

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

Joe Rodgers & Andriy Koval
Lunch Bunch, March 11, 2011

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 – **Andrey Koval**, current OU Quant grad student

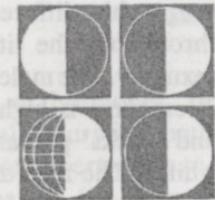
Introduction

- EMOSA Models – Epidemic Models of the Onset of Social Activities
- 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

Here's our first
EMOSA paper,
applied to the
onset of sexual
behavior – Social
Biology, 1989

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



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^bDepartment 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.

- We developed theory in a 1993 Psych Review paper and a 1998 Developmental Psych paper
- We applied EMOSA to smoking and drinking in a 1993 paper, and in two book chapters in 2007
- We applied EMOSA to crime and delinquency in another paper
- All along we've claimed that EMOSA models could be used for behaviors other than adolescent problem behaviors – now we're doing that, and will report results today
- But first, some background

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:

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

↑ ↑ ↑
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

'There is actually one of these equations for each sex:

$$P_{m(t+1)} = P_{mt} + T_m (P_{ft}) (1-P_{mt}) + k (1- P_{mt}) (1-P_{ft})$$

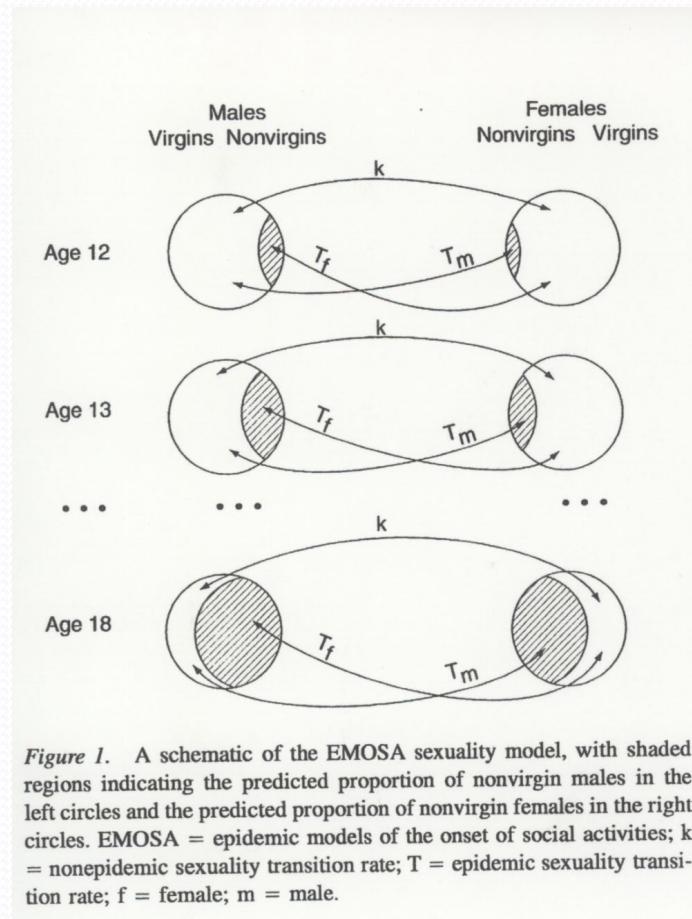
$$P_{f(t+1)} = P_{ft} + T_f (P_{mt}) (1-P_{ft}) + k (1- P_{ft}) (1-P_{mt})$$

Notice how the two equations communicate with one another

These equations tell a story, they define a model –

Here's another more conceptually straightforward look at the model

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



Fitting the first EMOSA Model to the ADSEX data from NC & FL

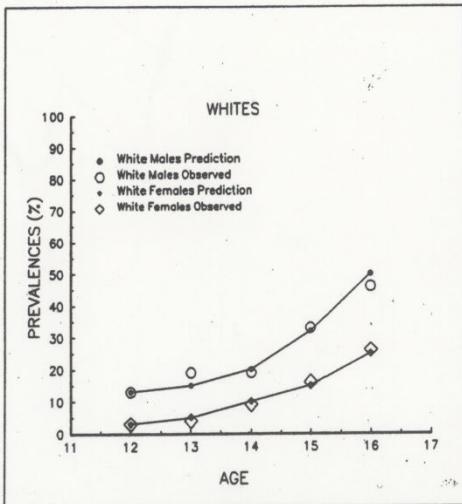
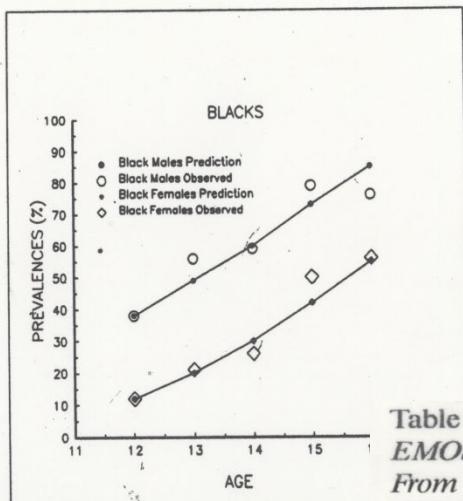


Figure 1. Raw ADSEX data and fitted Epidemic Modeling of the Onset of Social Activities intercourse-prevalence predictions from Rowe, Rodgers, and Meseck-Bushey (1989).



JOSEPH LEE RODGERS AND DAVID C.

Table 1
EMOSA Fit Statistics and Parameter Values Obtained From Fitting the ADSEX Data

Model	χ^2	df	T_m	T_f	k
No maturation					
Whites	9.6	5	1.00	0.23	0.04
Blacks	4.3	5	0.96	0.26	0.04
Whites and Blacks	14.1	13	1.00	0.25	0.04
Maturation					
Whites	5.7	5	0.90	0.22	0.10
Blacks	3.3	5	0.78	0.31	0.14
Whites and Blacks	10.3	13	1.00	0.33	0.08

Note. EMOSA = Epidemic Modeling of the Onset of Social Activities; T_m = male epidemic transition parameter; T_f = female epidemic transition parameter; k = nonepidemic transition parameter. From "An 'Epidemic' Model of Sexual Intercourse Prevalences for Black and White Adolescents" by D. C. Rowe, J. L. Rodgers, and S. Meseck-Bushey, 1989, *Social Biology*, 50, p. 136. Copyright 1989 by the Society for the Study of Social Biology. Adapted by permission.

Important findings

- Black/White differences in sexual behavior prevalences can be fully accounted for by pubertal development
- Transition probabilities are typically interpretable -- higher for females-converting-males than vice versa, relatively low non-epidemic transition
- Approximately constant probability of pregnancy across age -- contraceptive effectiveness and coital frequency compensatory?

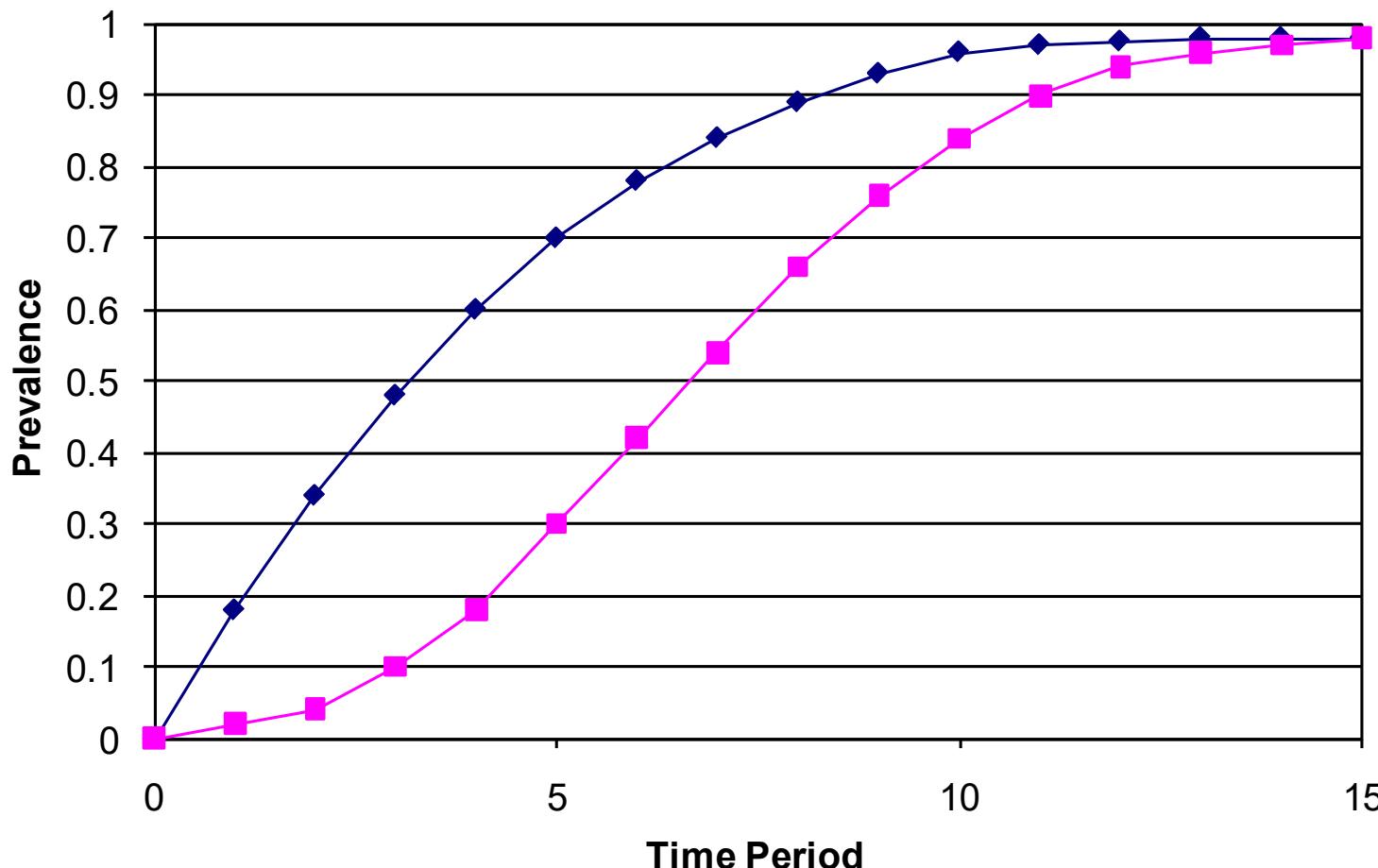
EMOSA Smoking and Drinking Models

- 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 conation versus General diffusion becomes important in the smoking/drinking models

- Social contagion – social influence is passed from person to person
- General diffusion – social influence is exerted through broader communication channels, like media, advertising, literature, 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

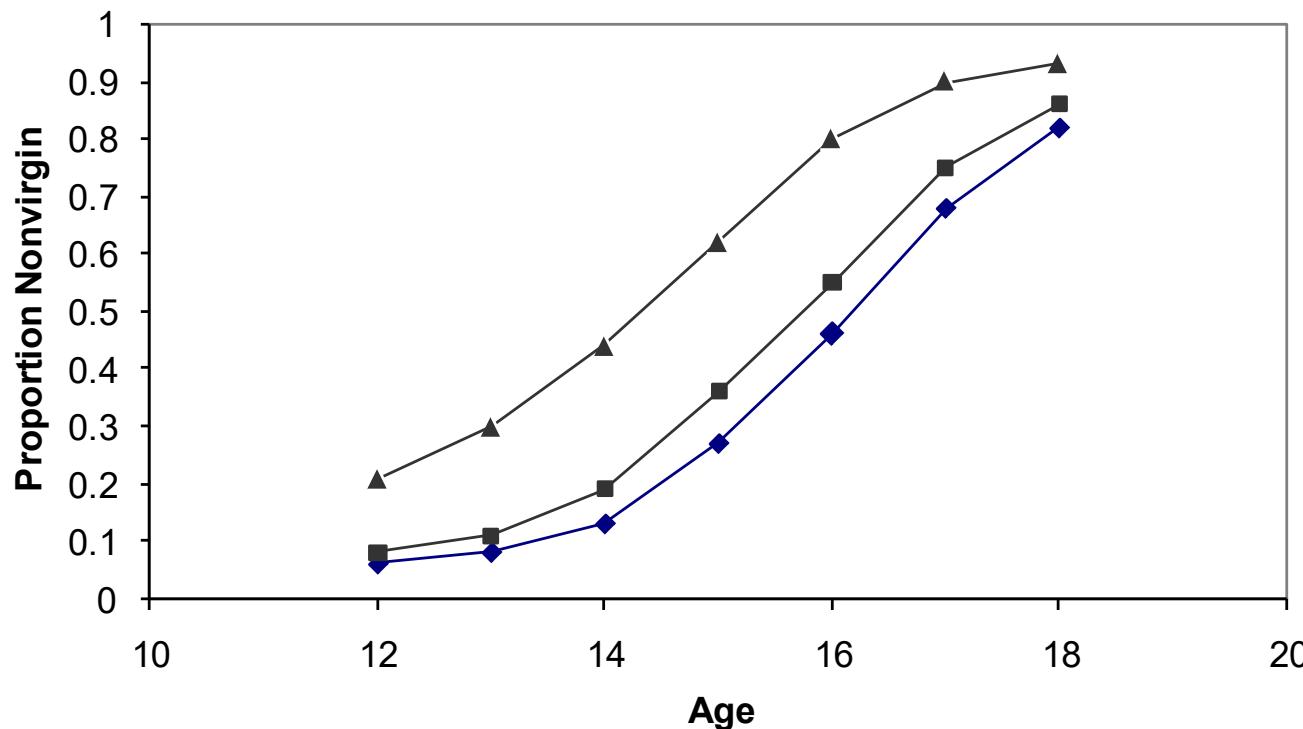
- These two processes can be distinguished within our mathematical models
- Specifically, the shape of the prevalence curve is indicative of which process is at work
 - S-shaped curve – social contagion
 - Negatively accelerated exponential – general diffusion
- Here are some graphical portrayals to illustrate, including some real data

Diffusion Curves: Diffusion without Contagion (Top Curve) and Diffusion with Contagion (Bottom Curve)



From Rodgers, Rowe, & Buster (1998)

1980 U.S. Male Sexuality Prevalences
Blacks (Top), Hispanics (Middle), Whites (Bottom)
Source: Rodgers, Rowe, & Buster (1998), p. 1103



From Rodgers & Johnson (2007)

**Smoking and Drinking Prevalence Curves, 2003 OU Survey,
Proportion Ever Drunk Alcohol (top) & Smoked a Cigarette (bottom)**



- Work by David Rowe (Rowe et al, 1994) showed that onset of smoking is all about social contagion
- But transition to regular smoking 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.

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

Some conclusions

- Nongoers are likely to stay goers. Highest resilience
- Goers rarely become Nongoers, they first become irregulars.
- Goers become irregulars before they become non-goers
- Nongoers never move back to goers, they become irregulars, for at least awhile
- Goers are the most resilient, but once out, lots happens



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?”

