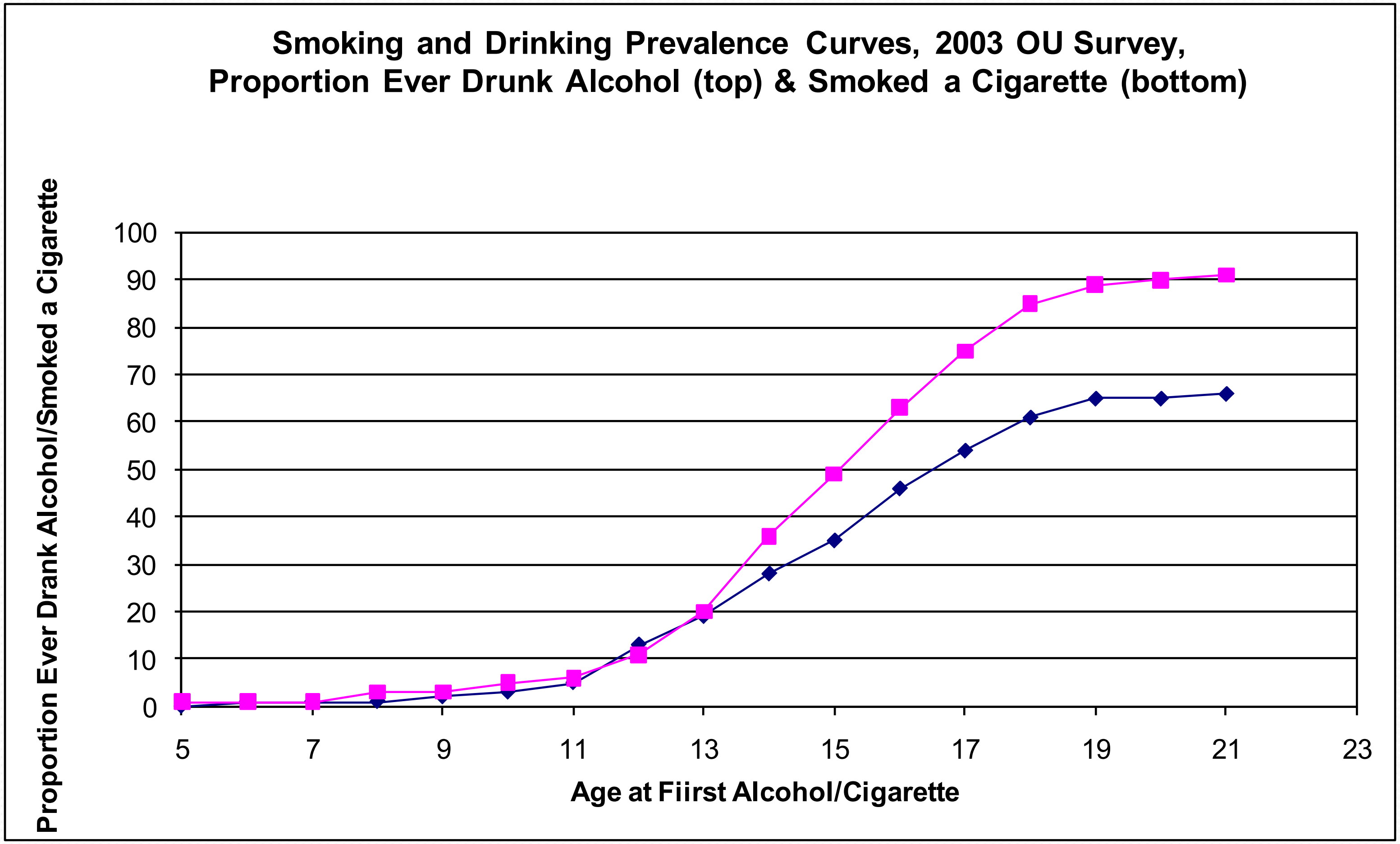
- These two processes are similar, but have tremendously different policy implications
- E.g., consider the delivery of a pregnancy reduction campaign to inner-city adolescent girls
- Social contagion implies intervene at the friendship/dyad level
- General diffusion implies use an ad campaign


Diffusion Curves: Diffusion without Contagion (Top Curve) and Diffusion with Contagion (Bottom Curve)
diffusion
contagion
Prevalence
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
5
10
15
Time Period

From Rodgers, Rowe, & Buster (1998)



From Rodgers & Johnson (2007)



EMOSA Religious Involvement Model

- In this new application, we posit the same type of social contagion – i.e., a social influence process that passes from person to person – as a potential for influencing religious behavior upon entry into college
- We have longitudinal data, from the NLSY97, that reflects ongoing religious involvement behavior
- Looking forward: Is there a visual tell-tale sign for free transitions?

Religious Involvement Data

- National Longitudinal Survey of Youth (NLSY97)

- Over 9,000 respondents
- 5,214 Included (No missing data)

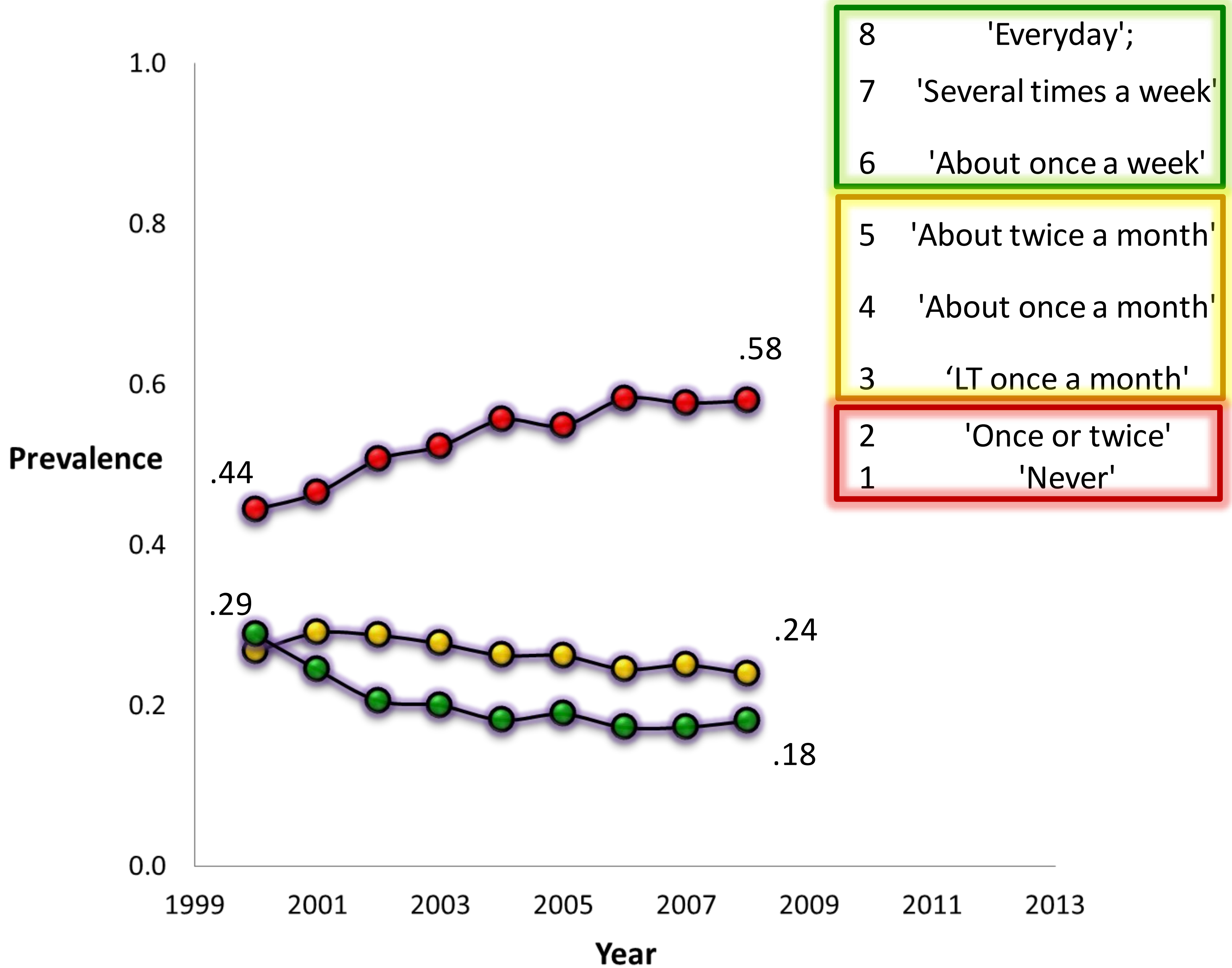
- Age at 2000 : 16-20
- Cohorts:

1980 – 1981 – 1982 – 1983 – 1984

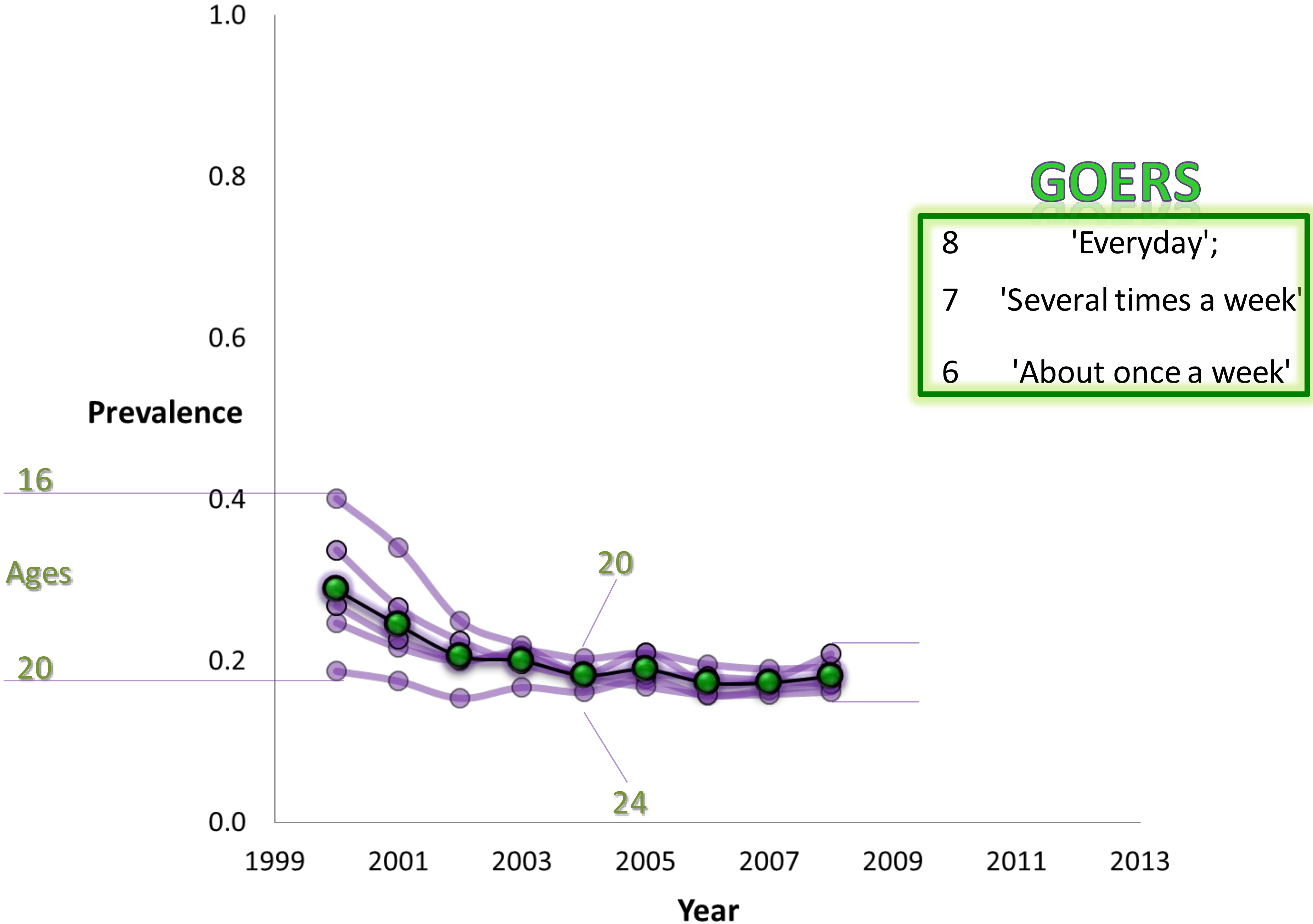
Religious Involvement Data

Q: "In the past year, how frequently did you attend a place of worship?"