Goer

- | | |
|---|------------------------|
| 8 | 'Everyday' |
| 7 | 'Several times a week' |
| 6 | 'About once a week' |

Irregular

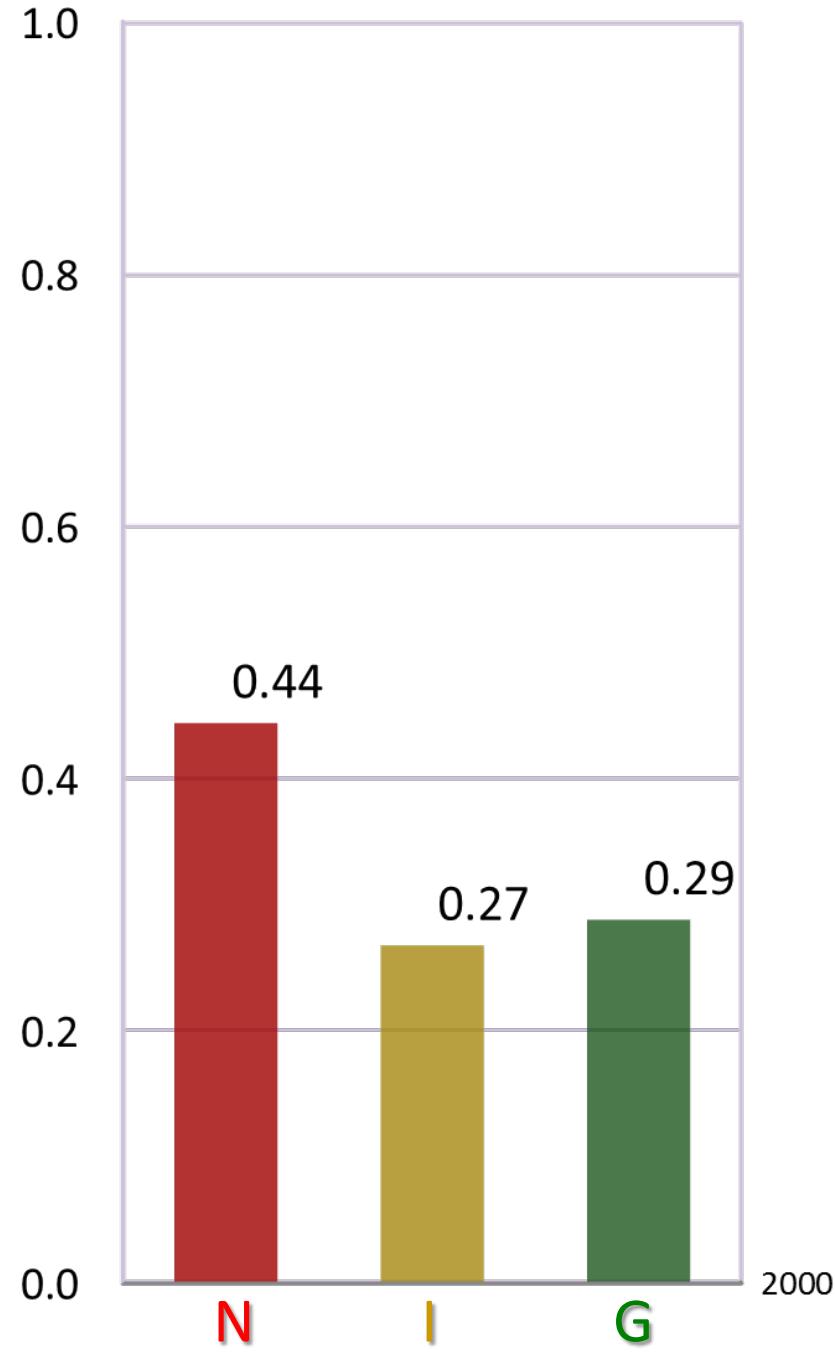
- | | |
|---|--------------------------|
| 5 | 'About twice a month' |
| 4 | 'About once a month' |
| 3 | 'Less than once a month' |

Non-goer

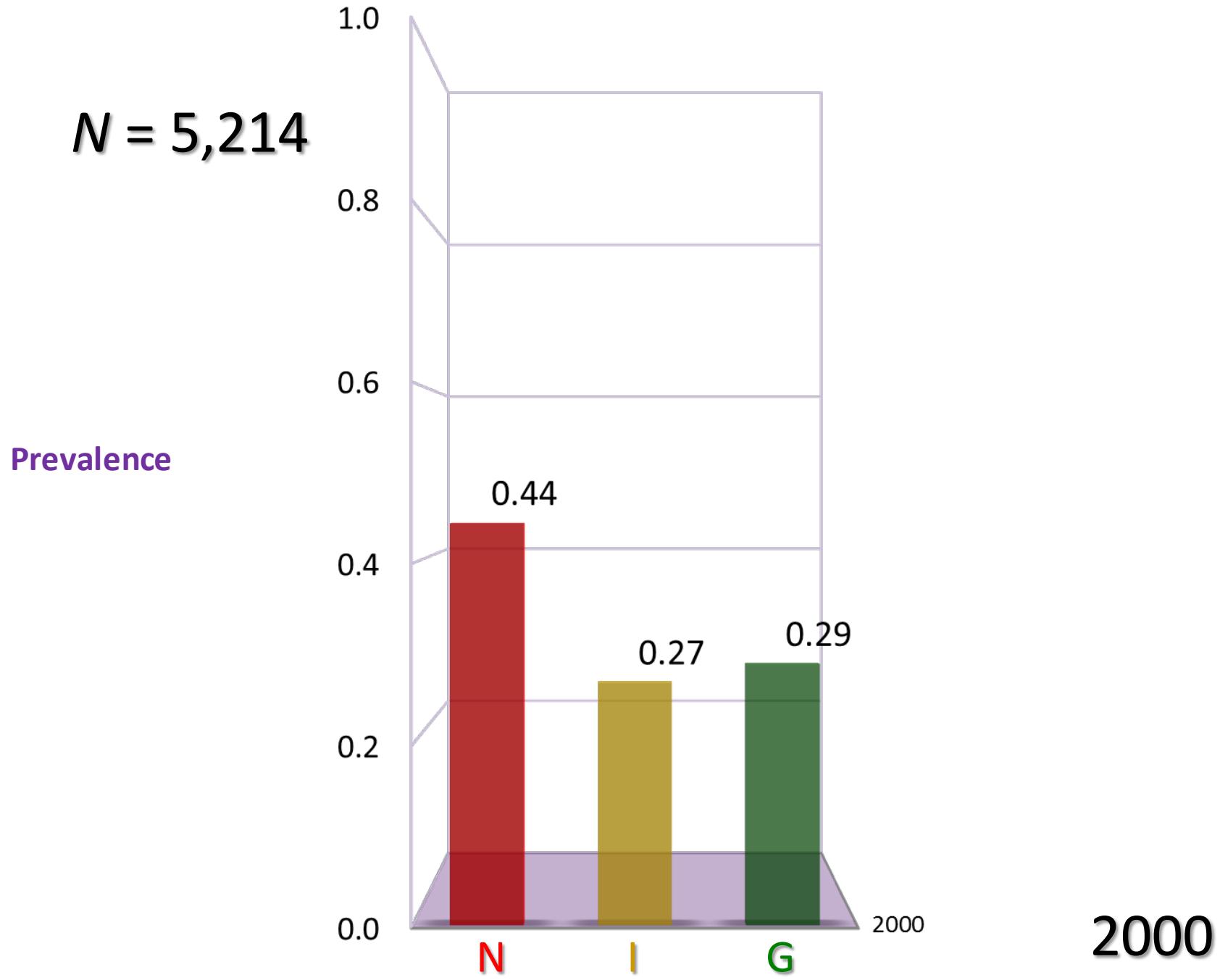
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|---|-----------------|
| 2 | 'Once or twice' |
| 1 | 'Never' |

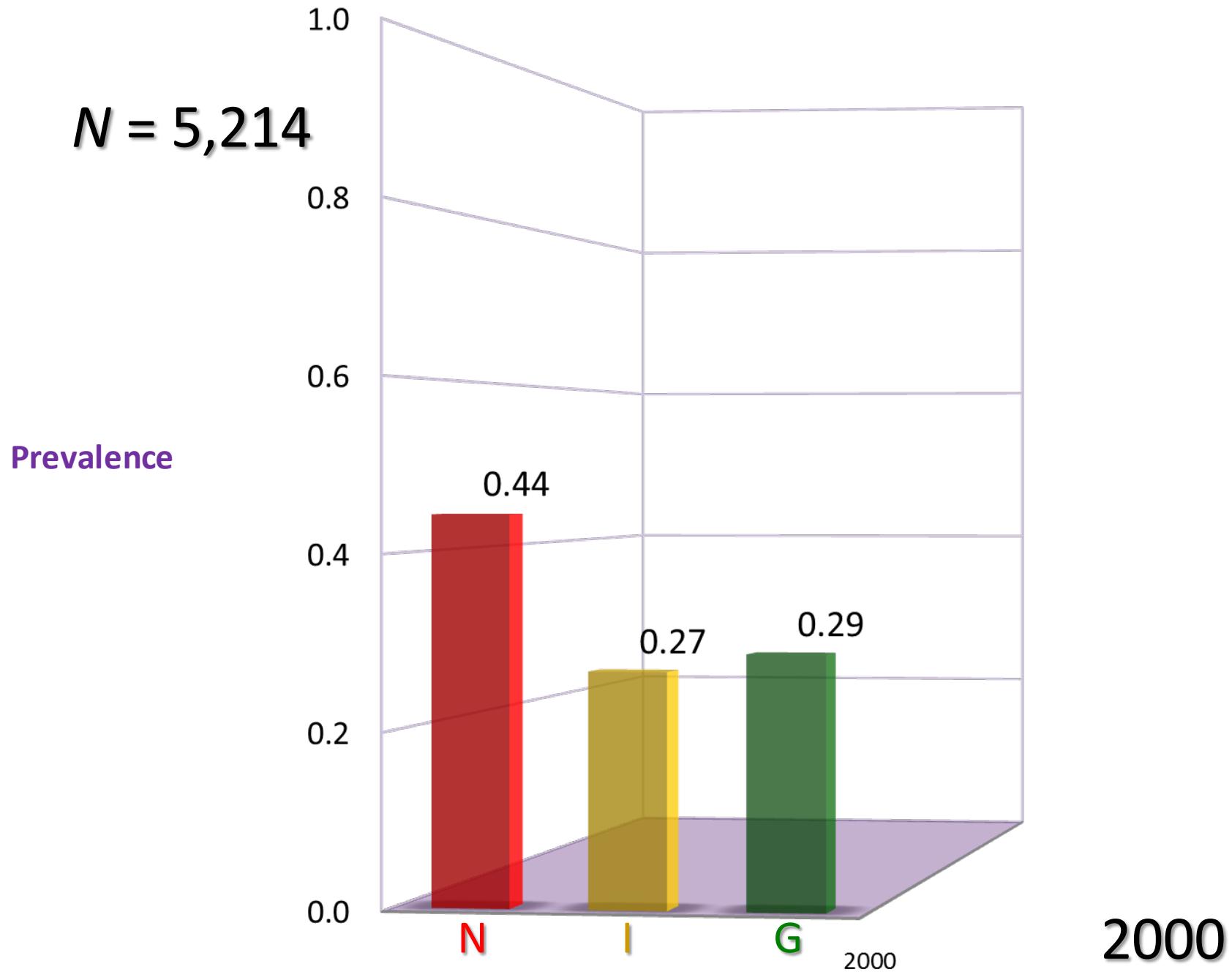
$N = 5,214$

Prevalence



2000





$N = 5,214$

Prevalence

0.8

0.6

0.4

0.2

0.0

0.44

0.27 0.29

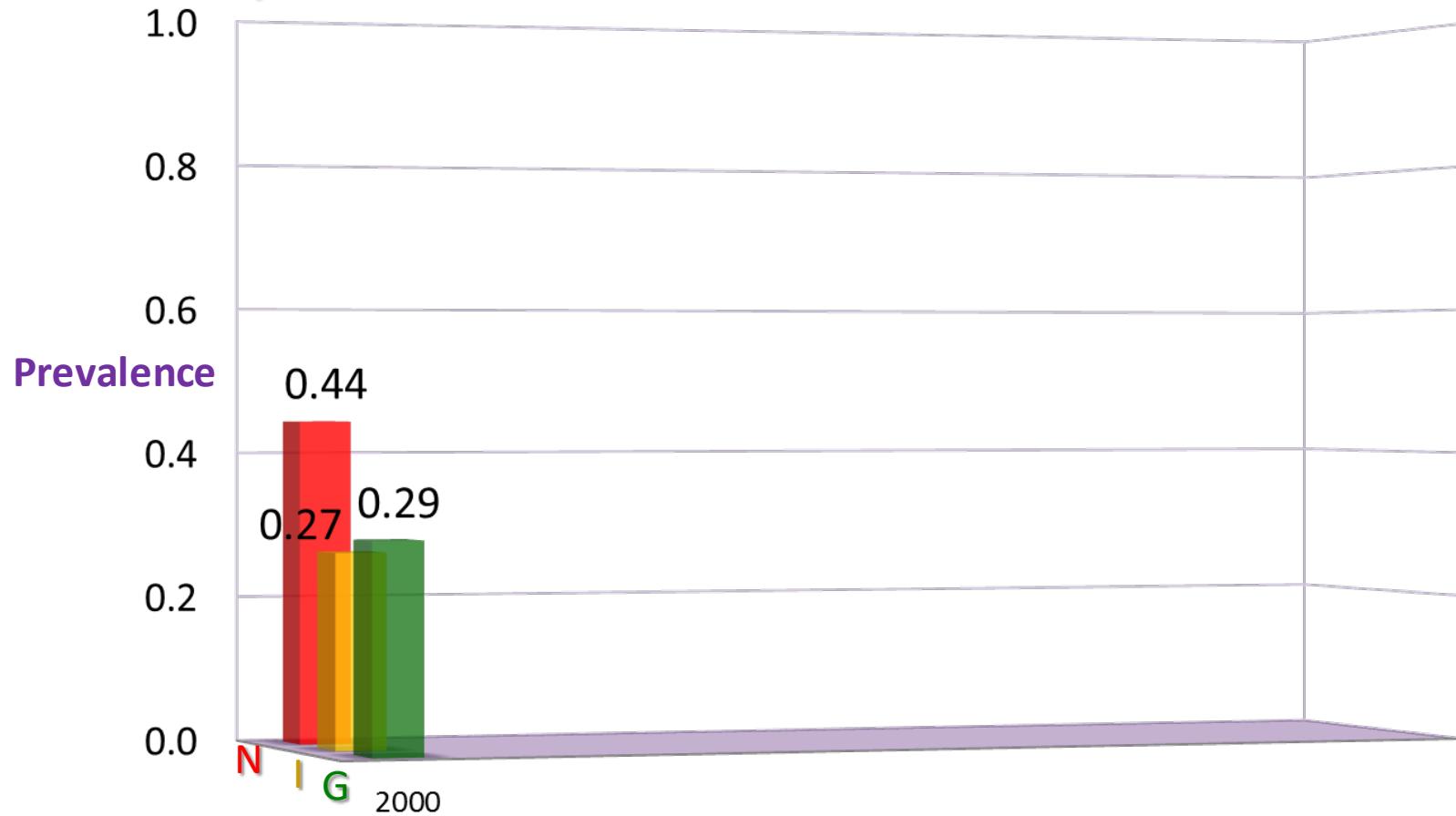
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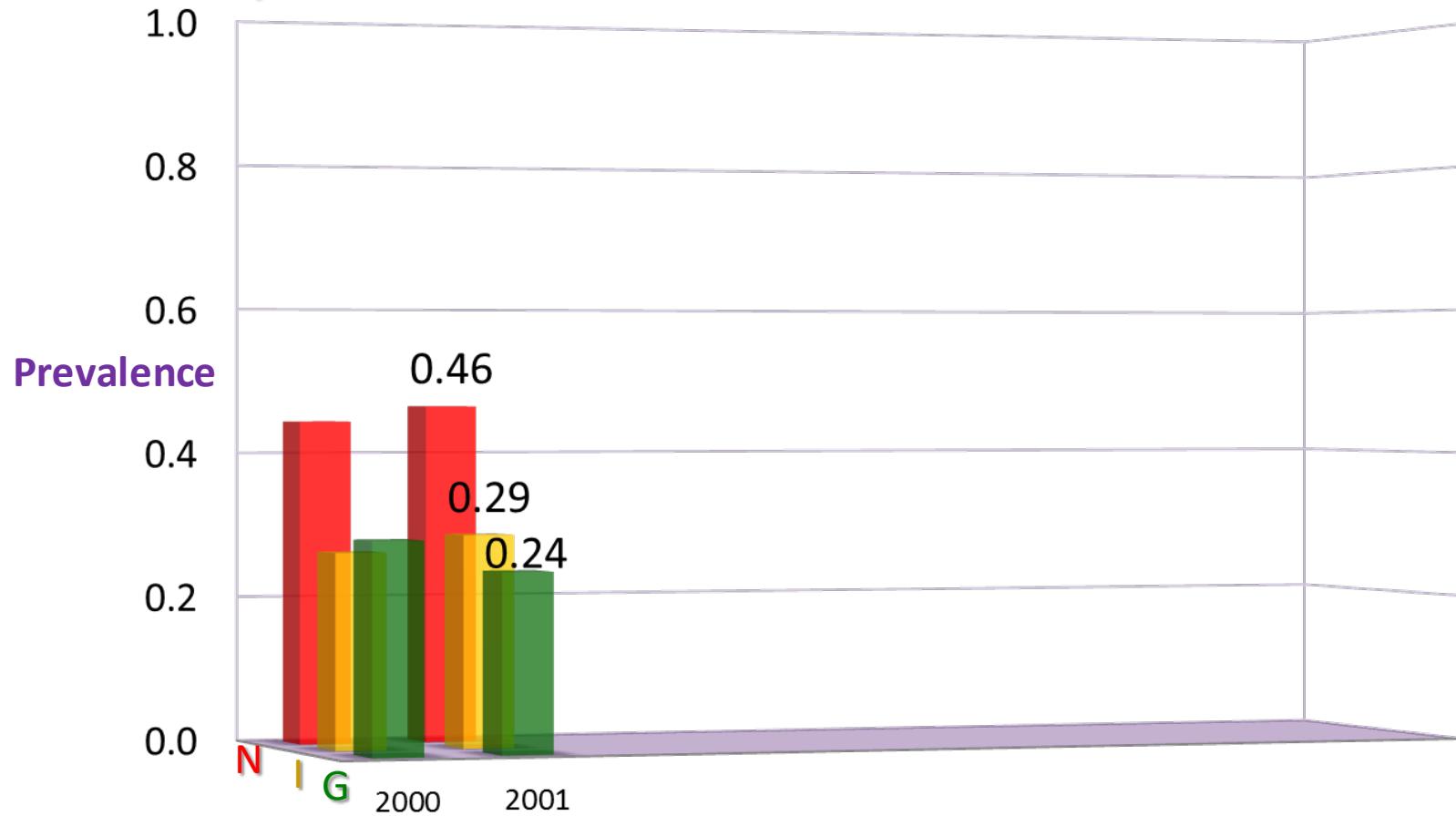
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2000