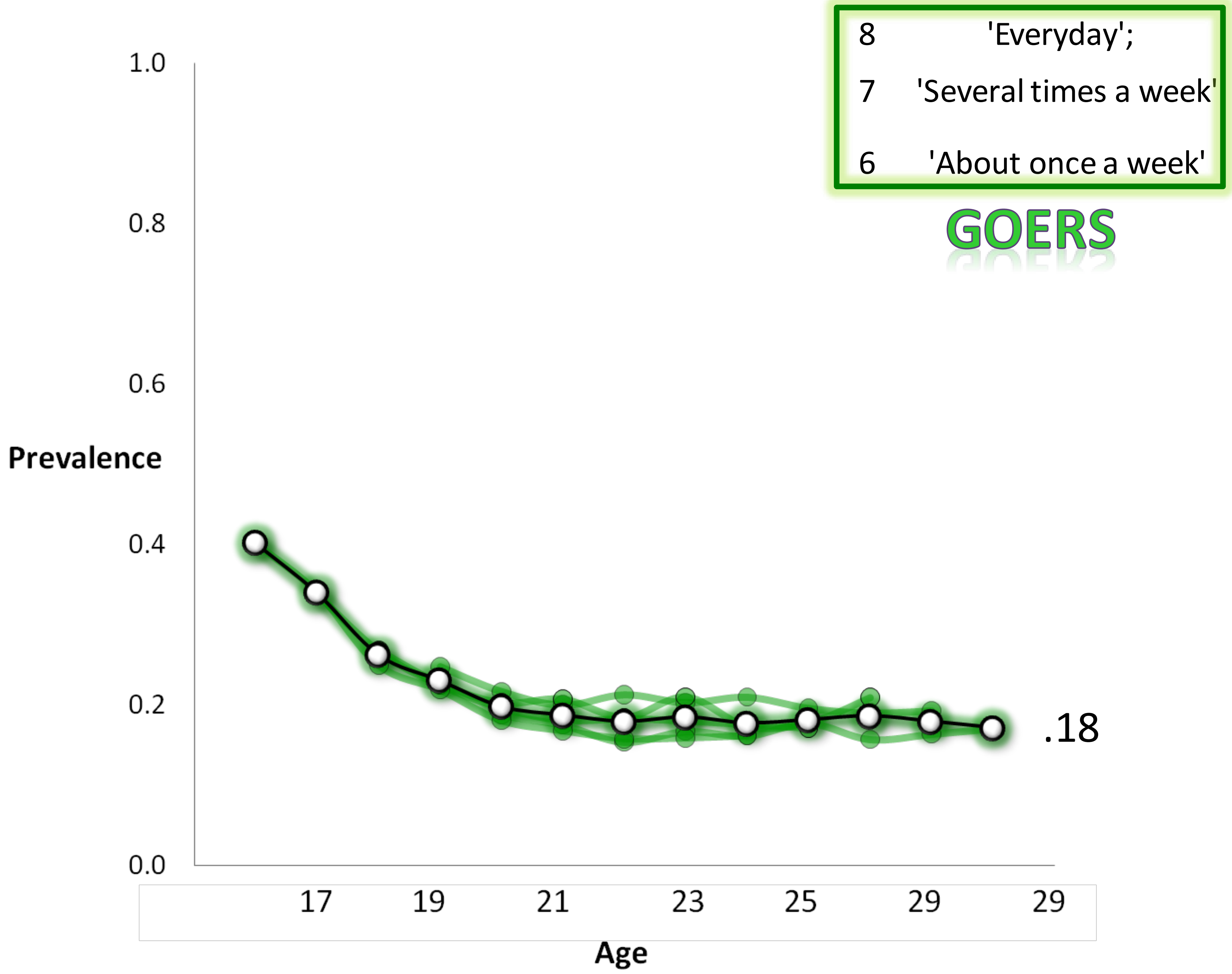
GOERS	8	'Everyday'
	7	'Several times a week'
	6	'About once a week'
IRREGULARS	5	'About twice a month'
	4	'About once a month'
	3	'Less than once a month'
ABSENTEES	2	'Once or twice'
	1	'Never'



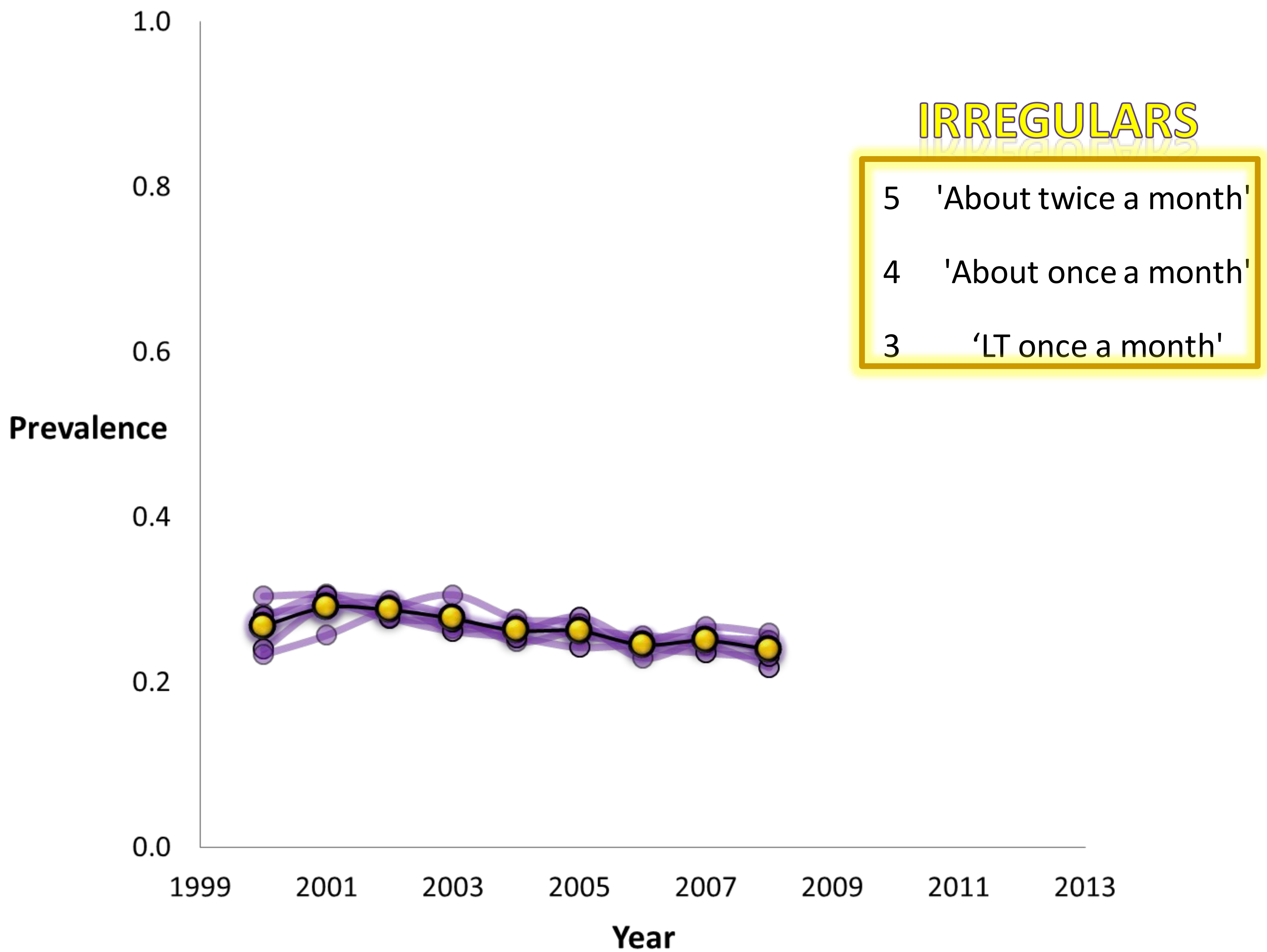
1.0
0.8
0.6
0.4
0.2
0.0
Prevalence
.44
.58
.29
.24
.18
1999
2001
2003
2005
2007
2009
2011
2013
Year
8 'Everyday';
7 'Several times a week'
6 'About once a week'
5 'About twice a month'
4 'About once a month'
3 ‘LT once a month'
2 'Once or twice'
1 'Never'



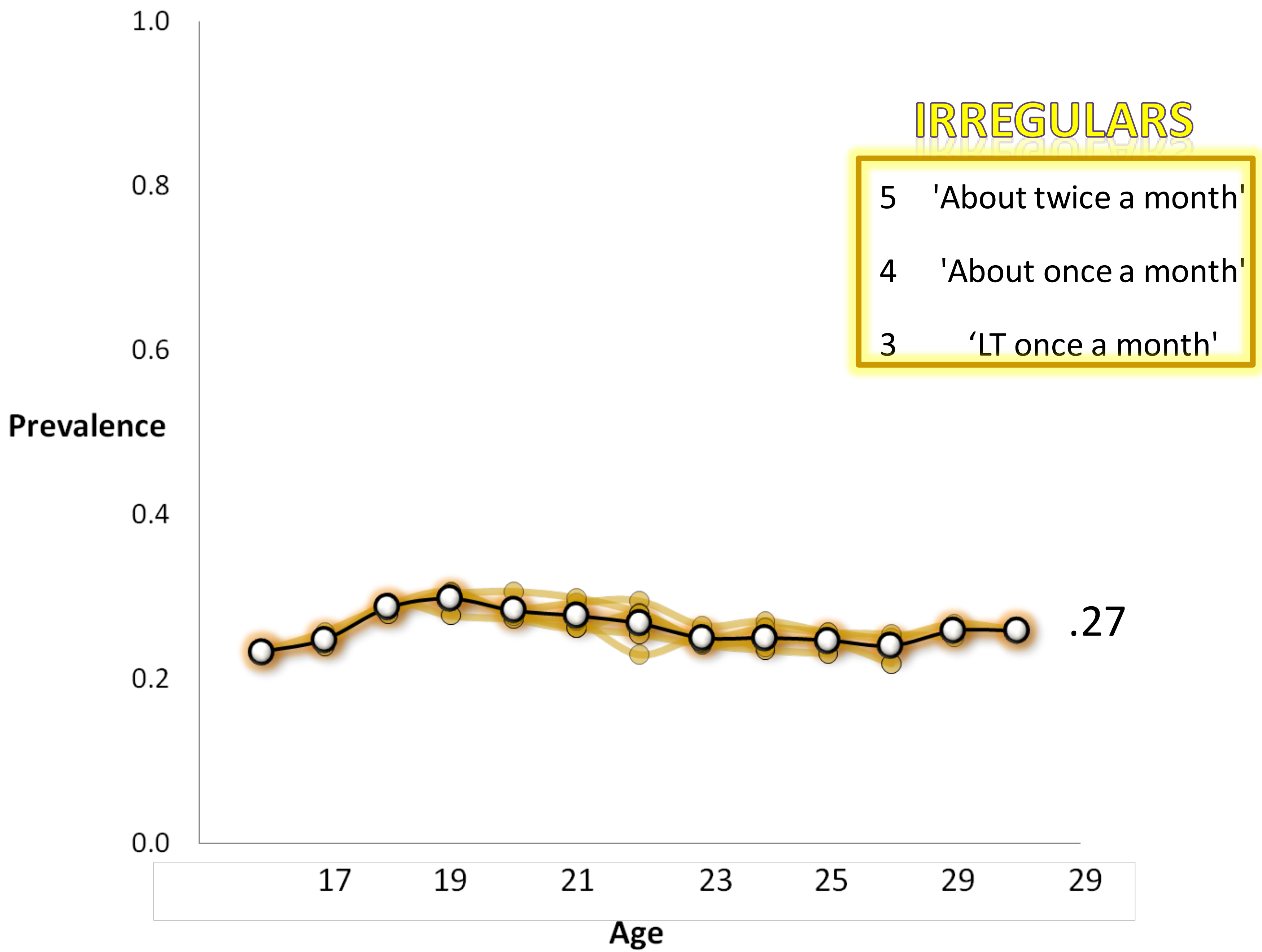
GOERS
8 'Everyday';
7 'Several times a week'
6 'About once a week'
1.0
0.8
0.6
Prevalence
16
0.4
Ages
20
0.2
20
24
0.0
1999
2001
2003
2005
2007
2009
2011
2013
Year



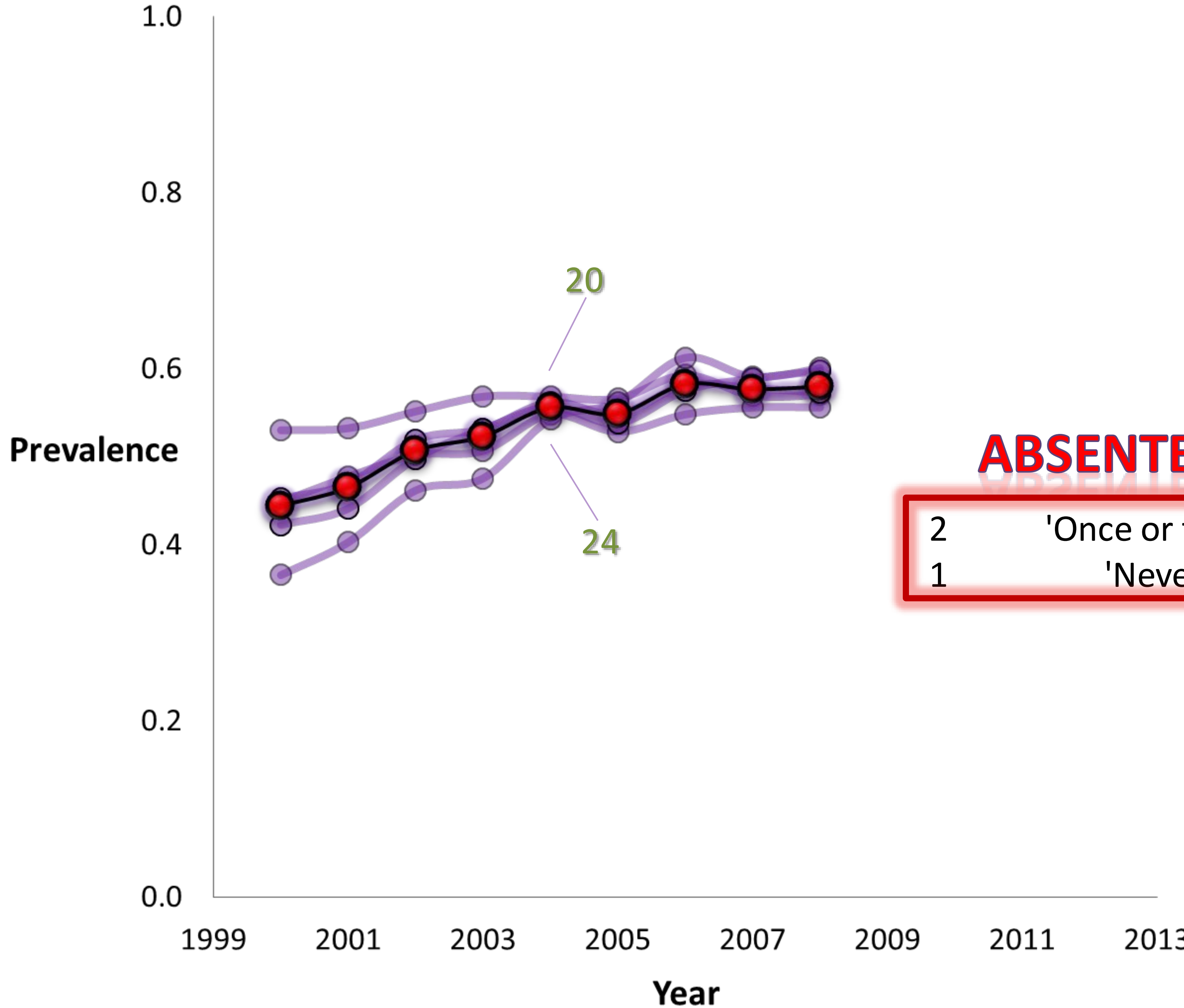
8 'Everyday';
7 'Several times a week'
6 'About once a week'
GOERS
1.0
0.8
0.6
Prevalence
0.4
0.2
0.0
.18
17
19
21
23
25
29
29
Age



IRREGULARS
5 'About twice a month'
4 'About once a month'
3 'LT once a month'
Prevalence
1.0
0.8
0.6
0.4
0.2
0.0
1999
2001
2003
2005
2007
2009
2011
2013
Year

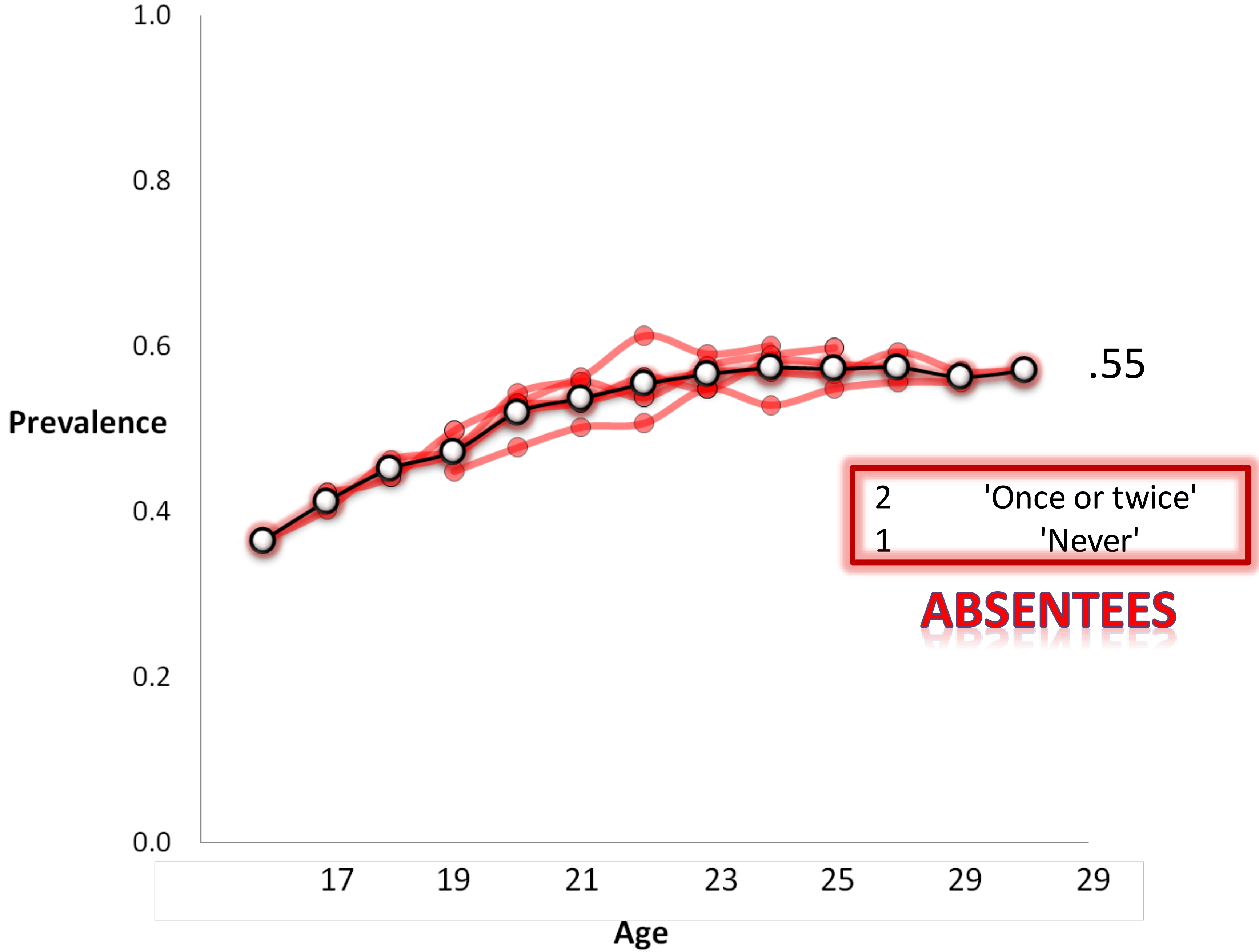


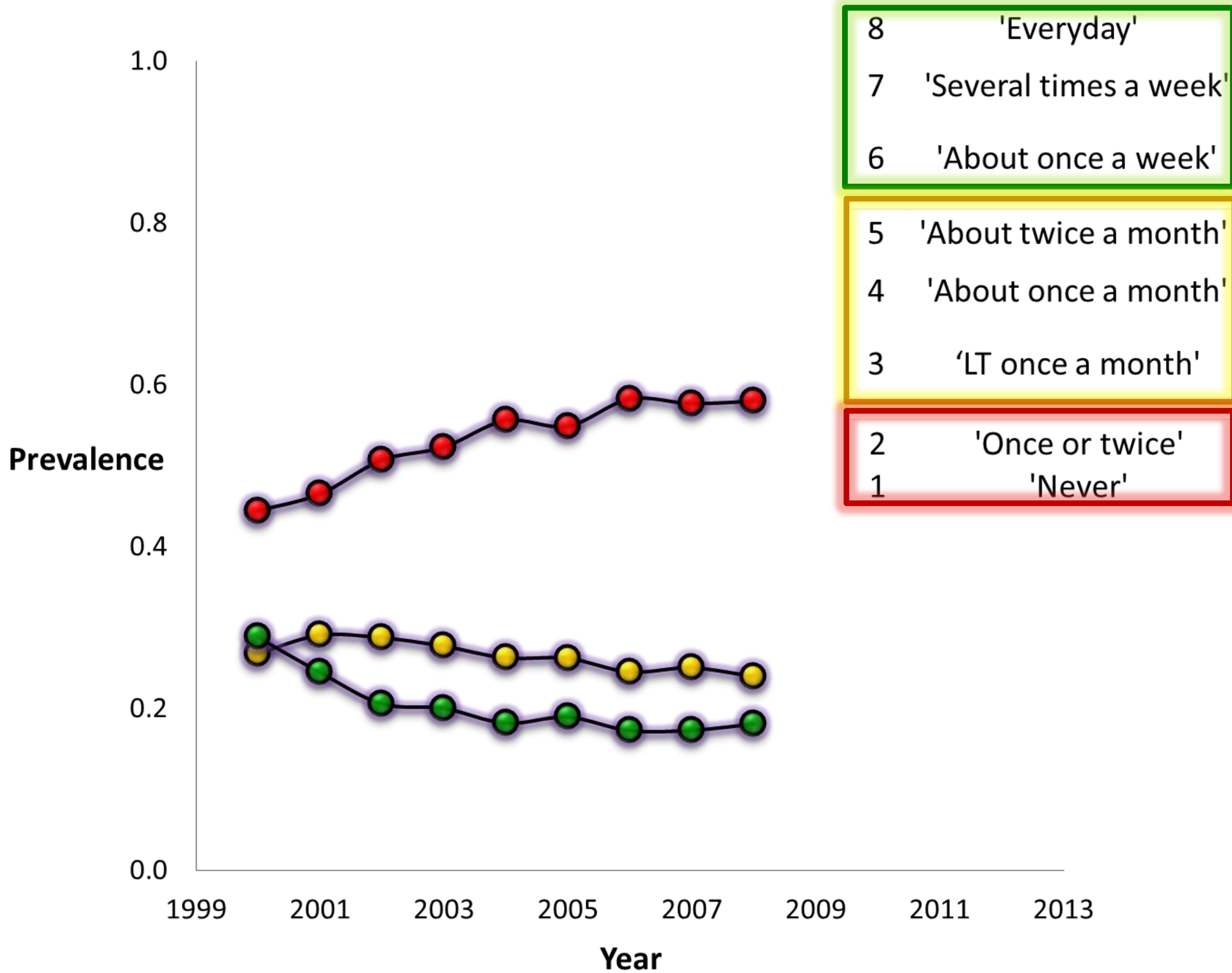
IRREGULARS
5 'About twice a month'
4 'About once a month'
3 'LT once a month'
1.0
0.8
0.6
0.4
0.2
0.0
Prevalence
.27
17
19
21
23
25
29
29
Age



ABSENTEES

2	'Once or twice'
1	'Never'


1.0
0.8
0.6
0.4
0.2
0.0
Prevalence
.55
2 'Once or twice'
1 'Never'
ABSENTEES
17
19
21
23
25
29
29
Age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6 'About once a week'
5 'About twice a month'
4 'About once a month'
3 'LT once a month'
2 'Once or twice'
1 'Never'
1.0
0.8
0.6
0.4
0.2
0.0
Prevalence
1999
2001
2003
2005
2007
2009
2011
2013
Year