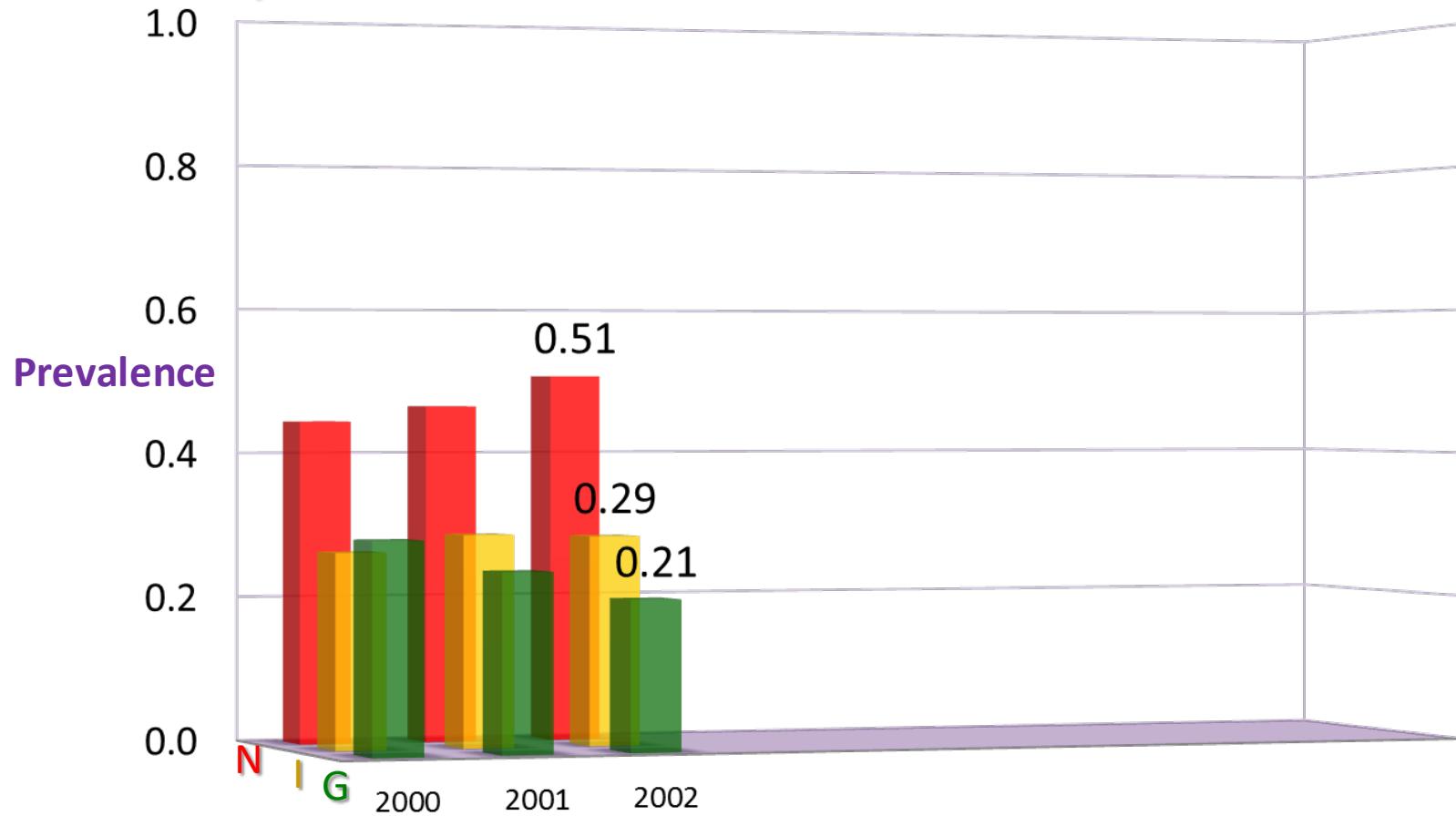
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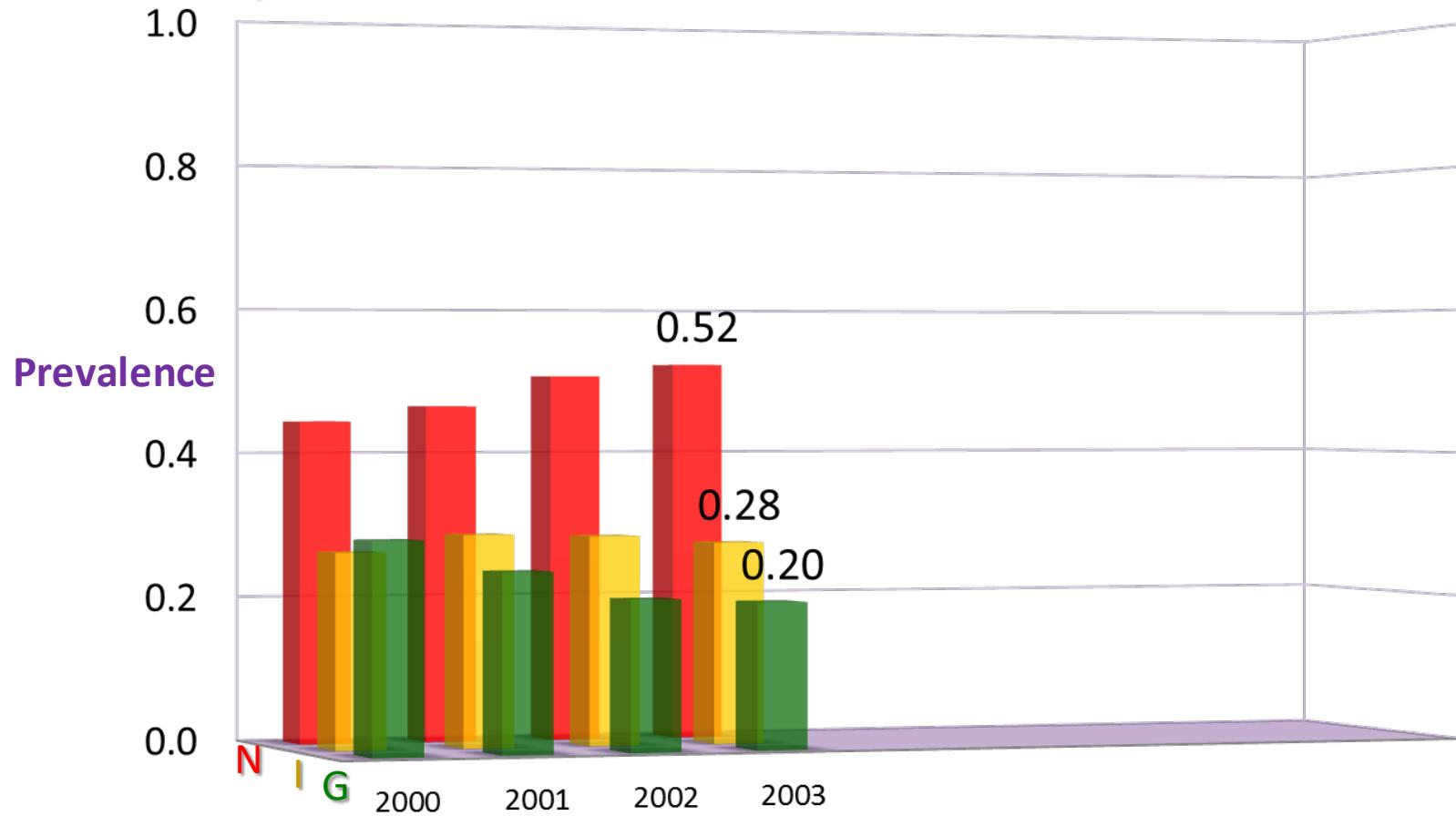
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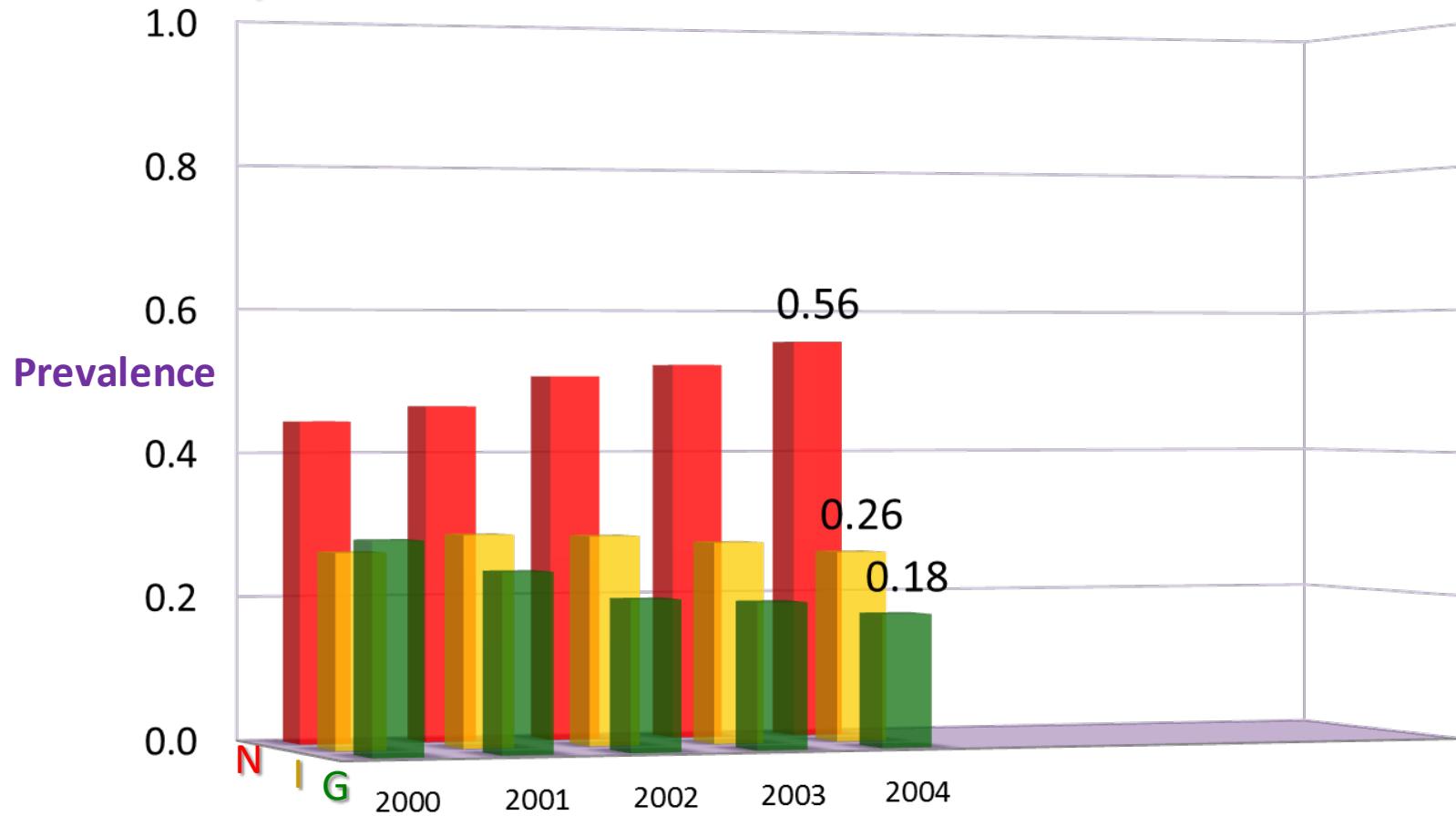
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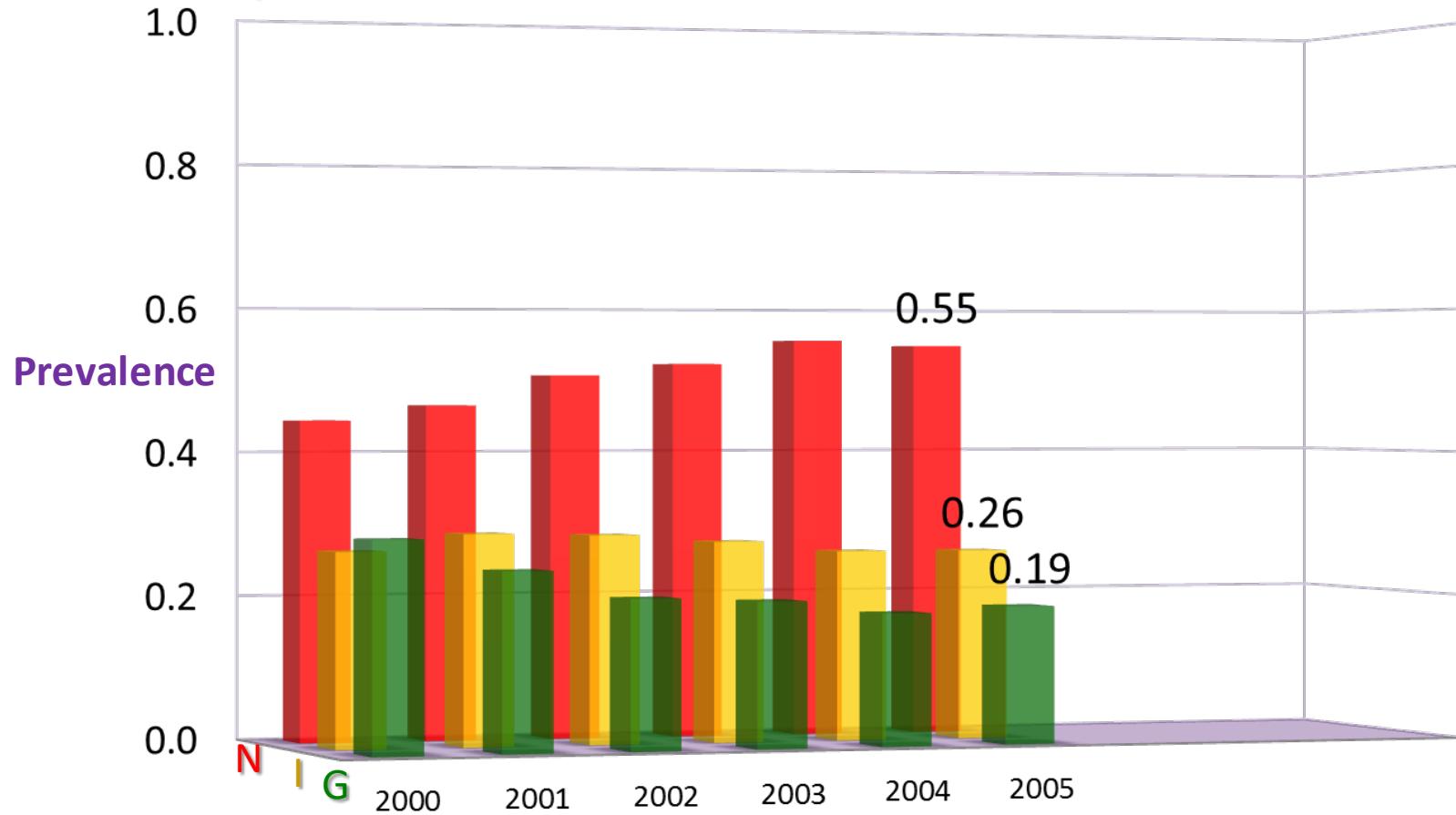