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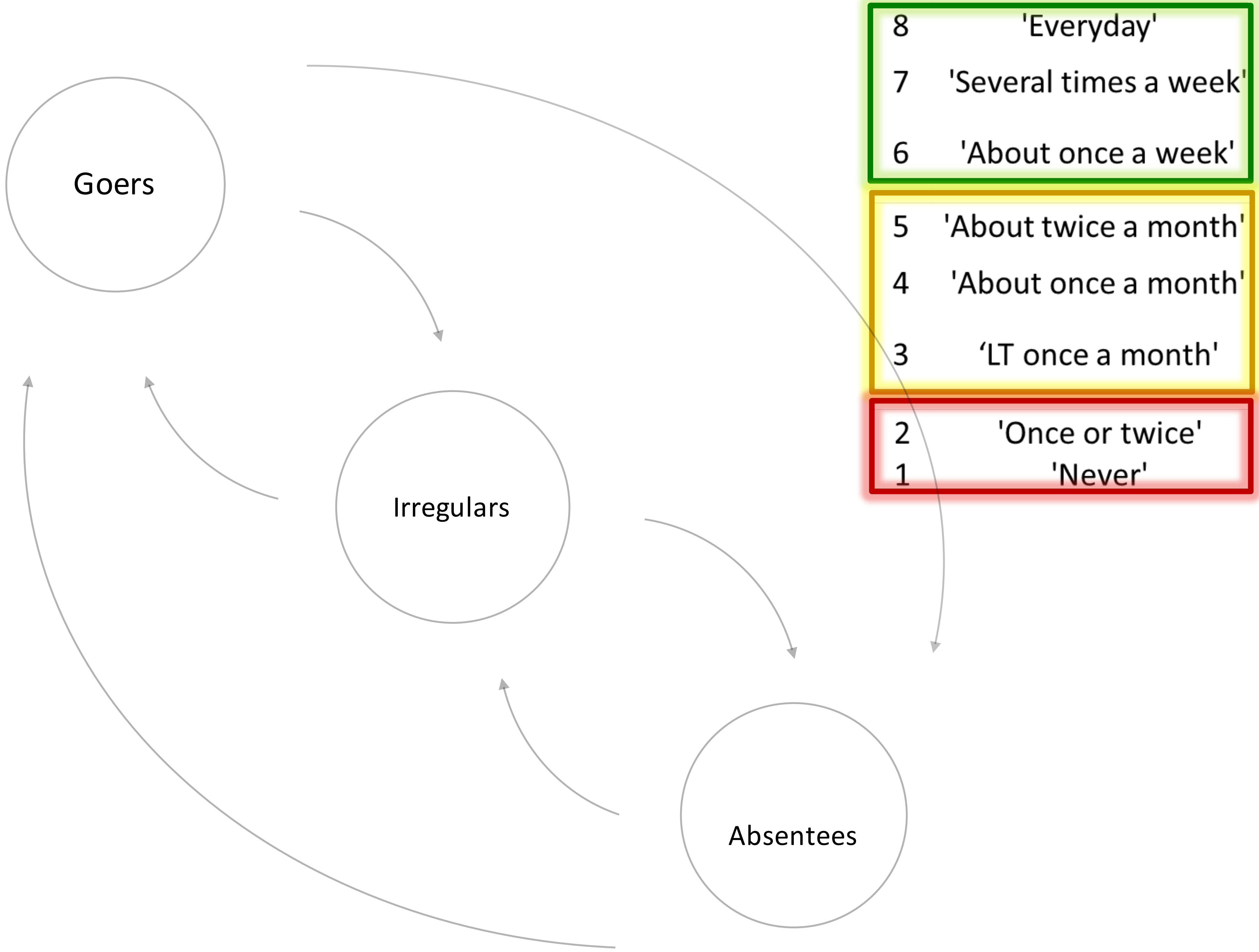
Goers
Irregulars
Absentees
8 'Everyday'
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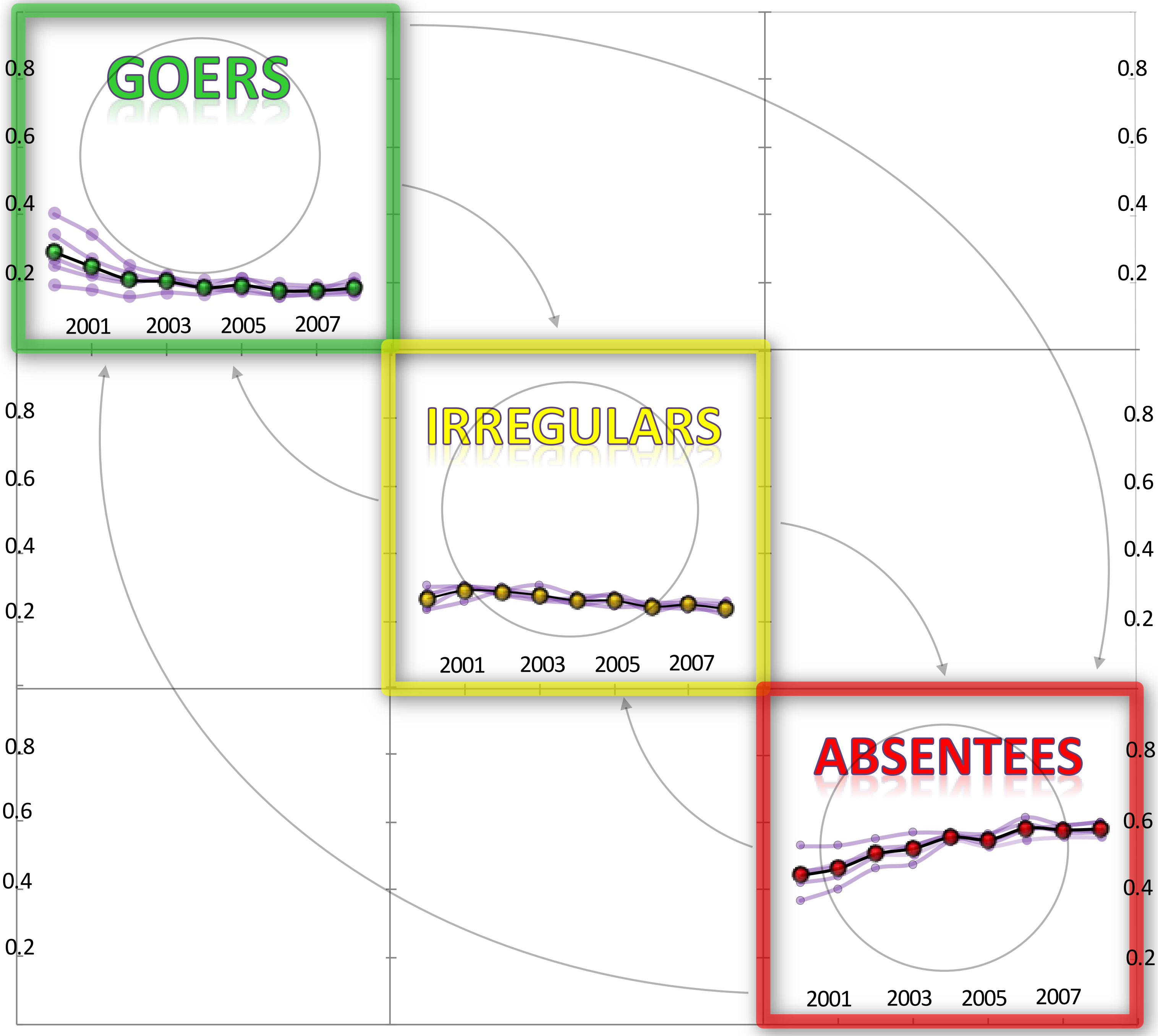
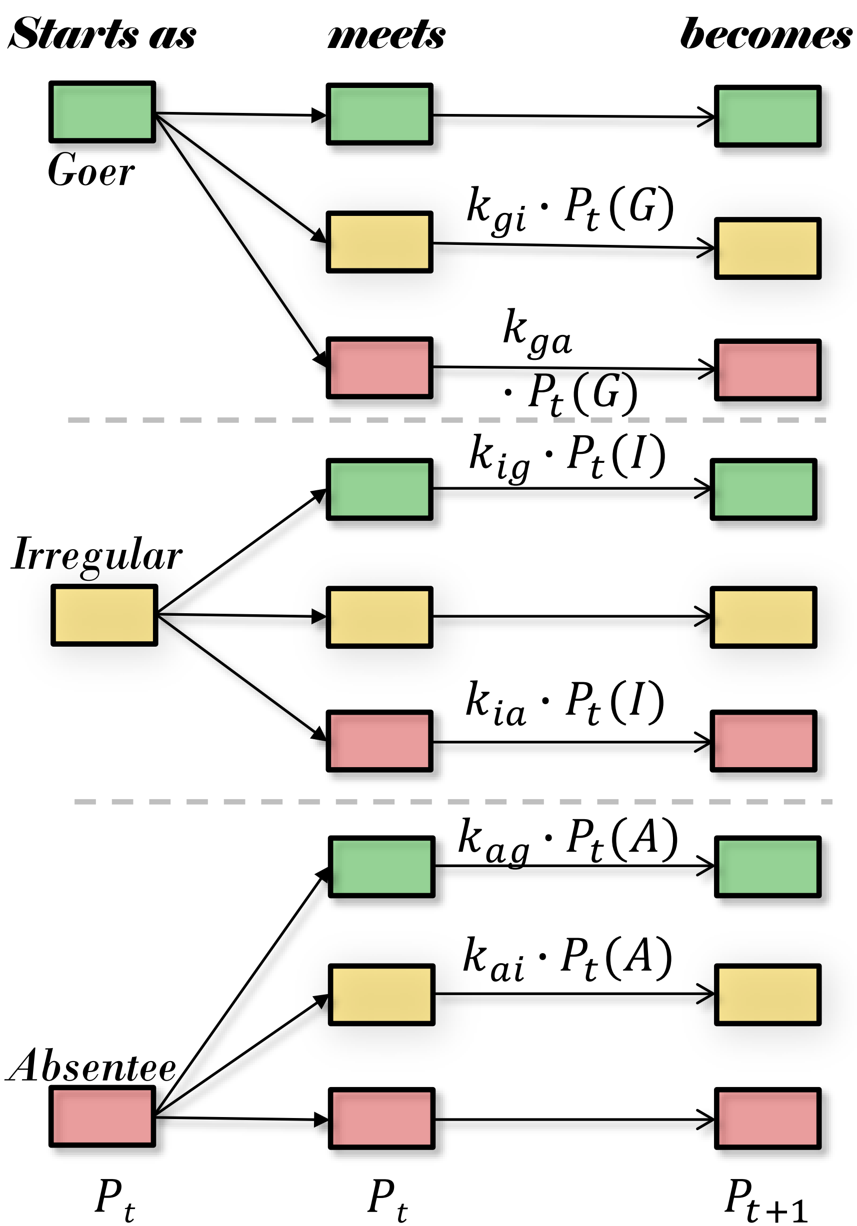
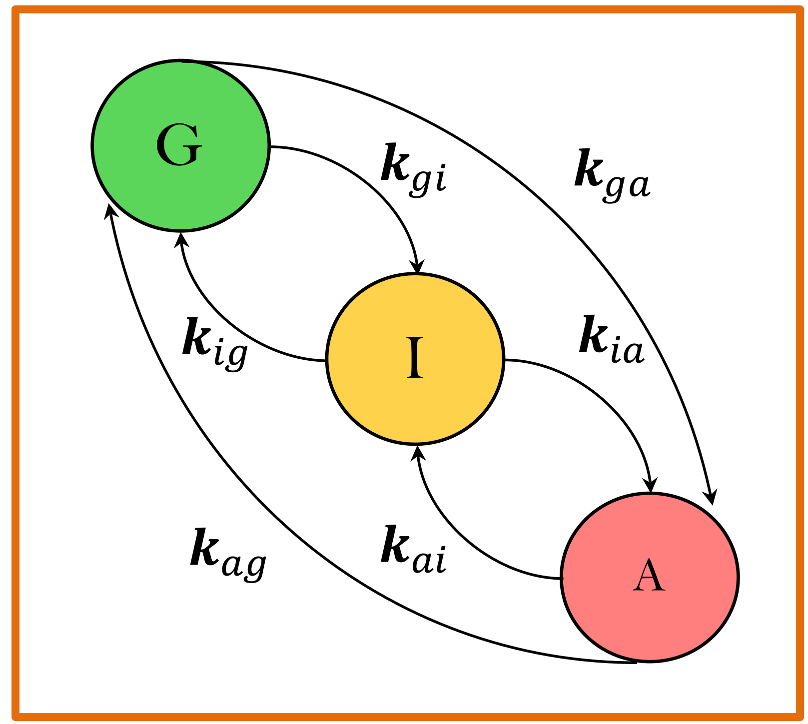


GOERS
IRREGULARS
ABSENTEES
0.8
0.6
0.4
0.2
2001
2003
2005
2007

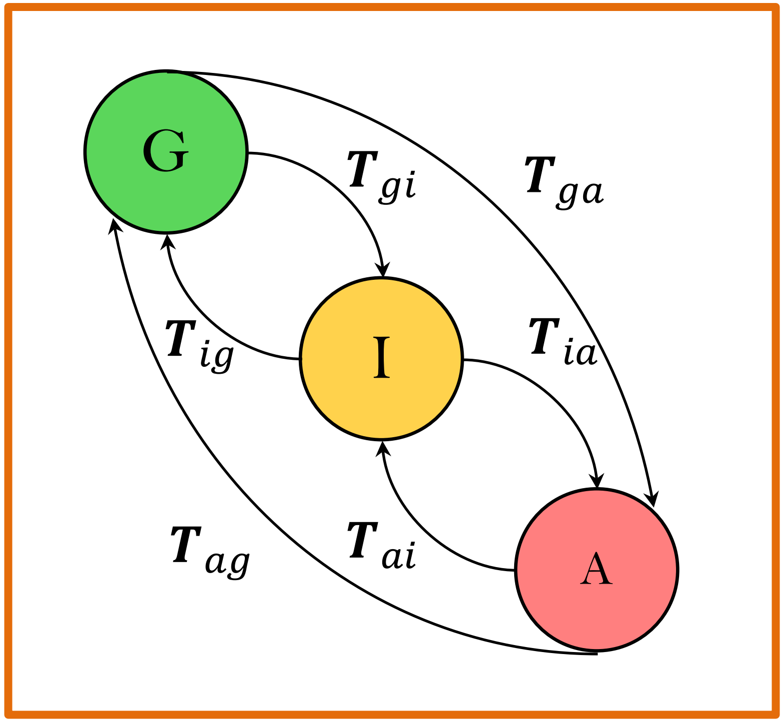
Specification : Diffusion

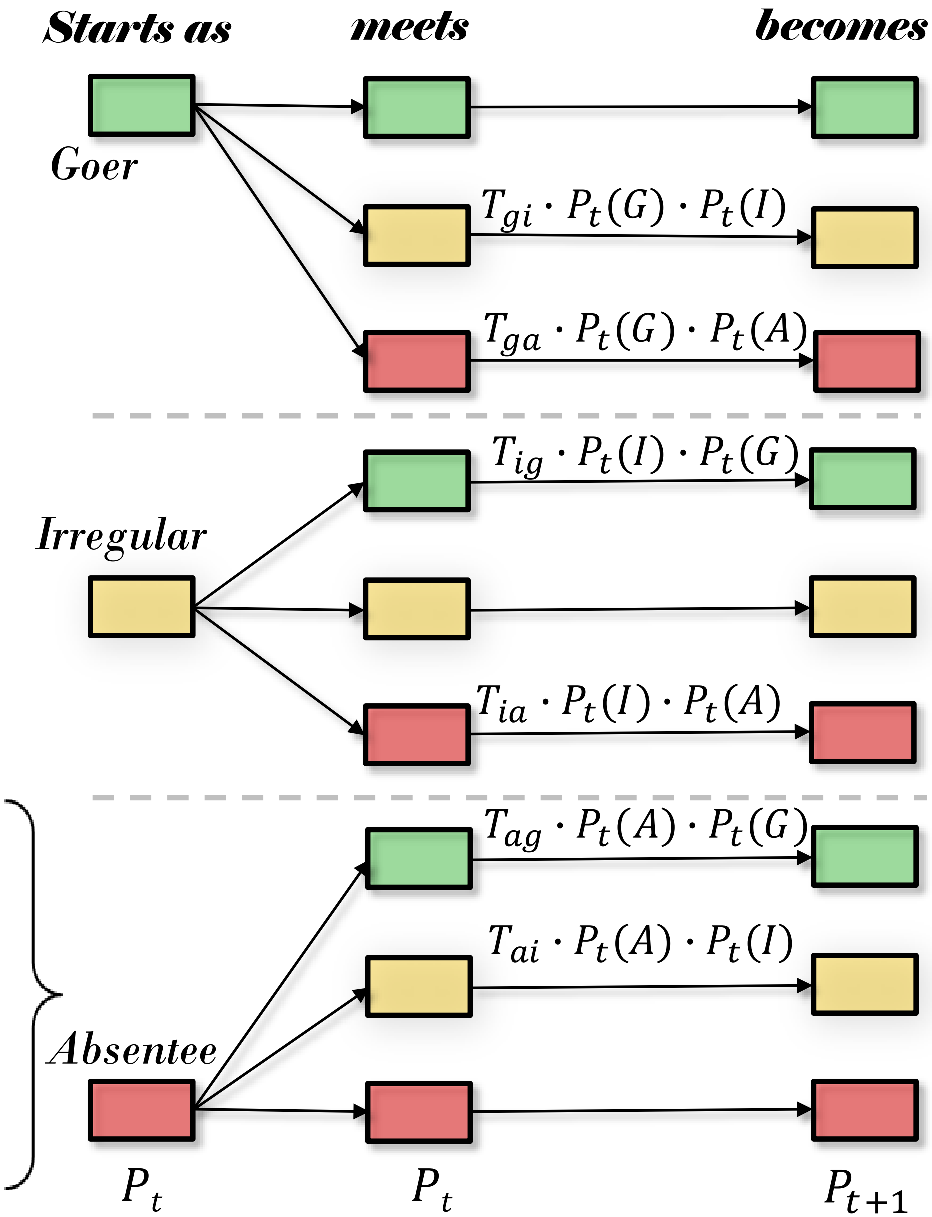




$$
\left\{
\begin{aligned}
P_{t+1}(G) &= P_t(G) + k_{ig} \cdot P_t(I) + k_{ag} \cdot P_t(A) \\
&\quad - k_{gi} \cdot P_t(G) - k_{ga} \cdot P_t(G) \\
P_{t+1}(I) &= P_t(I) + k_{gi} \cdot P_t(G) + k_{ai} \cdot P_t(A) \\
&\quad - k_{ig} \cdot P_t(I) - k_{ia} \cdot P_t(I) \\
P_{t+1}(A) &= 1 - P_{t+1}(G) - P_{t+1}(I)
\end{aligned}
\right\}
$$

Specification : Contagion



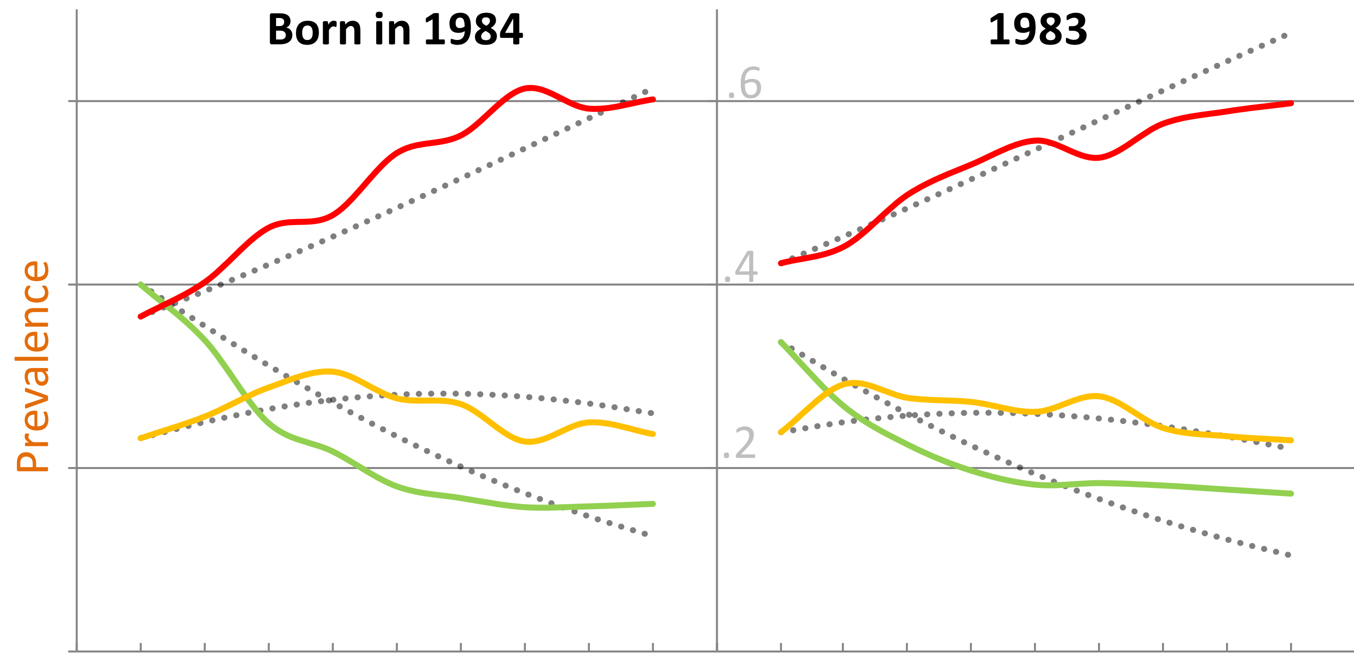


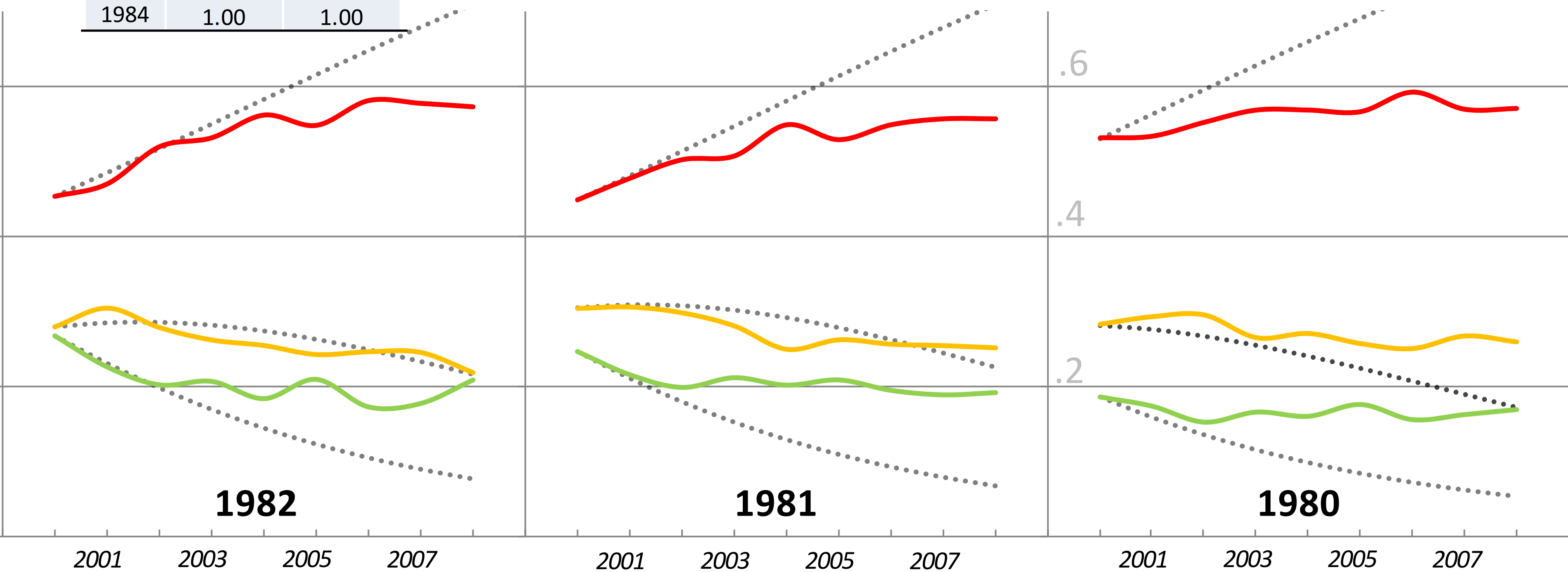
$$\left\{ \begin{aligned} P_{t+1}(G) &= P_t(G) + T_{ig} \cdot P_t(I) \cdot P_t(G) + T_{ag} \cdot P_t(A) \cdot P_t(G) \\ &\quad - T_{gi} \cdot P_t(G) \cdot P_t(I) - T_{ga} \cdot P_t(G) \cdot P_t(A) \\ P_{t+1}(I) &= P_t(I) + T_{gi} \cdot P_t(G) \cdot P_t(I) + T_{ai} \cdot P_t(A) \cdot P_t(I) \\ &\quad - T_{ig} \cdot P_t(I) \cdot P_t(G) - T_{ia} \cdot P_t(I) \cdot P_t(A) \\ P_{t+1}(A) &= 1 - P_{t+1}(G) - P_{t+1}(I) \end{aligned} \right\}$$

Contagion

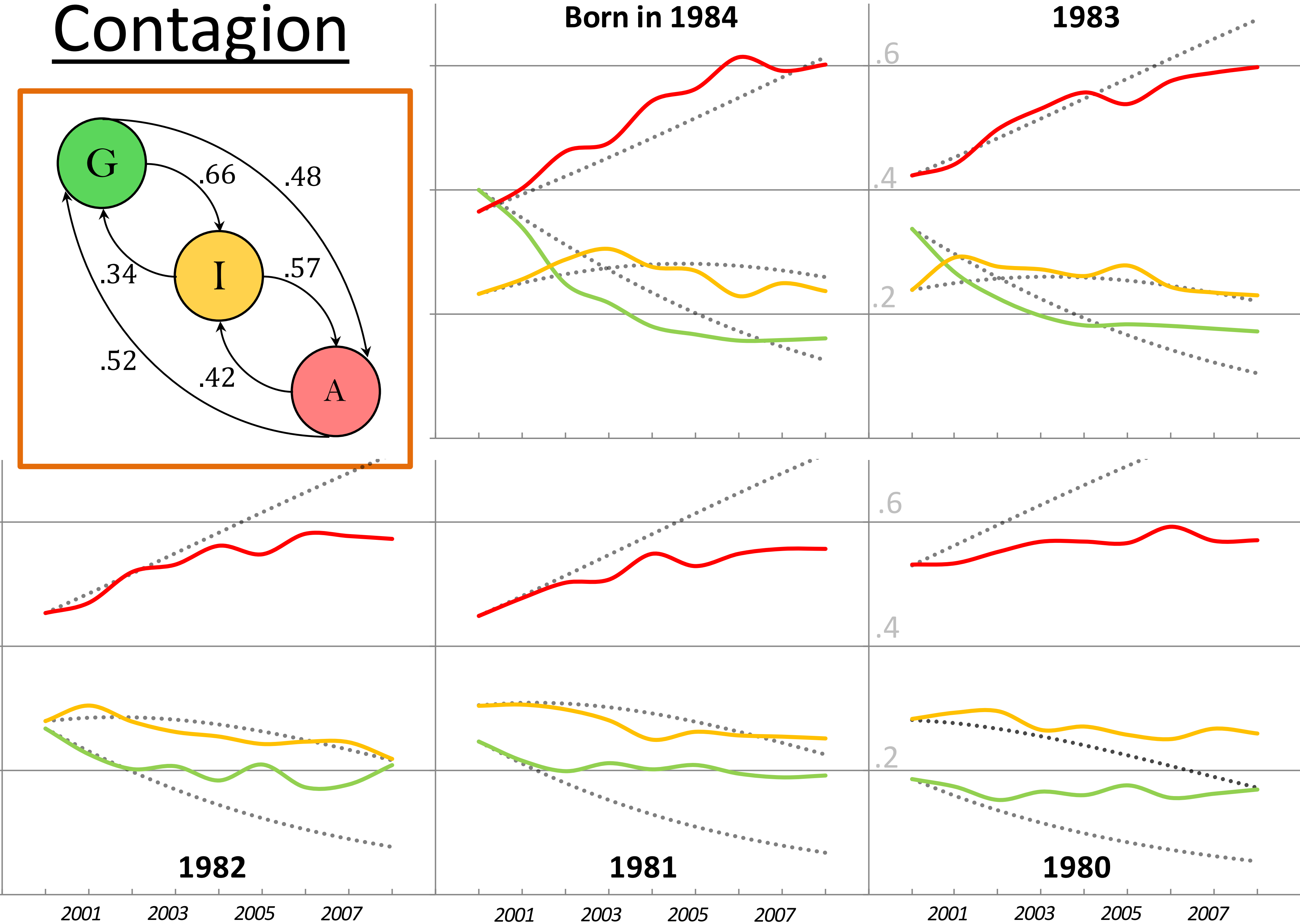
Bayesian MCMC
Chains : 5
Min Burn-In : 5,000
Min Retained: 15,000