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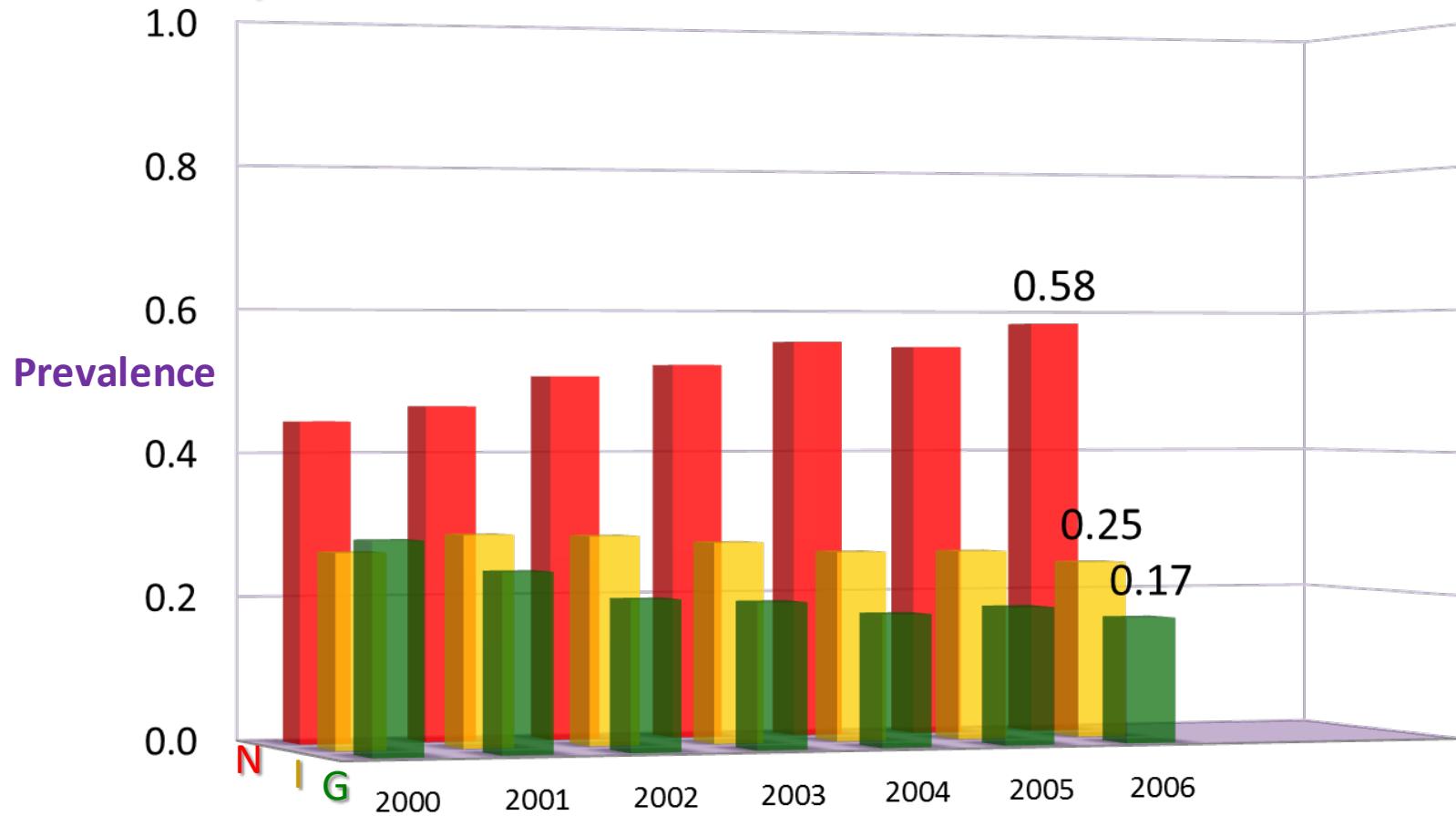
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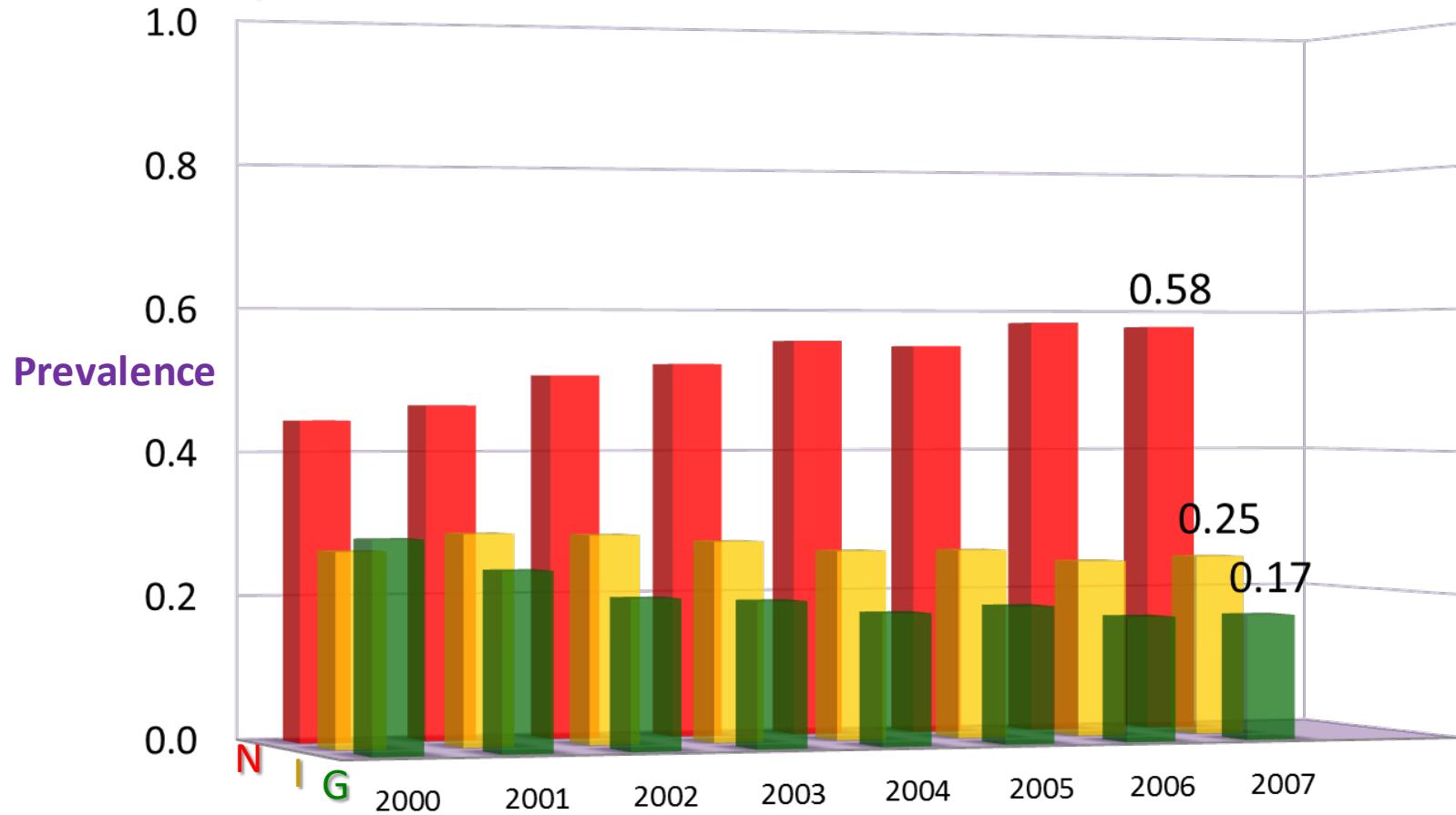
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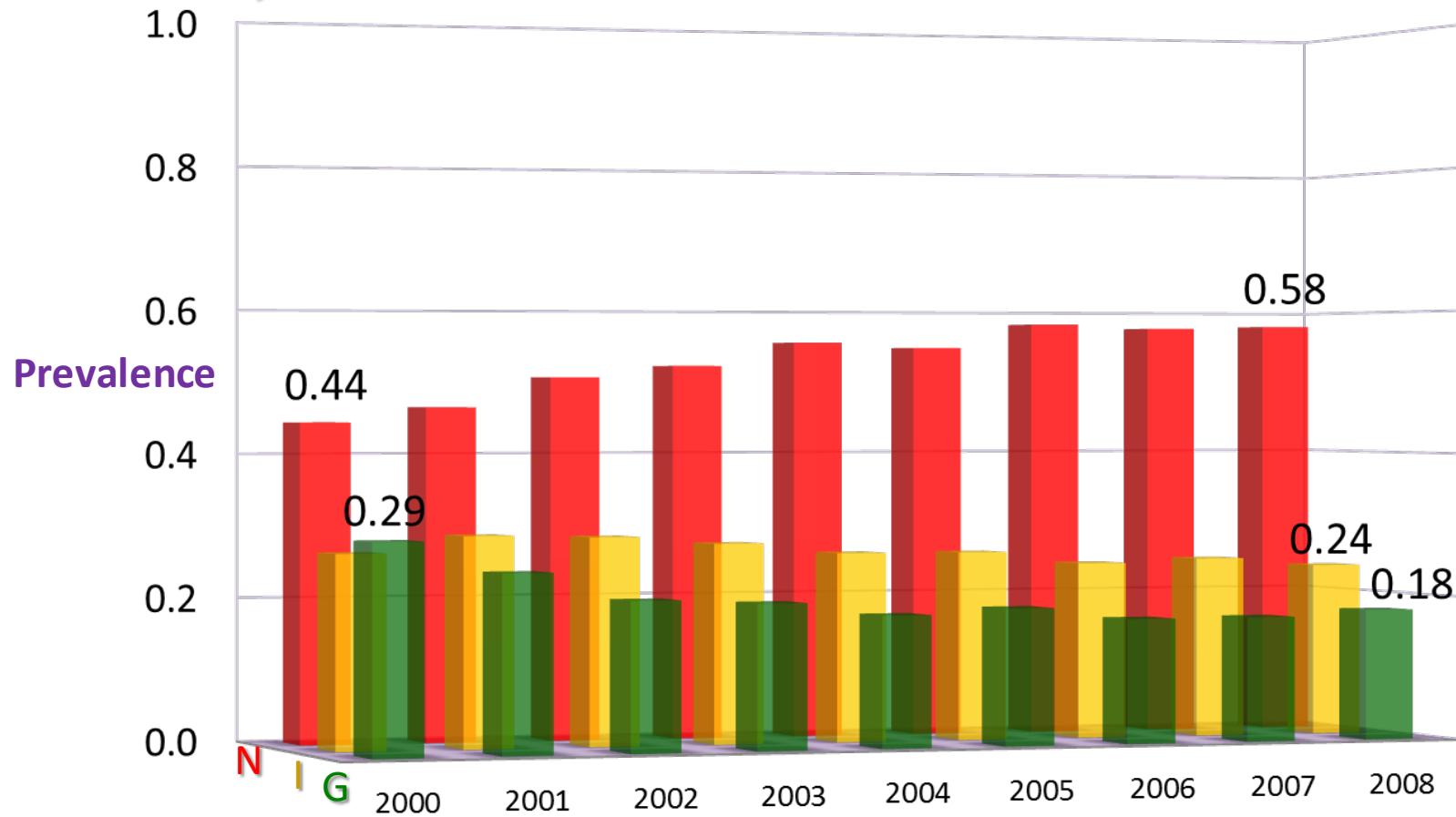
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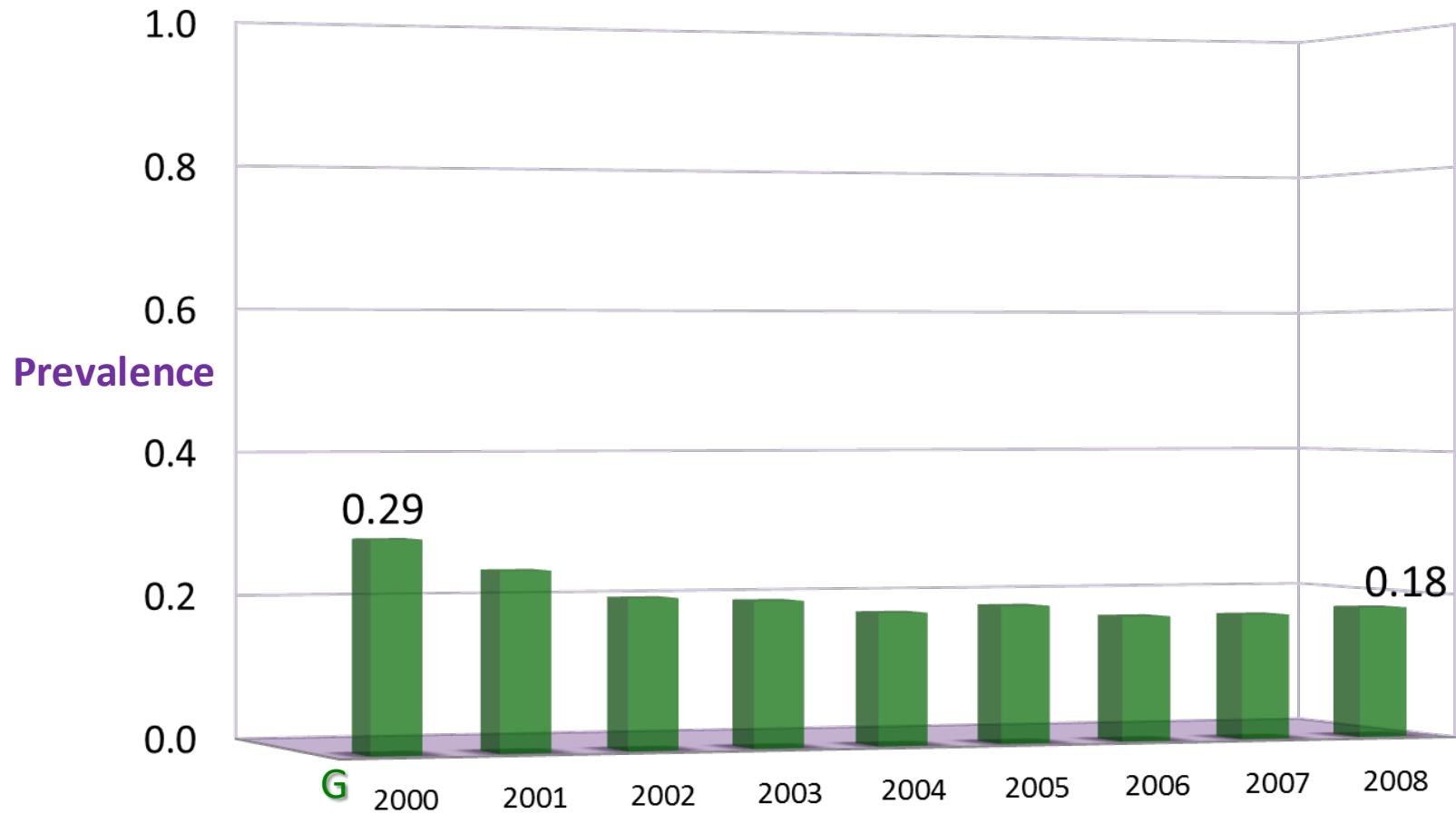


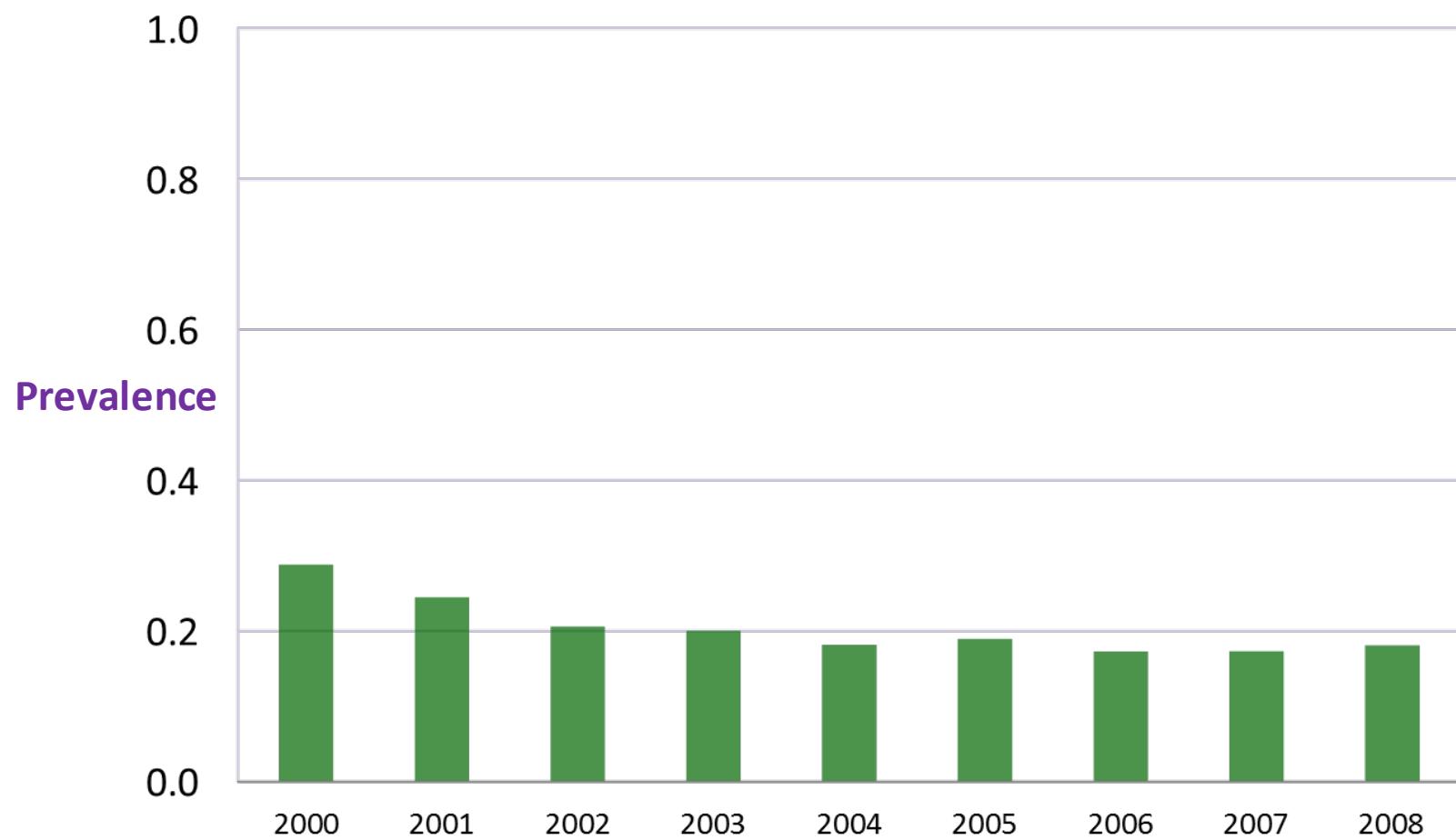
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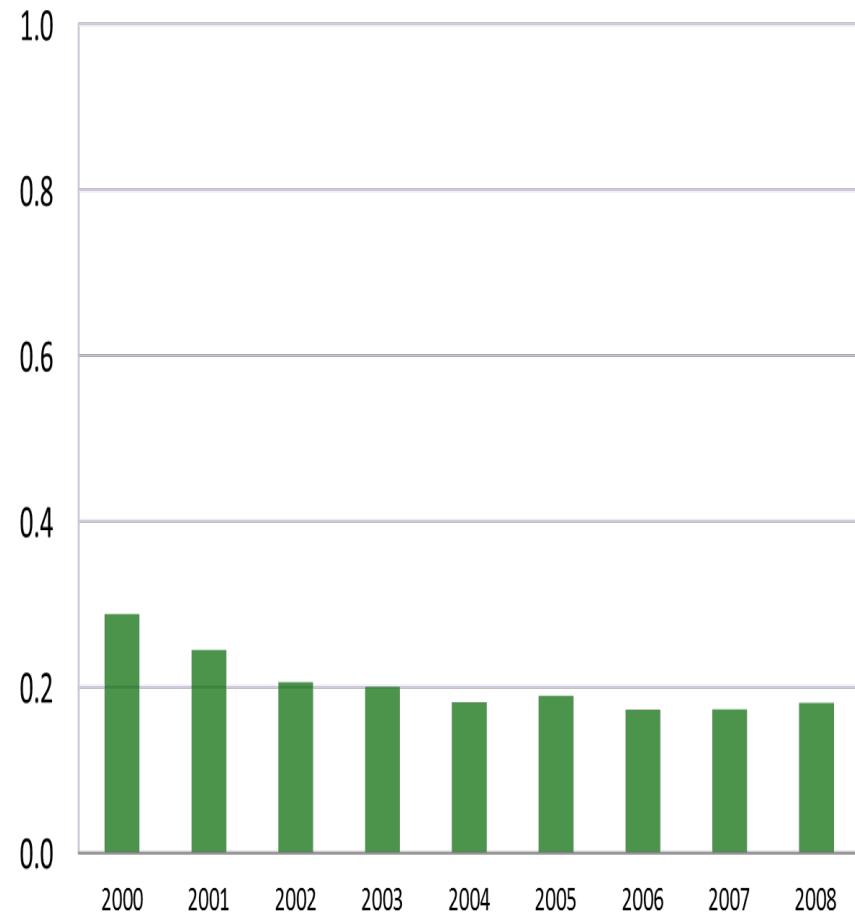
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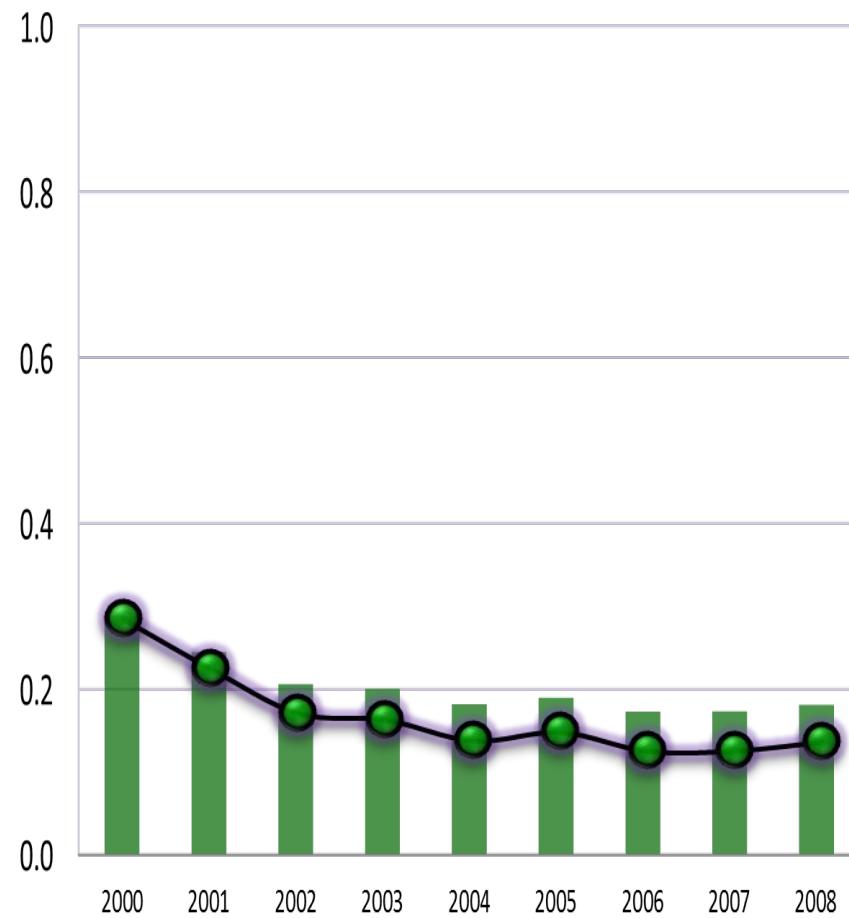