Cohort	PSRF	$max\ \hat{R}$
1980	1.00	1.00
1981	1.00	1.00
1982	1.00	1.00
1983	1.00	1.00
1984	1.00	1.00





Contagion

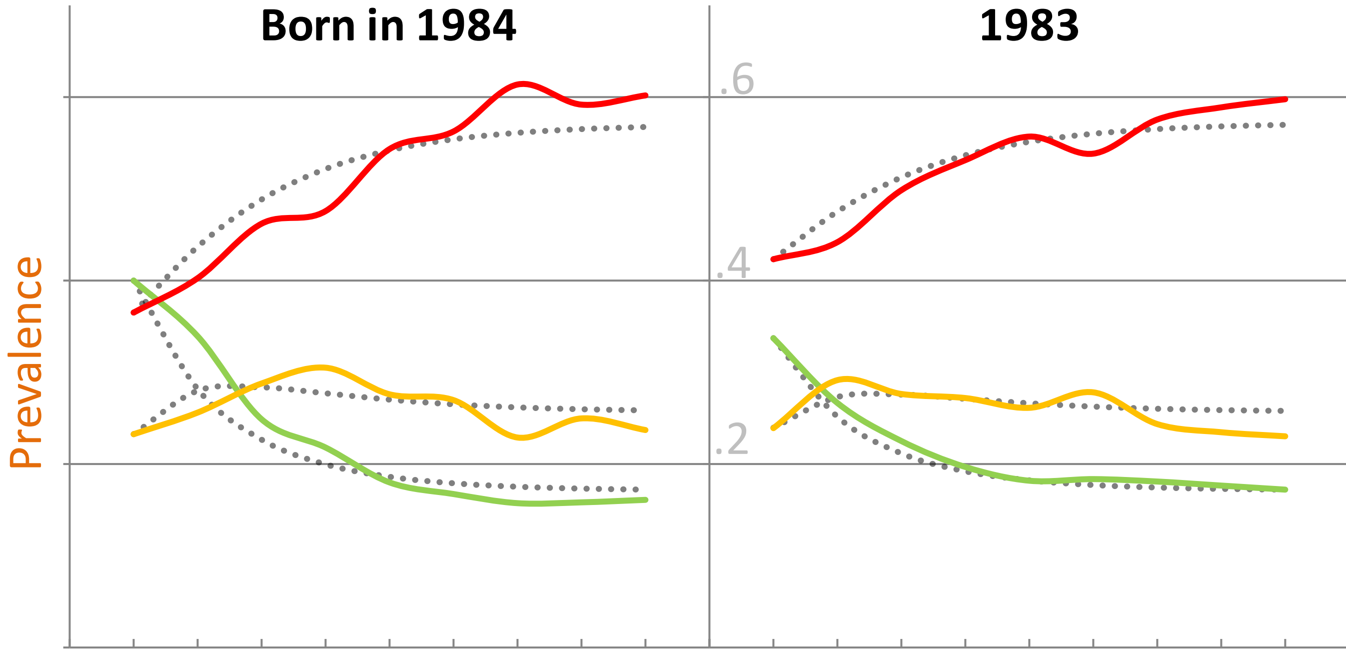


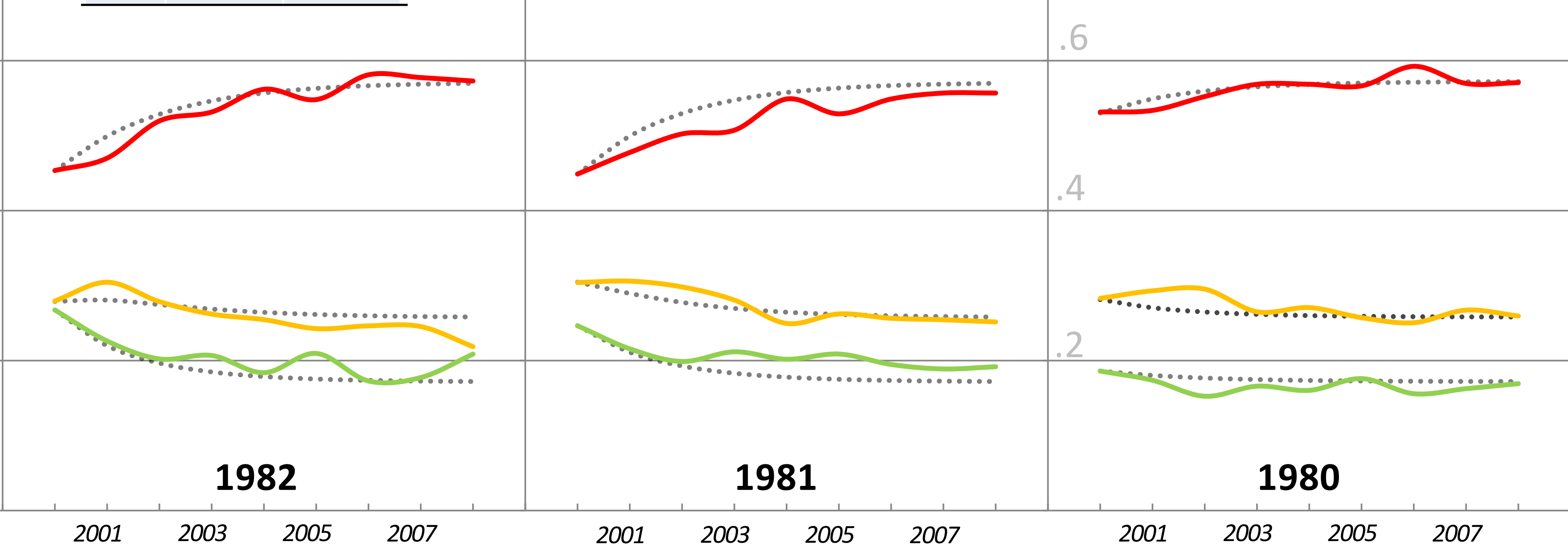
G
I
A
.66
.48
.34
.57
.52
.42
Born in 1984
1983
1982
1981
1980
.6
.4
.2
2001
2003
2005
2007

Diffusion

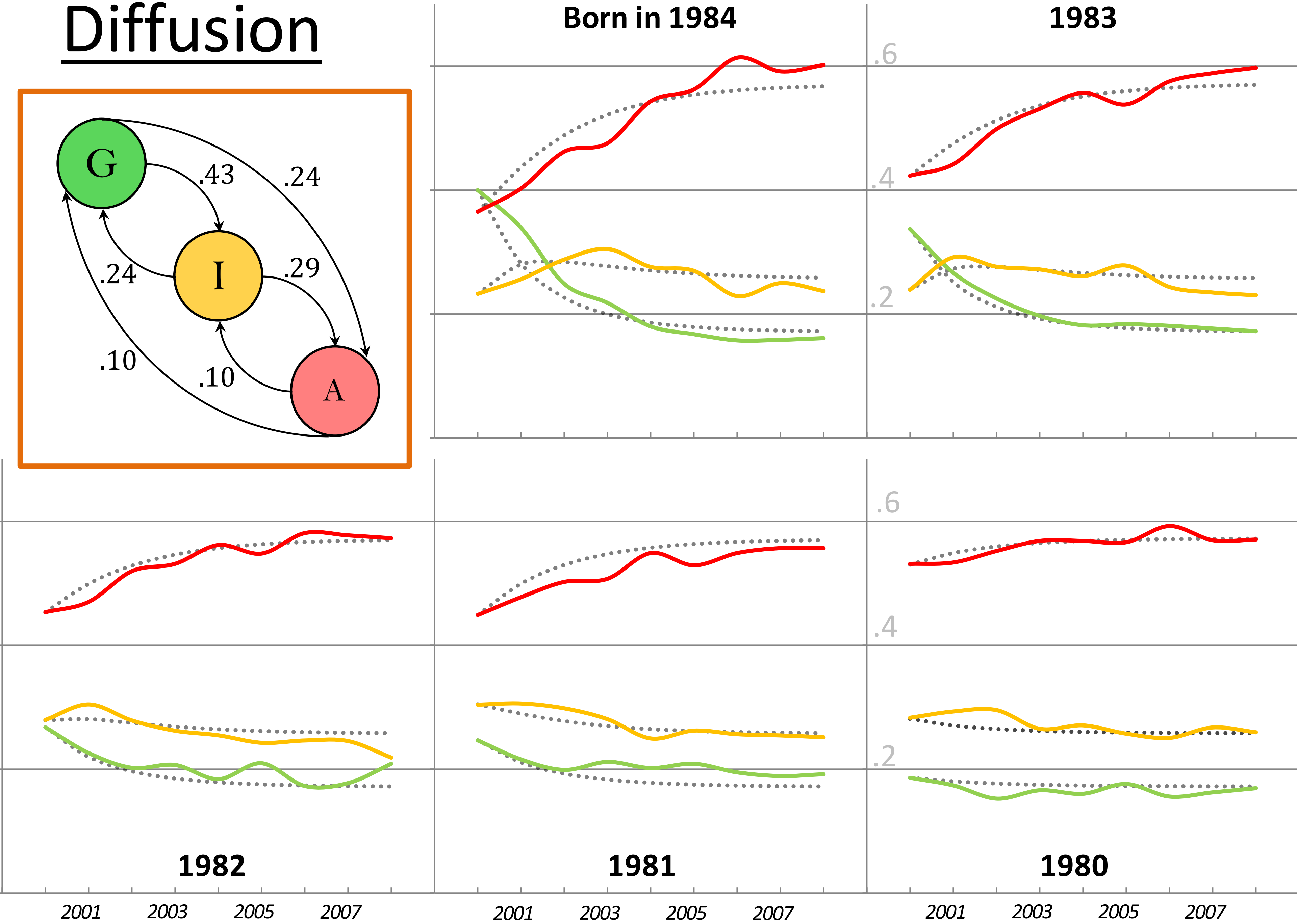
Bayesian MCMC
Chains : 5
Min Burn-In : 5,000
Min Retained: 15,000

Cohort	PSRF	$max\ \hat{R}$
1980	1.01	1.03
1981	1.01	1.03
1982	1.01	1.02
1983	1.00	1.00
1984	1.00	1.00