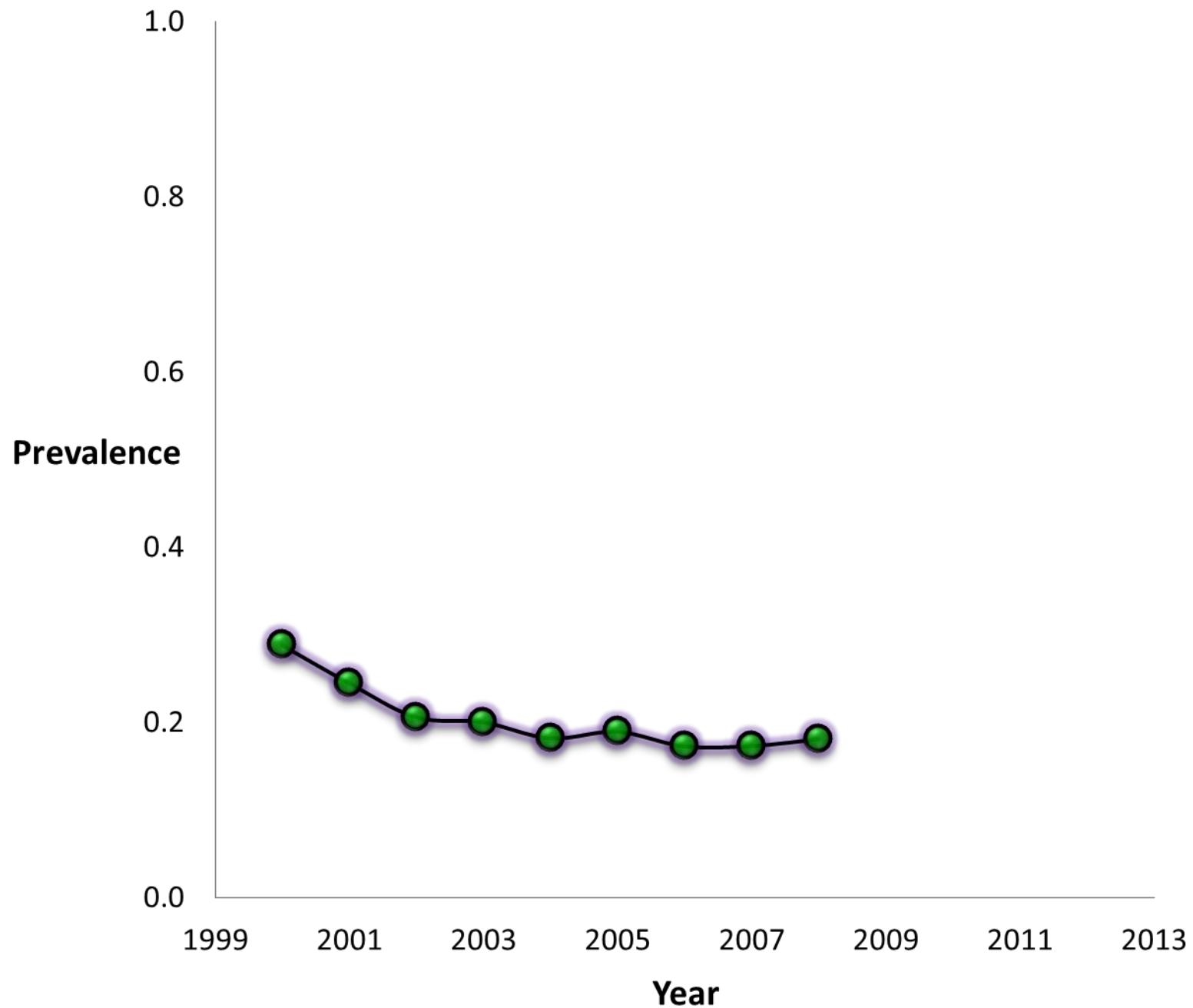


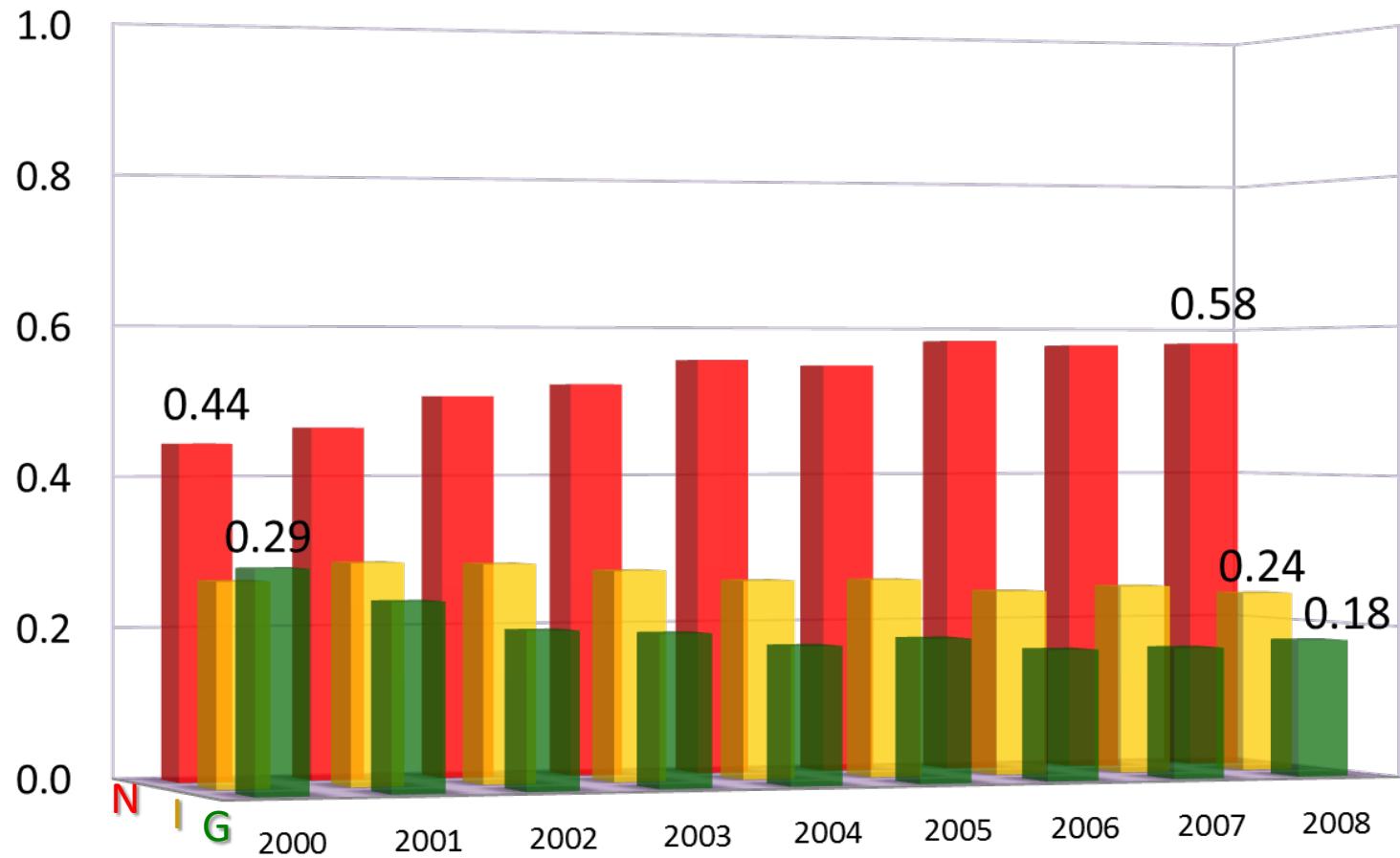
Prevalence

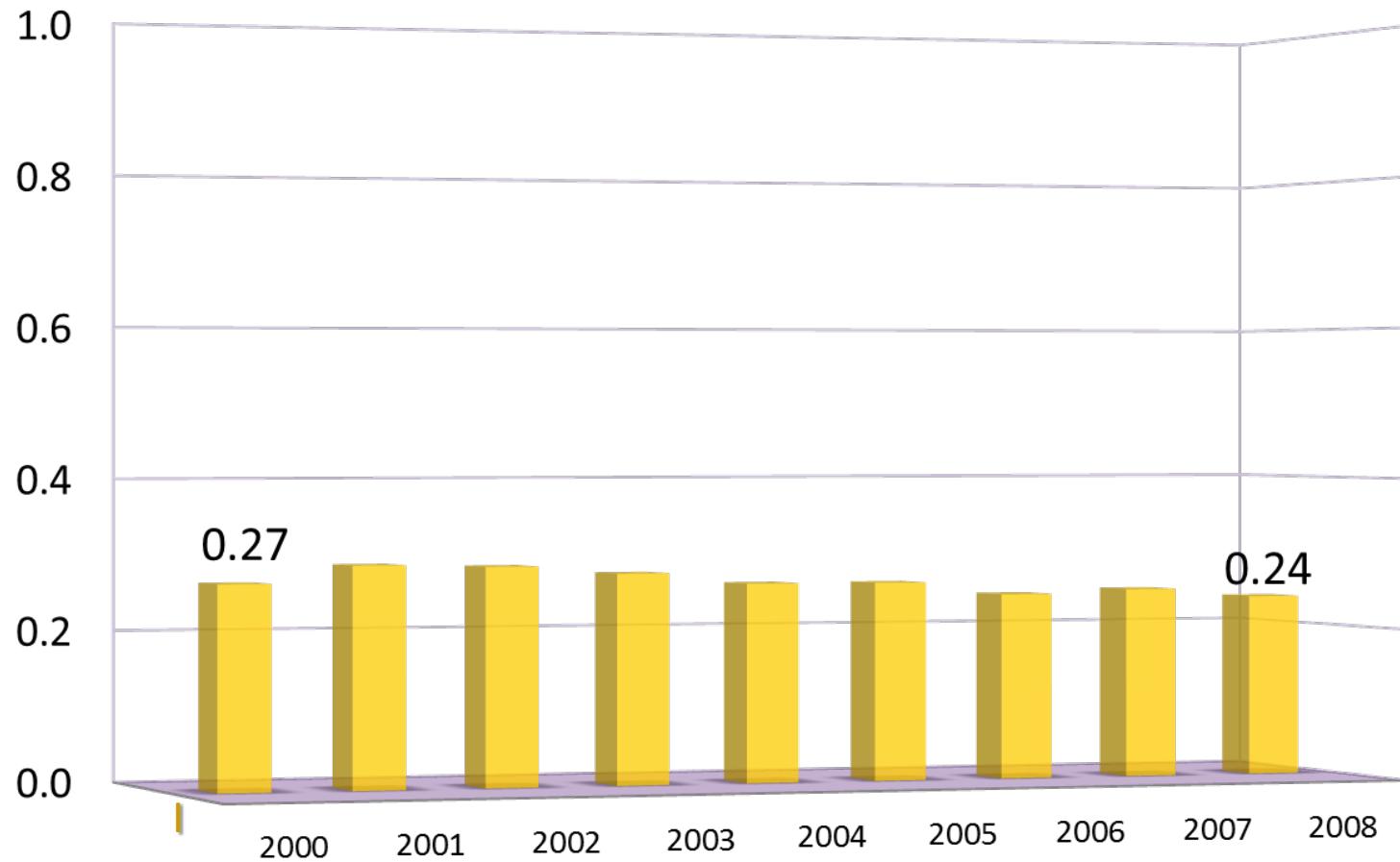


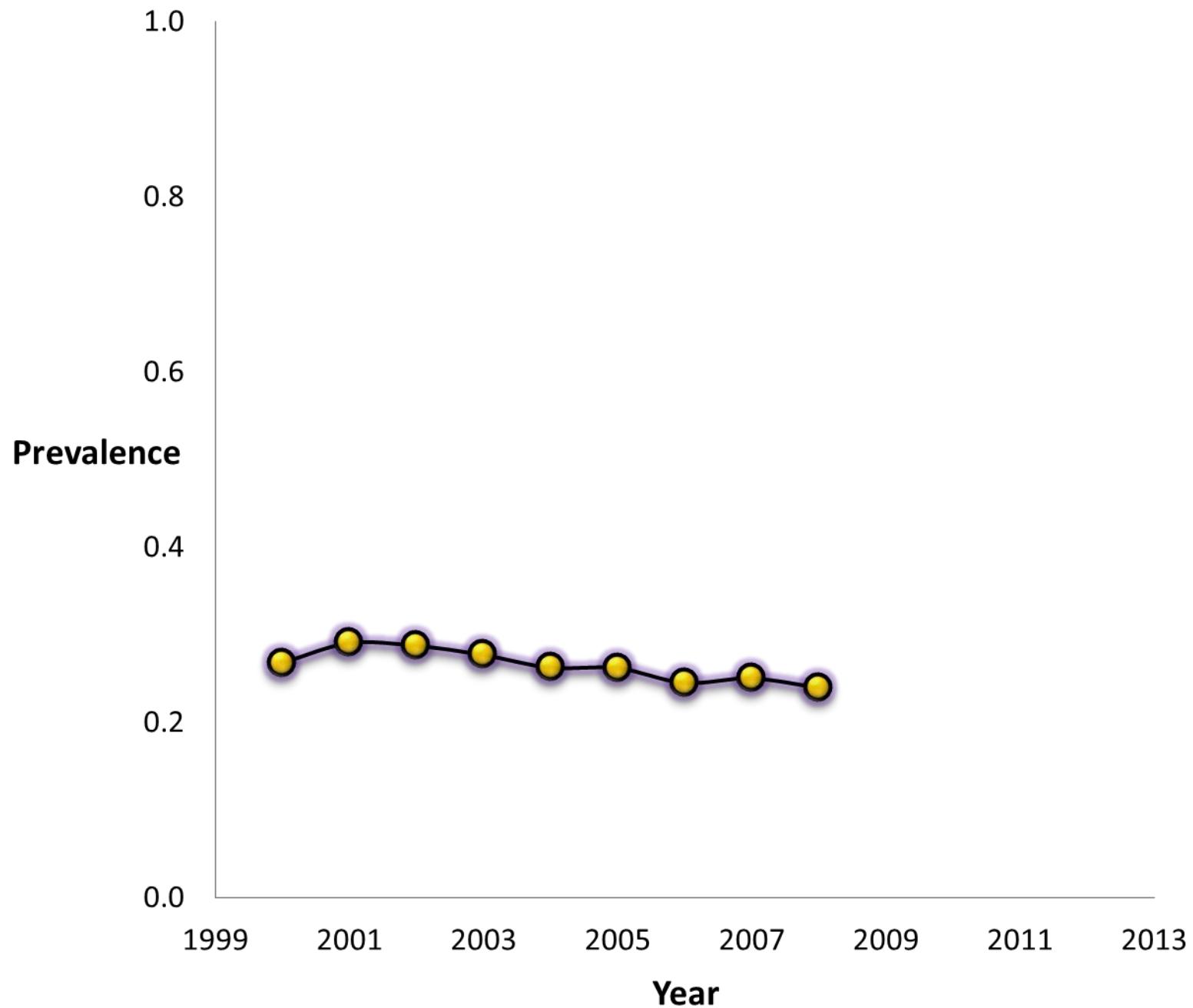
Prevalence

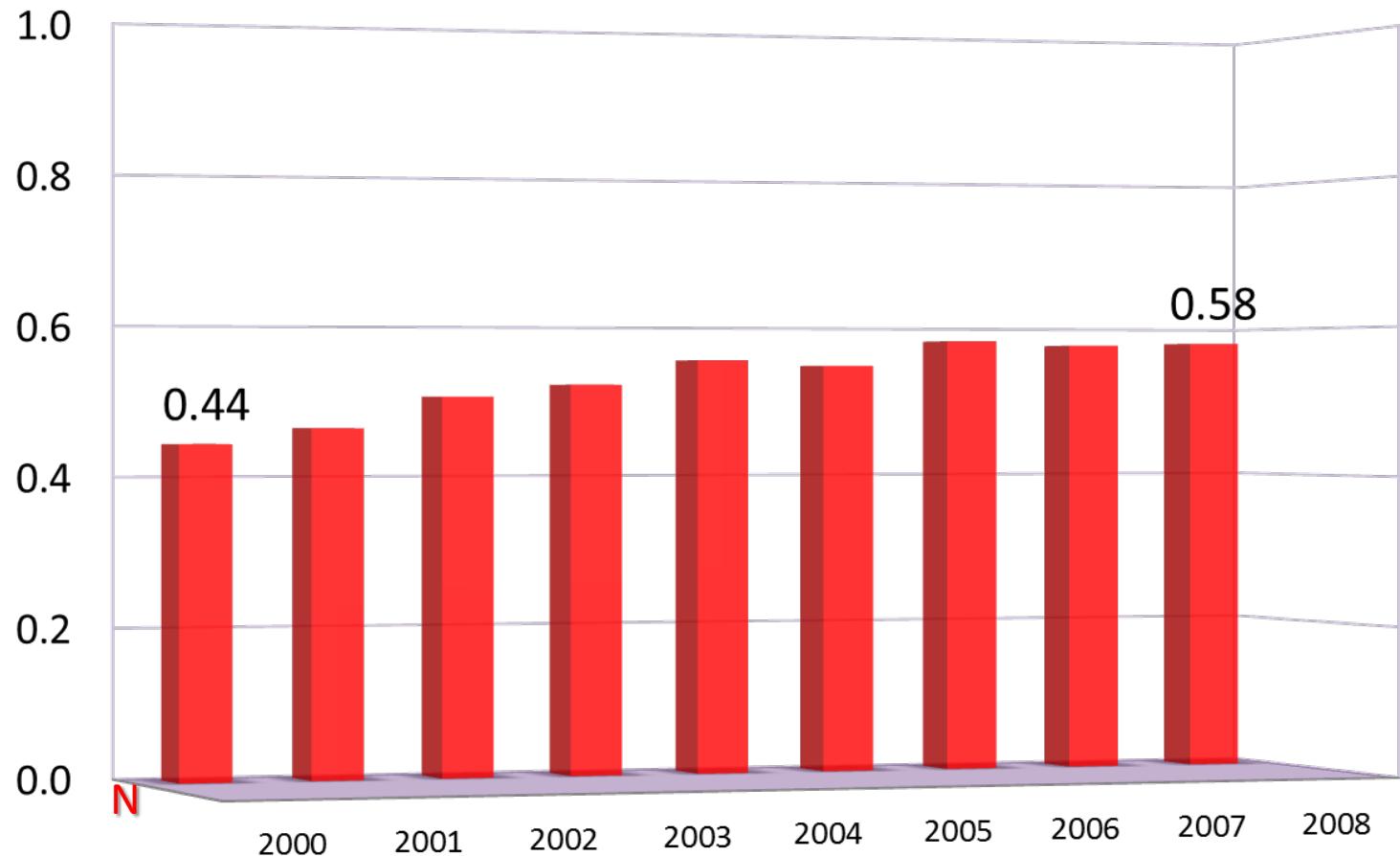


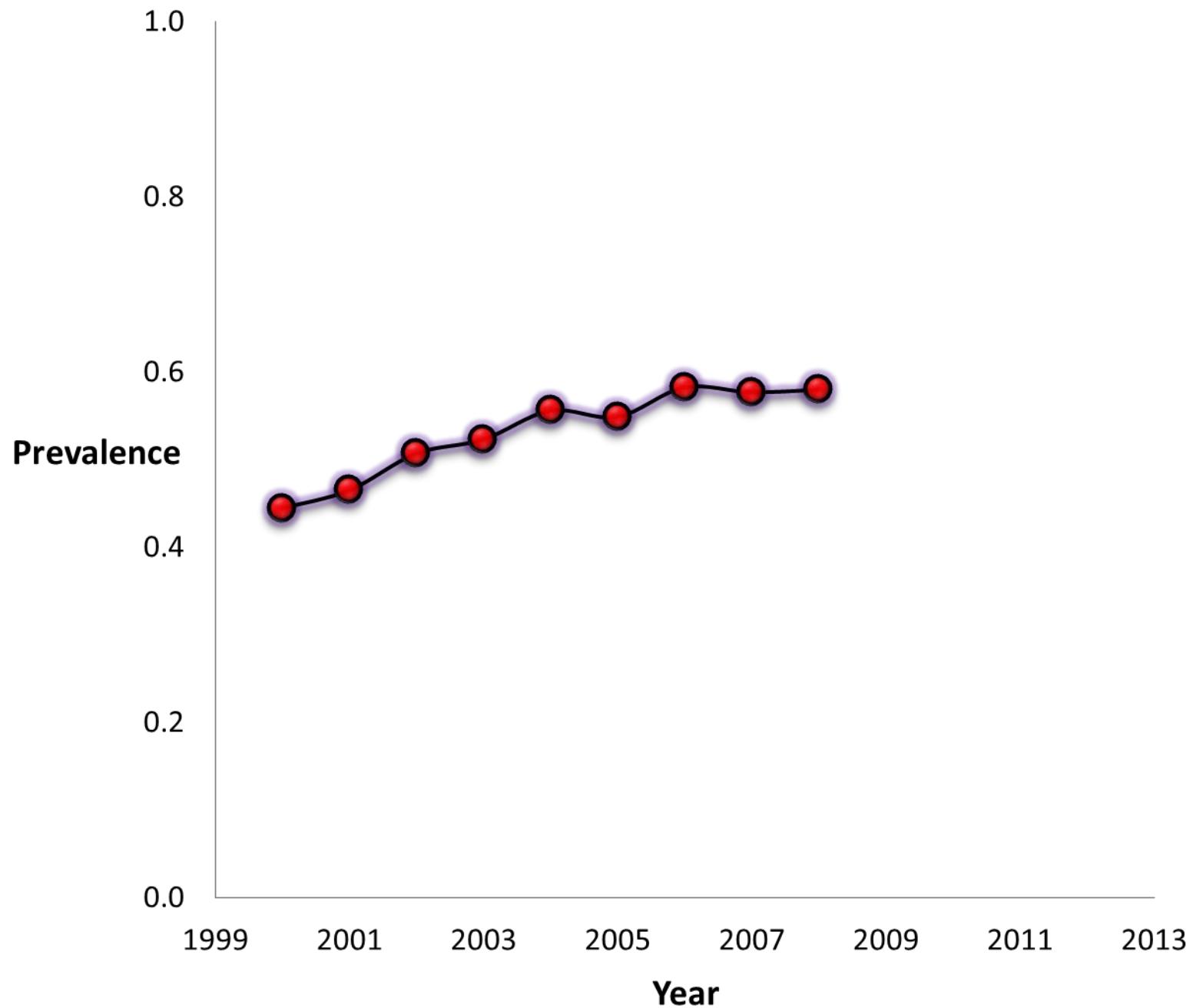


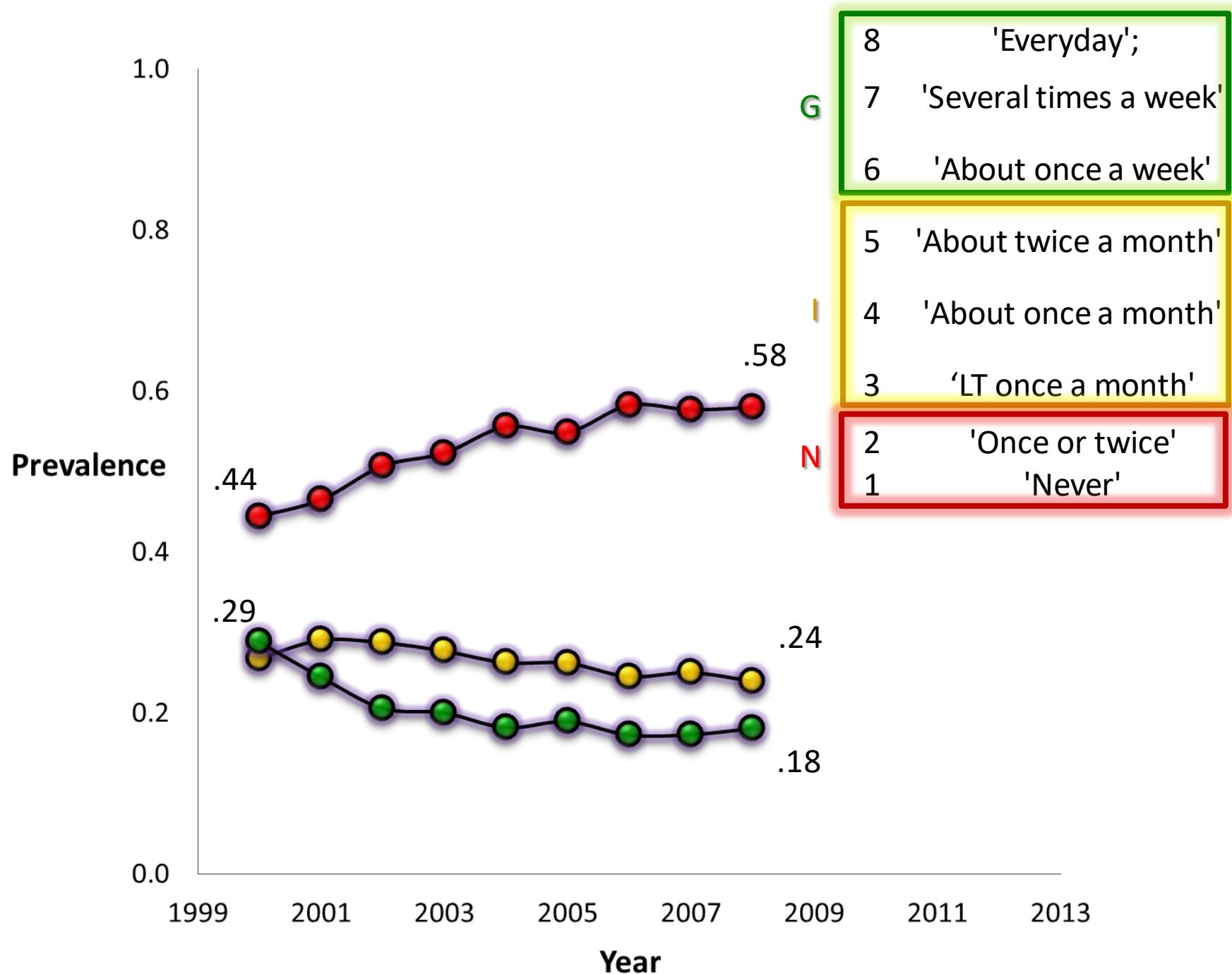


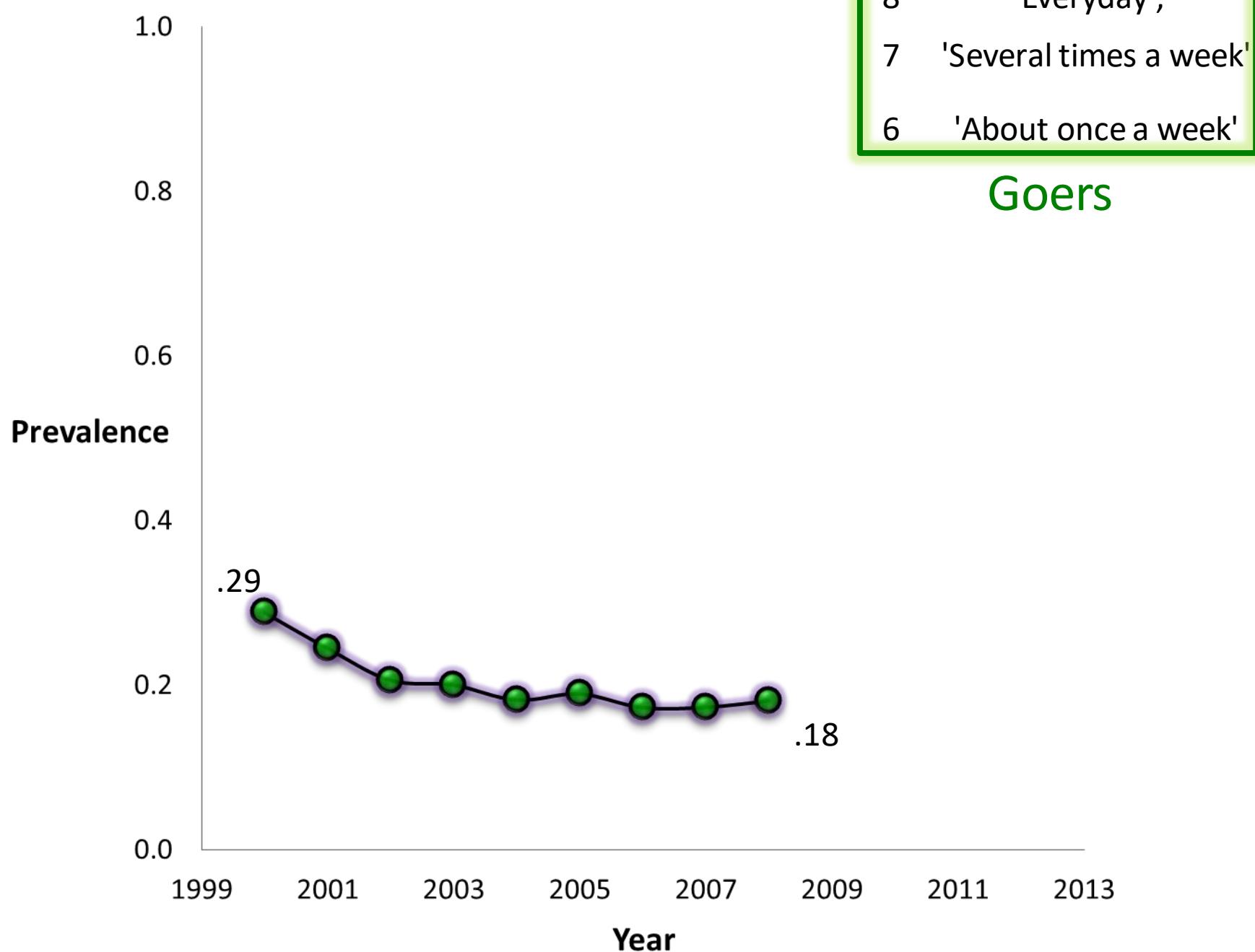


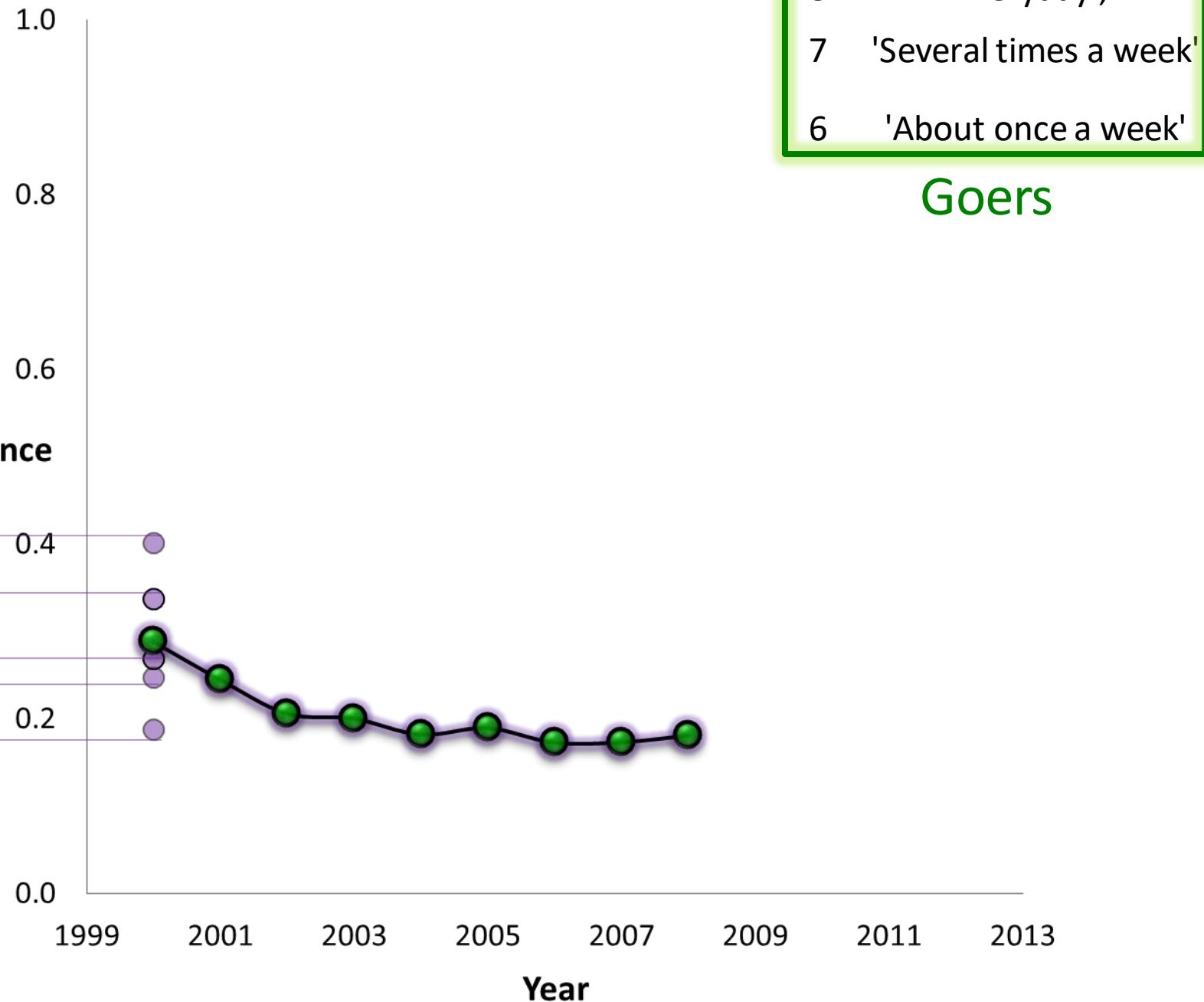






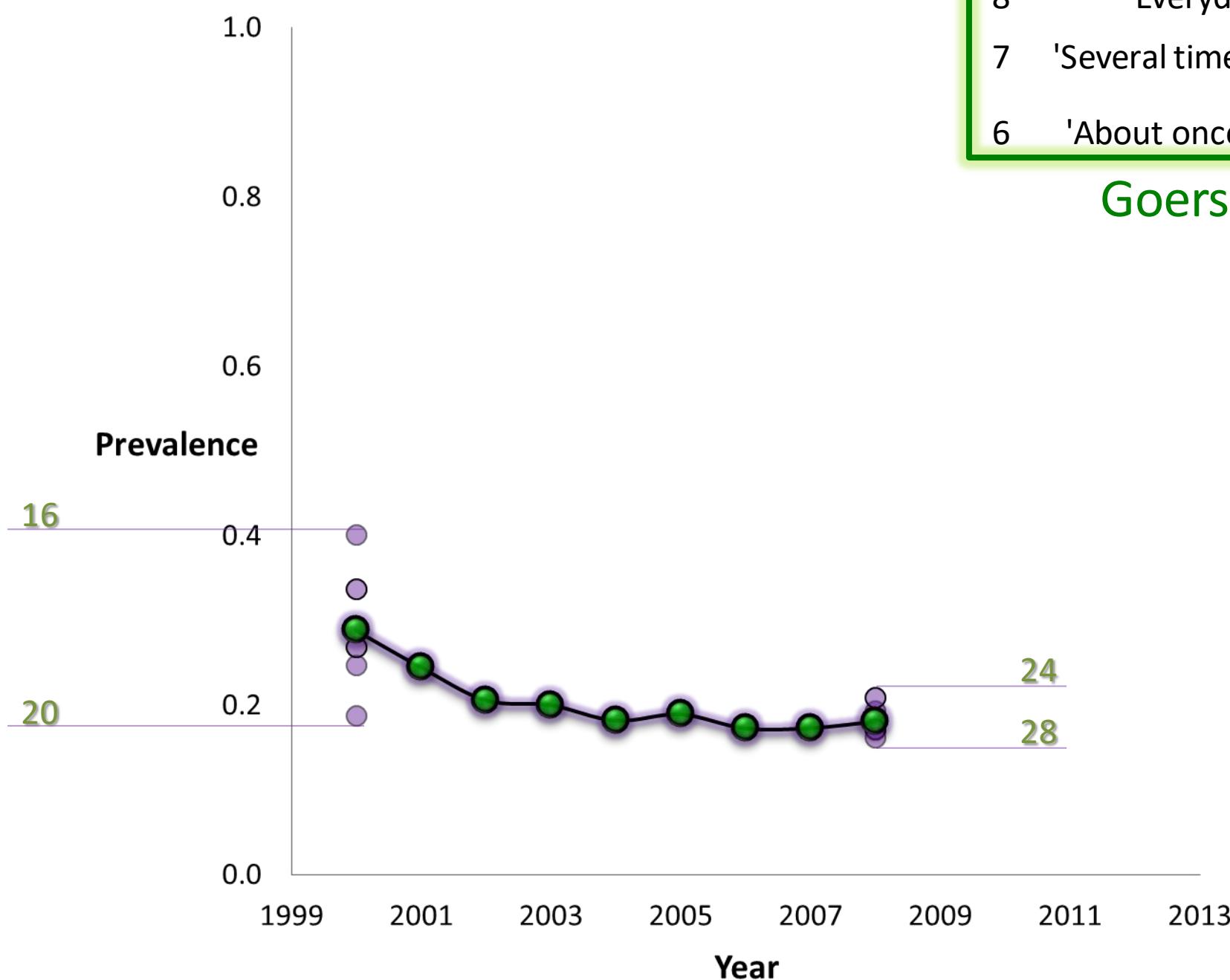






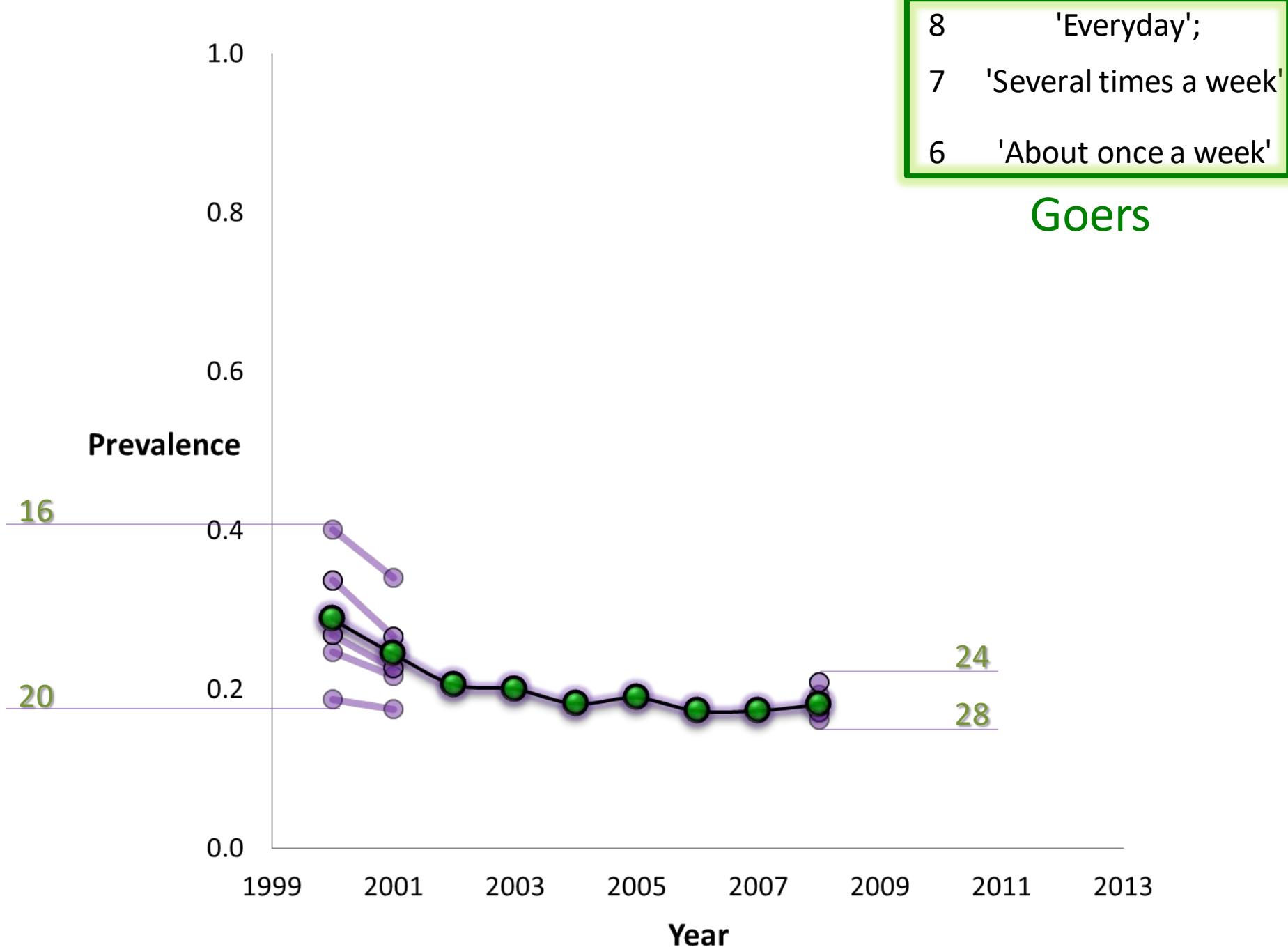
- 8 'Everyday';
- 7 'Several times a week'
- 6 'About once a week'

Goers



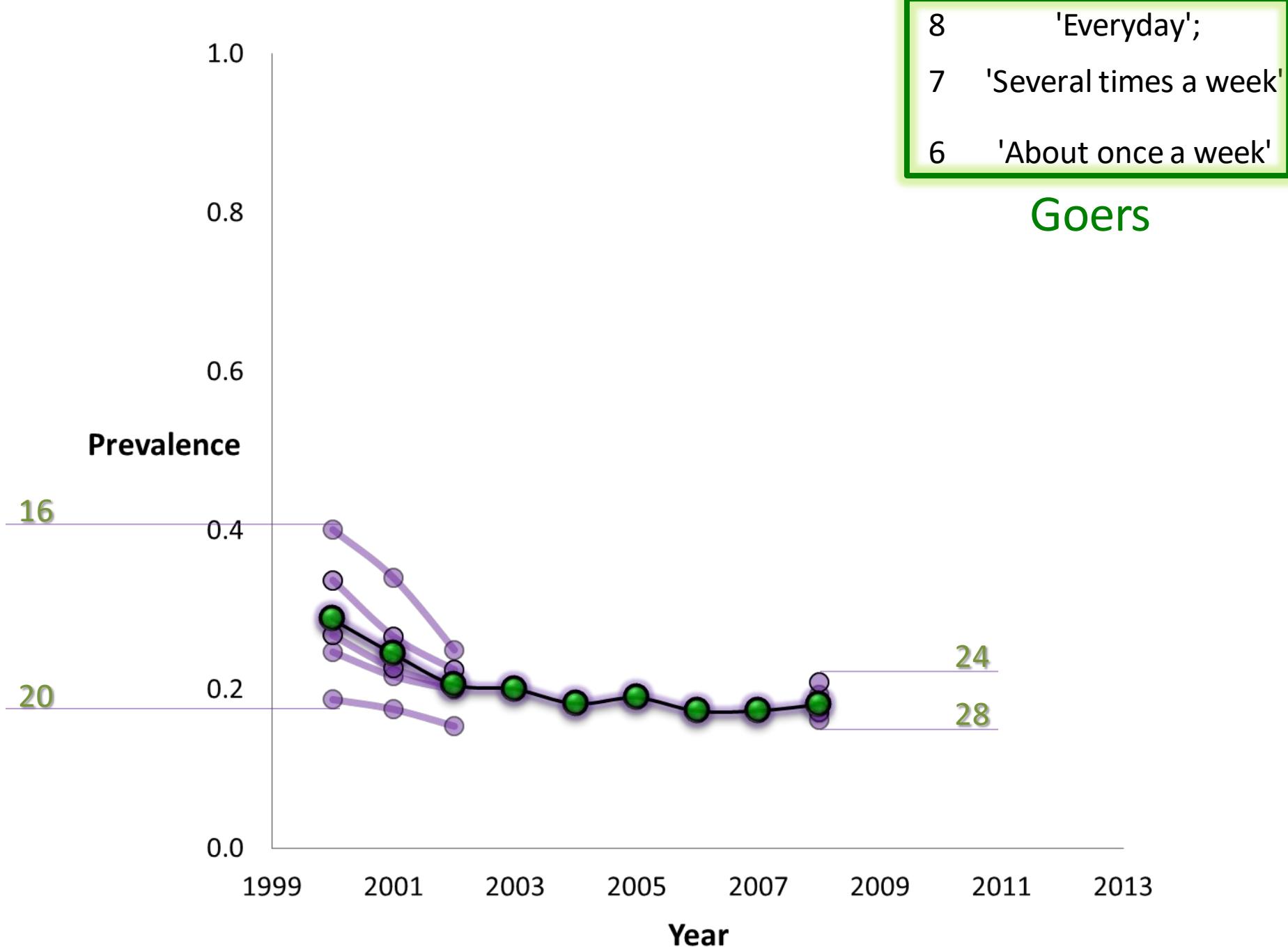
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Goers