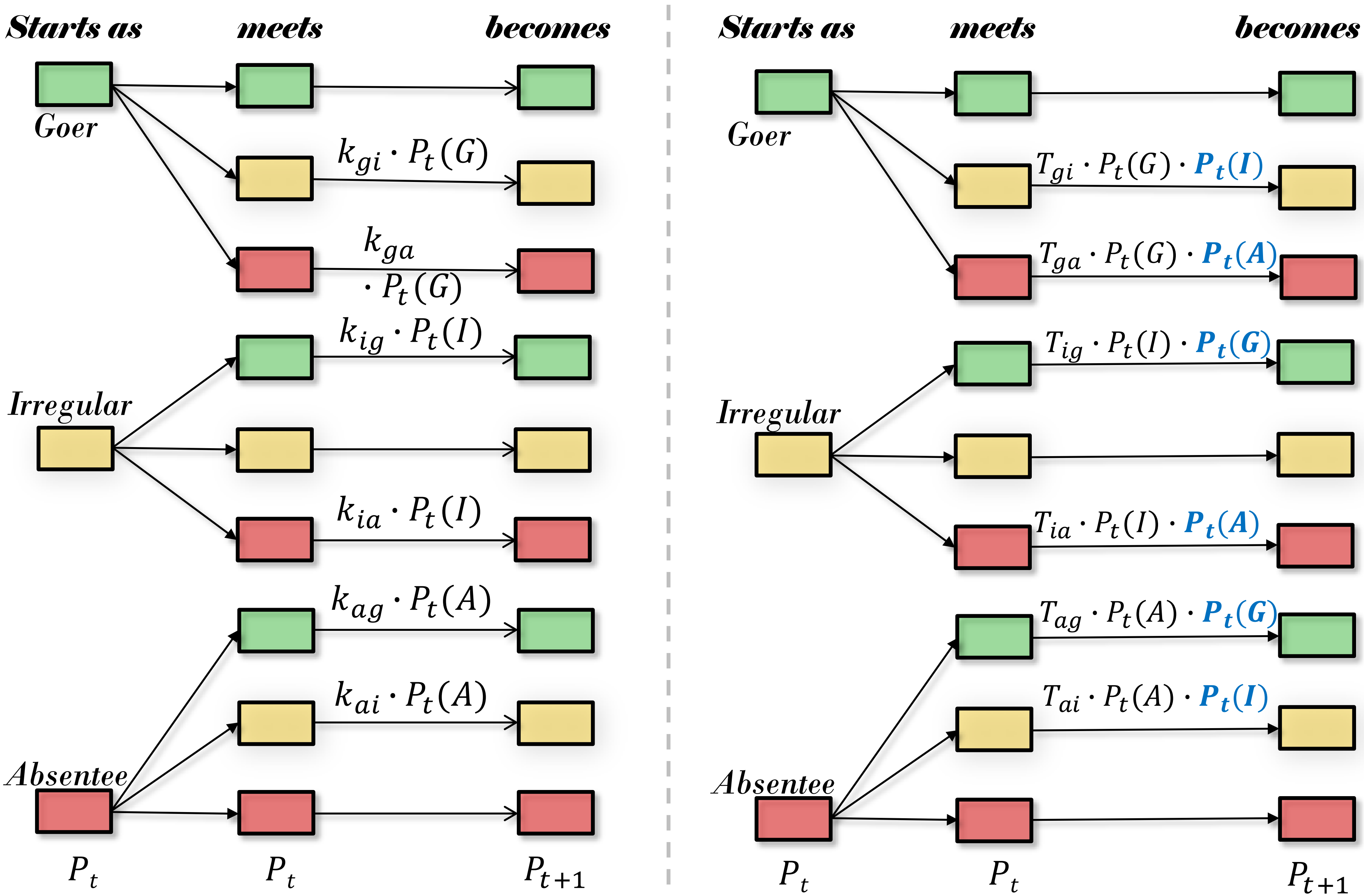
Diffusion


G
I
A
.43
.24
.24
.29
.10
.10
Born in 1984
1983
1982
1981
1980
.6
.4
.2
2001
2003
2005
2007

Do we have a winner?

- Diffusion model works, but…
- Simply better than Contagion
- Treats all transitions as having the same nature
- Flashback to smoking model: some processes might have different mode of spreading
- Closer look at Diffusion vs. Contagion

Diffusion vs. Contagion



Closer look:

Diffusion vs. Contagion

$$
\begin{cases}
P_{t+1}(G) = P_t(G) + k_{ig} \cdot P_t(I) + k_{ag} \cdot P_t(A) \\
\qquad\qquad - k_{gi} \cdot P_t(G) - k_{ga} \cdot P_t(G) \\
P_{t+1}(I) = P_t(I) + k_{gi} \cdot P_t(G) + k_{ai} \cdot P_t(A) \\
\qquad\qquad - k_{ig} \cdot P_t(I) - k_{ia} \cdot P_t(I) \\
P_{t+1}(A) = 1 - P_{t+1}(G) - P_{t+1}(I)
\end{cases}
$$

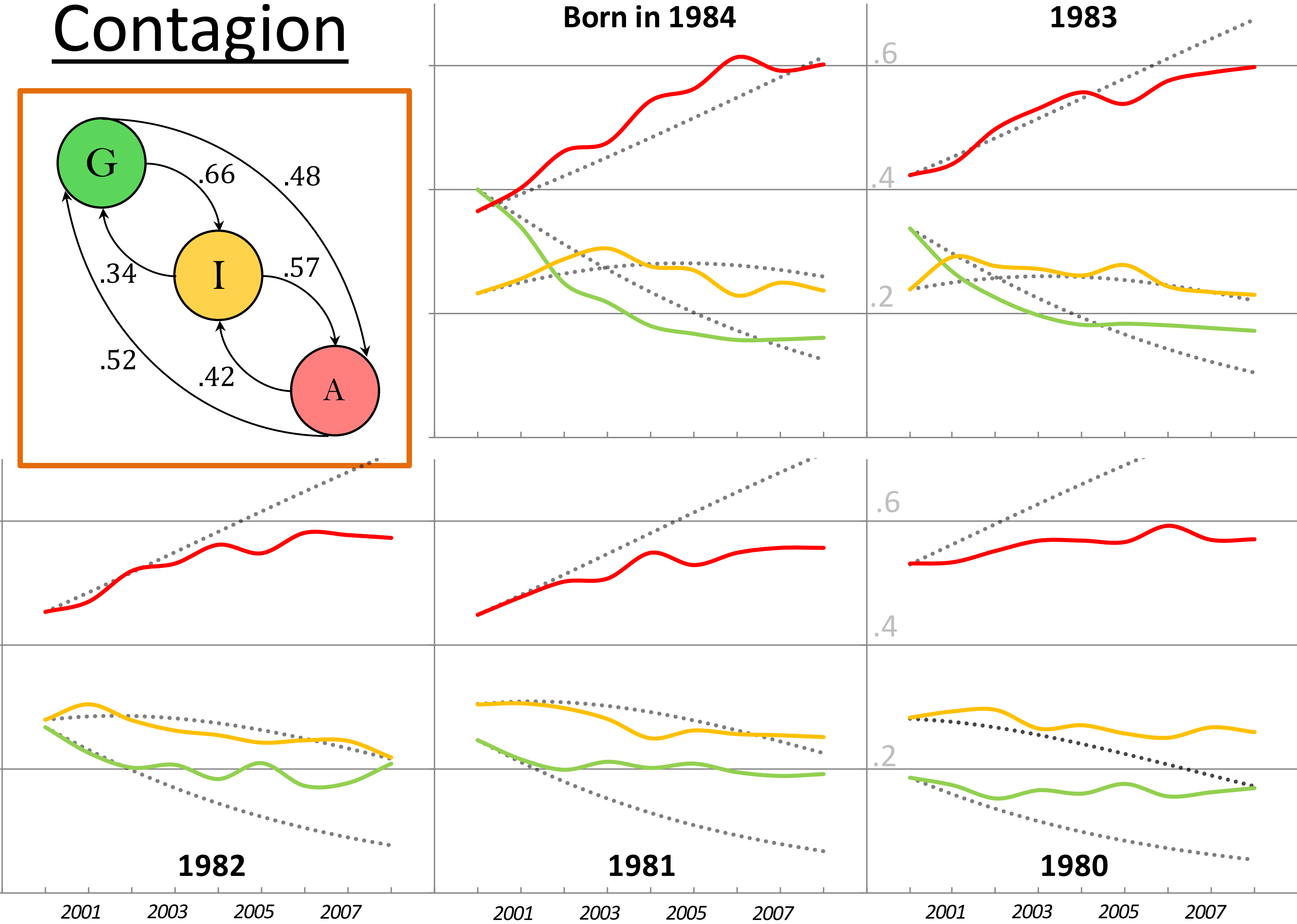
The difference between the models

$$
\begin{cases}
P_{t+1}(G) = P_t(G) + T_{ig} \cdot P_t(I) \cdot P_t(G) + T_{ag} \cdot P_t(A) \cdot P_t(G) \\
\qquad\qquad - T_{gi} \cdot P_t(G) \cdot P_t(I) - T_{ga} \cdot P_t(G) \cdot P_t(A) \\
P_{t+1}(I) = P_t(I) + T_{gi} \cdot P_t(G) \cdot P_t(I) + T_{ai} \cdot P_t(A) \cdot P_t(I) \\
\qquad\qquad - T_{ig} \cdot P_t(I) \cdot P_t(G) - T_{ia} \cdot P_t(I) \cdot P_t(A) \\
P_{t+1}(A) = 1 - P_{t+1}(G) - P_{t+1}(I)
\end{cases}
$$

Hybrid Model

$$\begin{cases} P_{t+1}(G) = P_t(G) + T_{ig} \cdot P_t(I) \cdot P_t(G)^{C_{ig}} + T_{ag} \cdot P_t(A) \cdot P_t(G)^{C_{ag}} \\ \qquad - T_{gi} \cdot P_t(G) \cdot P_t(I)^{C_{gi}} - T_{ga} \cdot P_t(G) \cdot P_t(A)^{C_{ga}} \\ \\ P_{t+1}(I) = P_t(I) + T_{gi} \cdot P_t(G) \cdot P_t(I)^{C_{gi}} + T_{ai} \cdot P_t(A) \cdot P_t(I)^{C_{ai}} \\ \qquad - T_{ig} \cdot P_t(I) \cdot P_t(G)^{C_{ig}} - T_{ia} \cdot P_t(I) \cdot P_t(A)^{C_{ia}} \\ \\ P_{t+1}(A) = 1 - P_{t+1}(G) - P_{t+1}(I) \end{cases}$$

Contagion


G
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.66
.48
.34
.57
.52
.42
Born in 1984
1983
1982
1981
1980
.6
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.2
2001
2003
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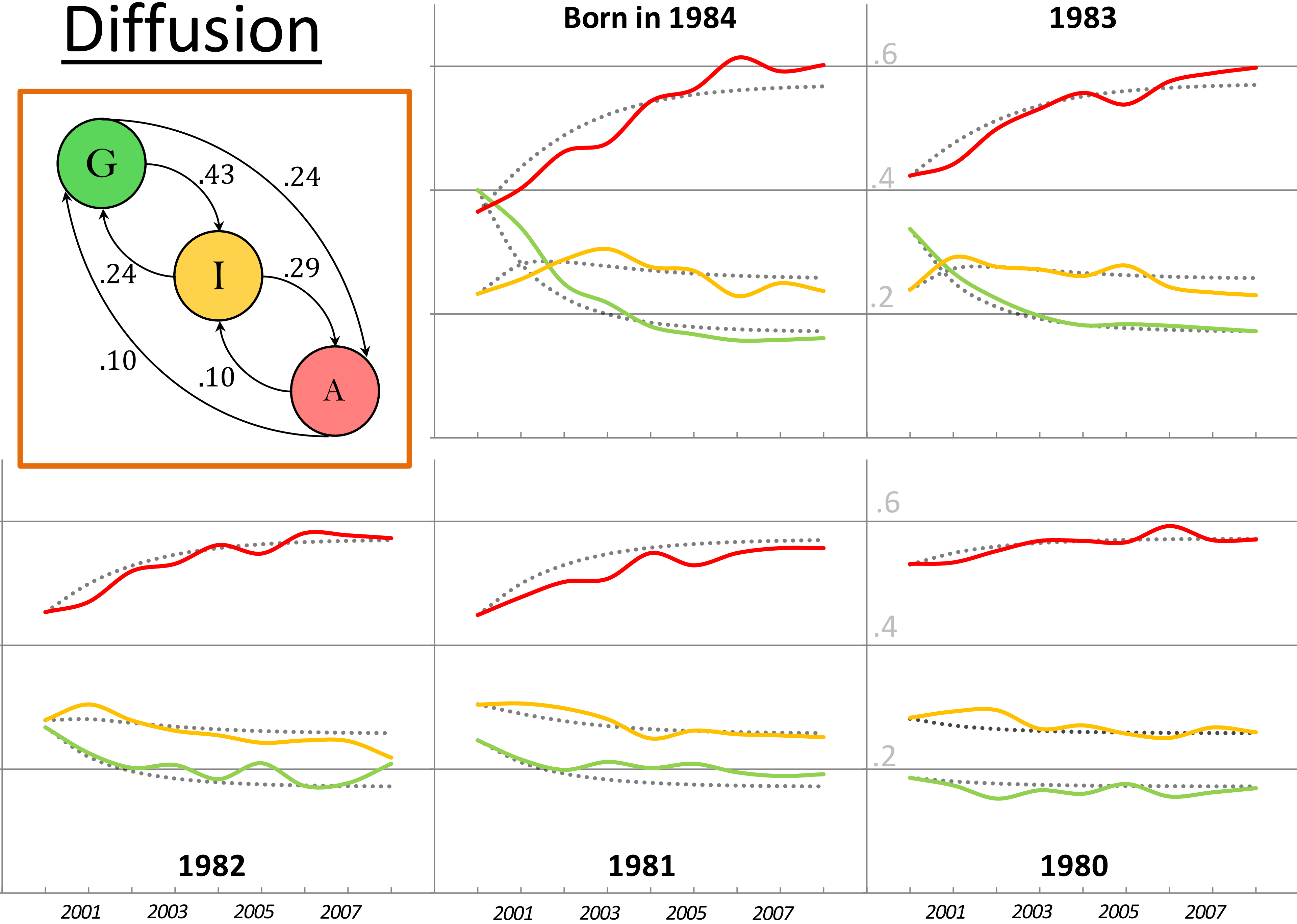
2007

Diffusion



G
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.43
.24
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Born in 1984
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1982
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1980
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2003
2005
2007

Hybrid

Bayesian MCMC
Chains : 5
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Min Retained: 15,000

Cohort	PSRF	$max \hat{R}$
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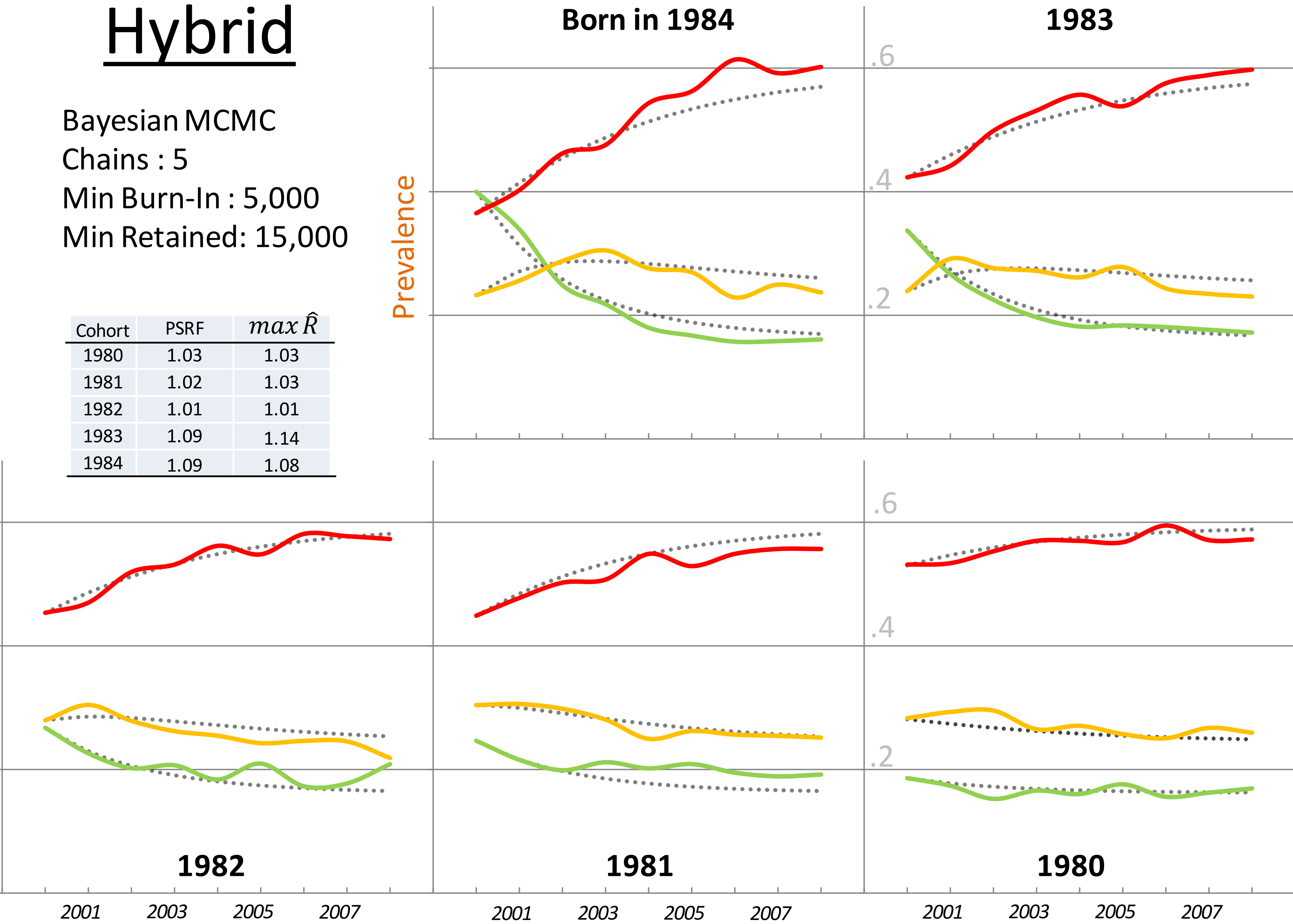
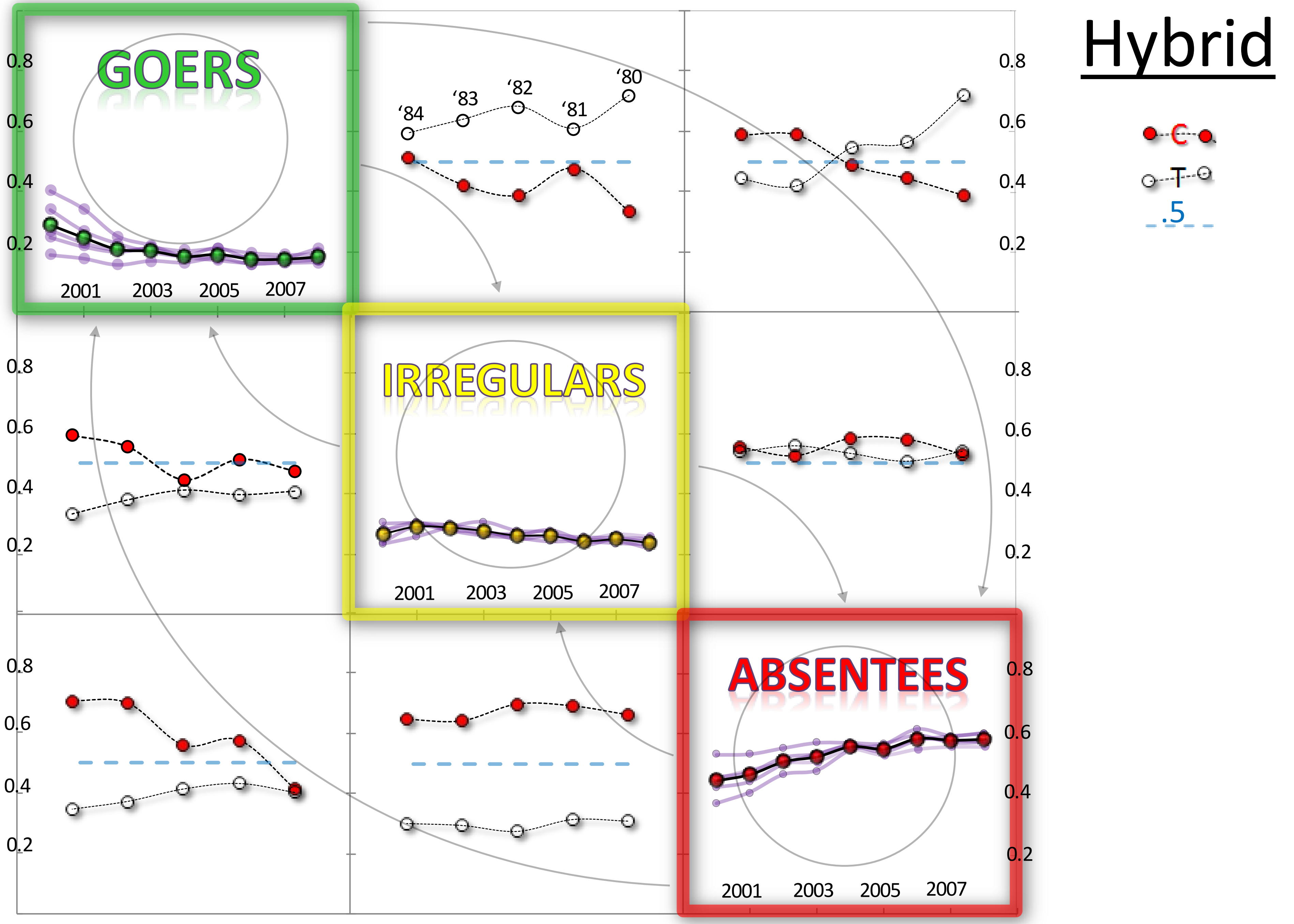
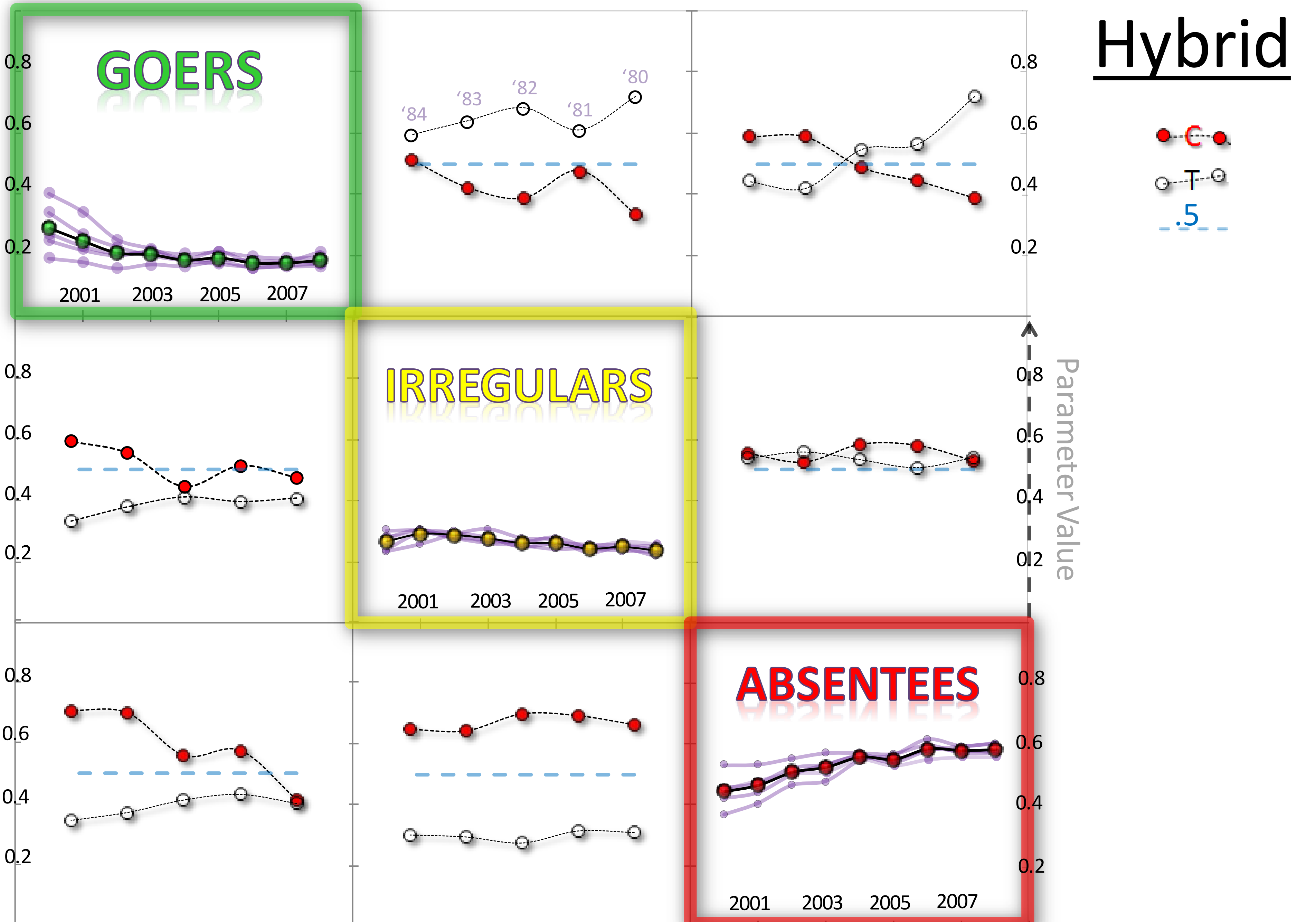
1981	1.02	1.03
1982	1.01	1.01
1983	1.09	1.14
1984	1.09	1.08





Hybrid
C
T
.5
GOERS
IRREGULARS
ABSENTEES
'84
'83
'82
'81
'80
0.8
0.6
0.4
0.2
2001
2003
2005
2007



Hybrid
C
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.5
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0.8
0.6
0.4
0.2
2001
2003
2005
2007
'84
'83
'82
'81
'80
Parameter Value

Substantive Conclusions

- Abrupt changes in religiosity are getting less contagious with age
- Moderate increase in religiosity has an noticeable contagious component
- Overall: Change in religious behavior tends to become less contagious with age

Methodological Conclusions

- EMOSA works for 3-categories
  - Free transitions among categories
  - Mixed model = most illuminating
  - Good convergences, stability
  - Verified by simulation studies
  - Reproduces data well
  - C-parameters can tell the nature of the transition
- Successful novel application
  - Can plug in other behaviors

Q/A