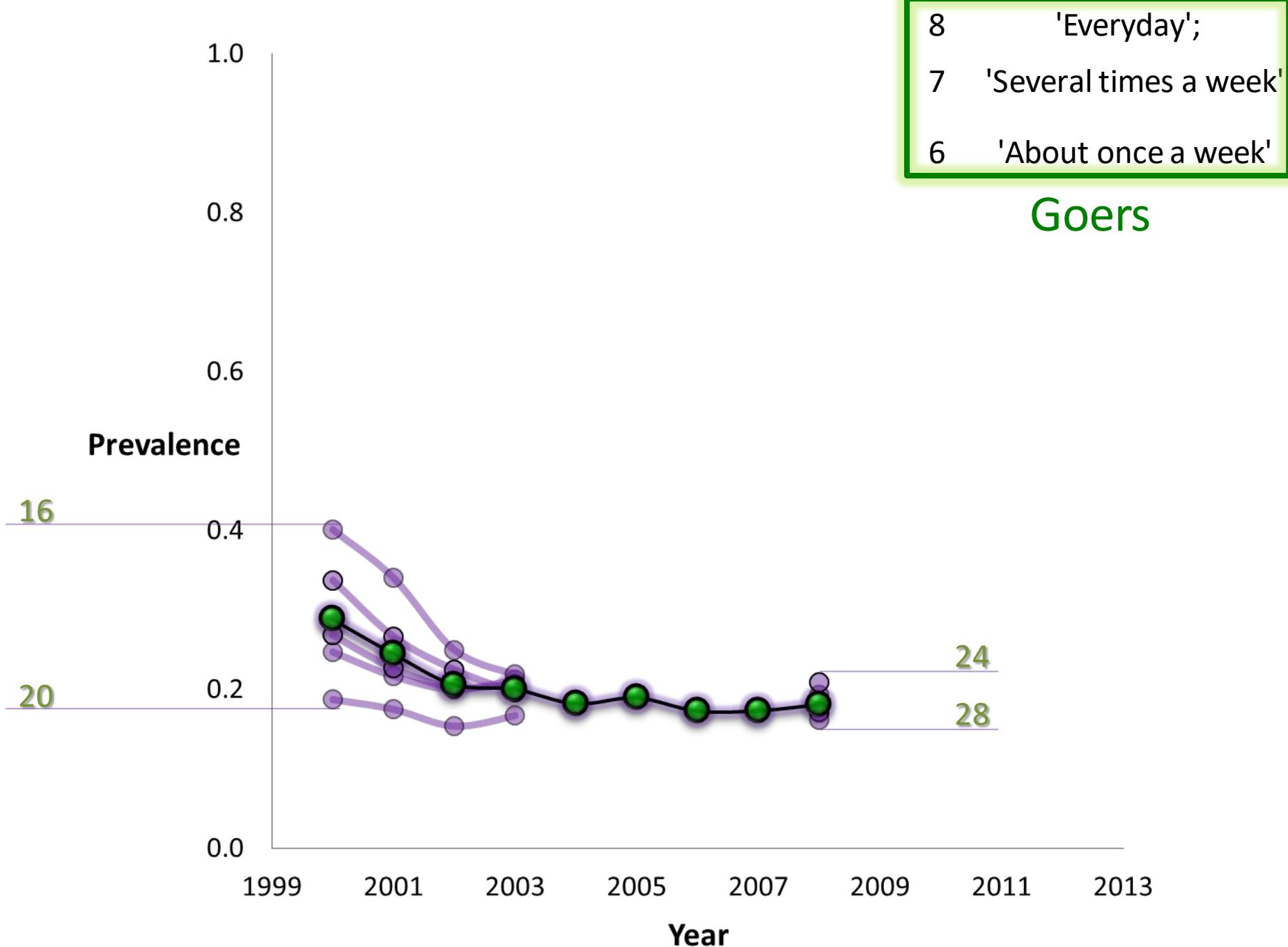
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Goers



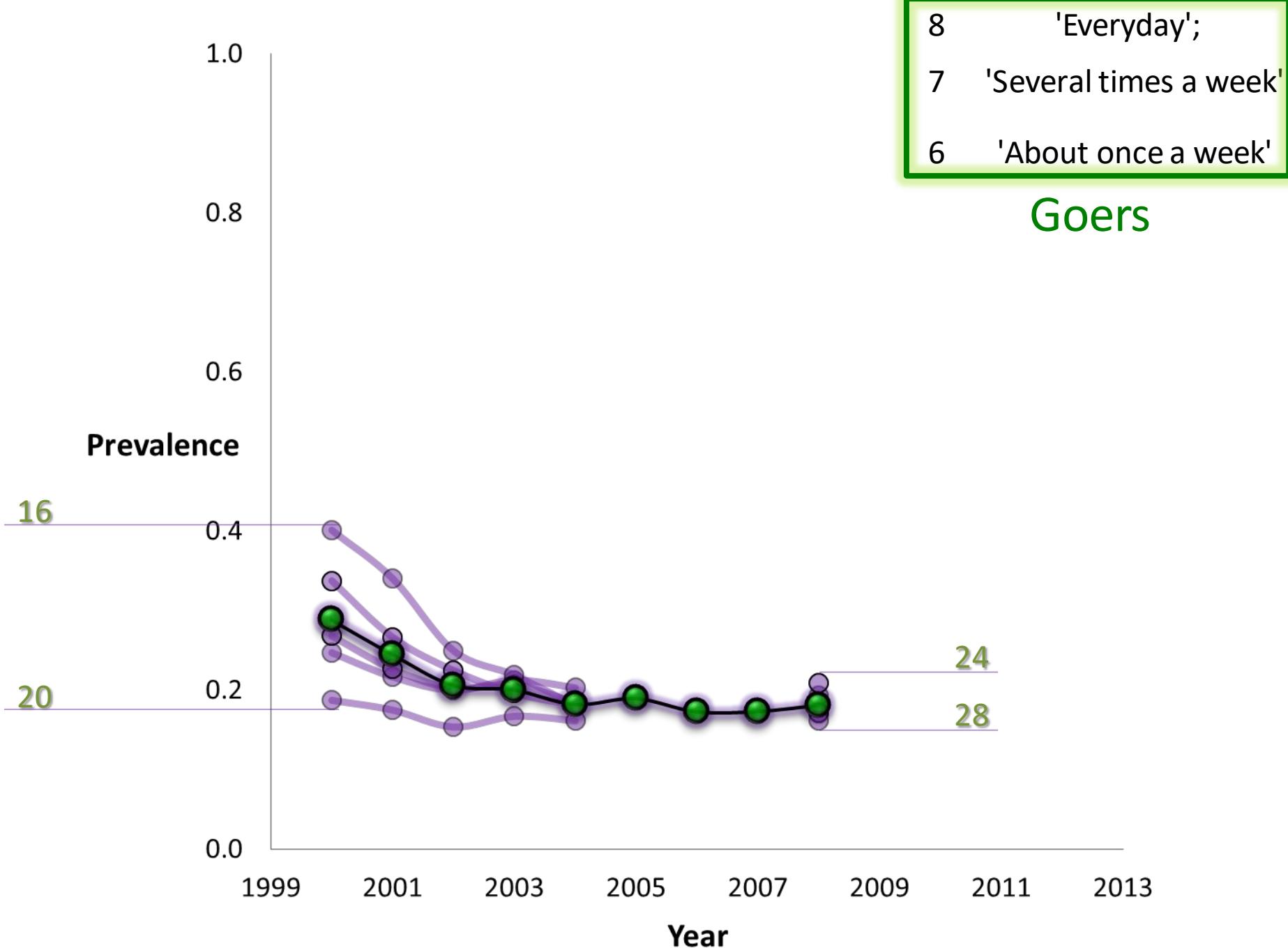
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Goers



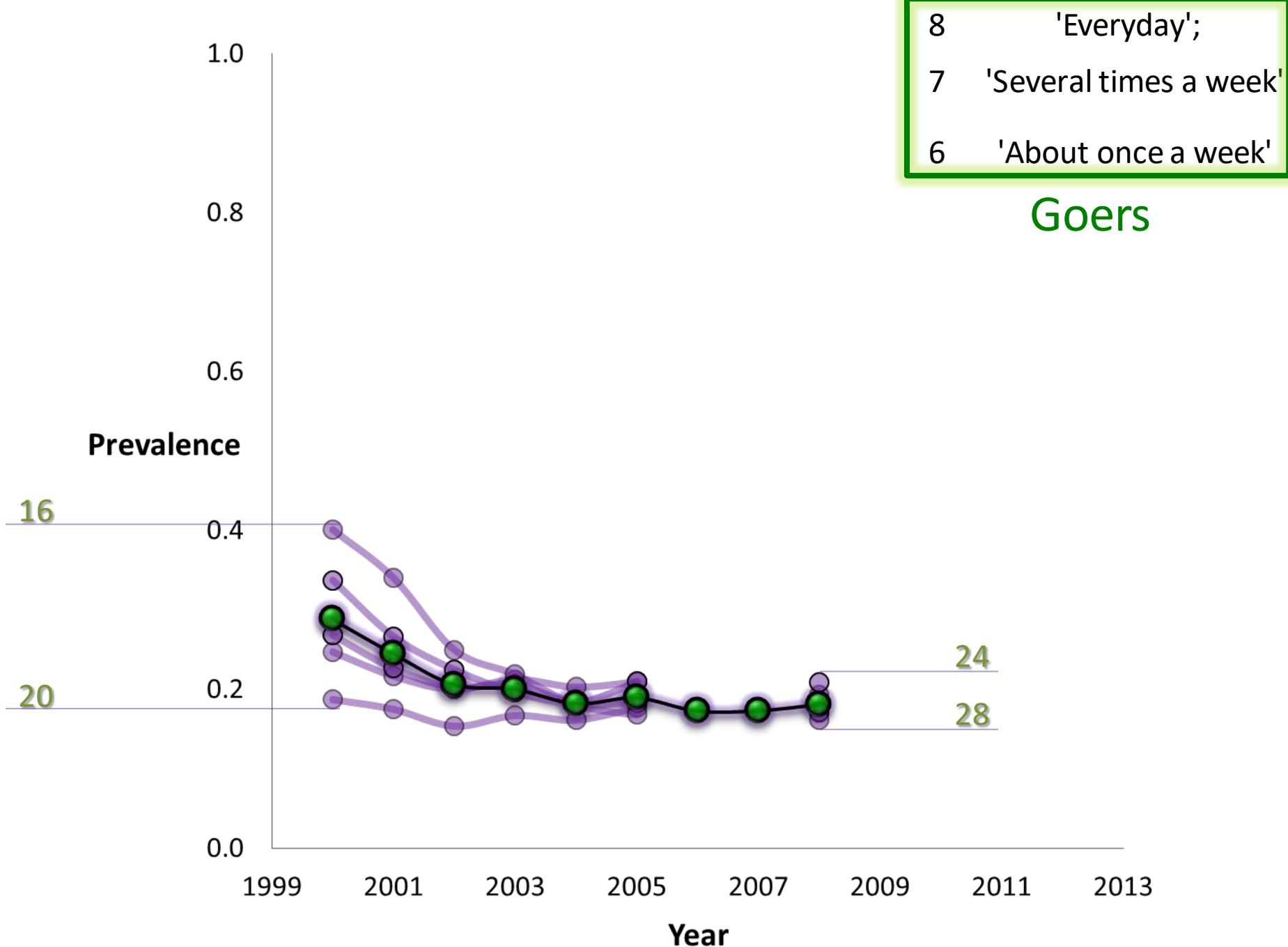
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Goers



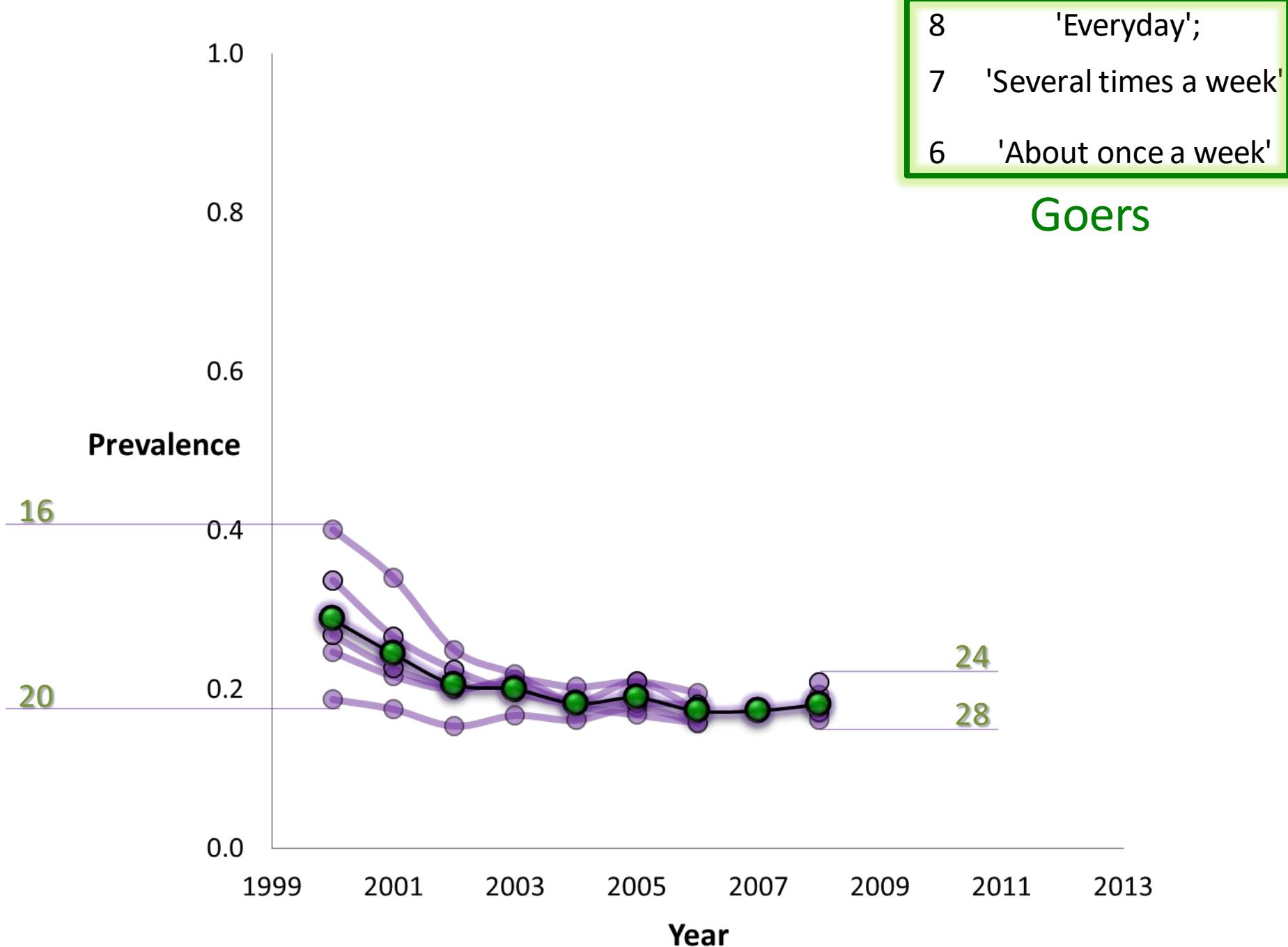
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Goers



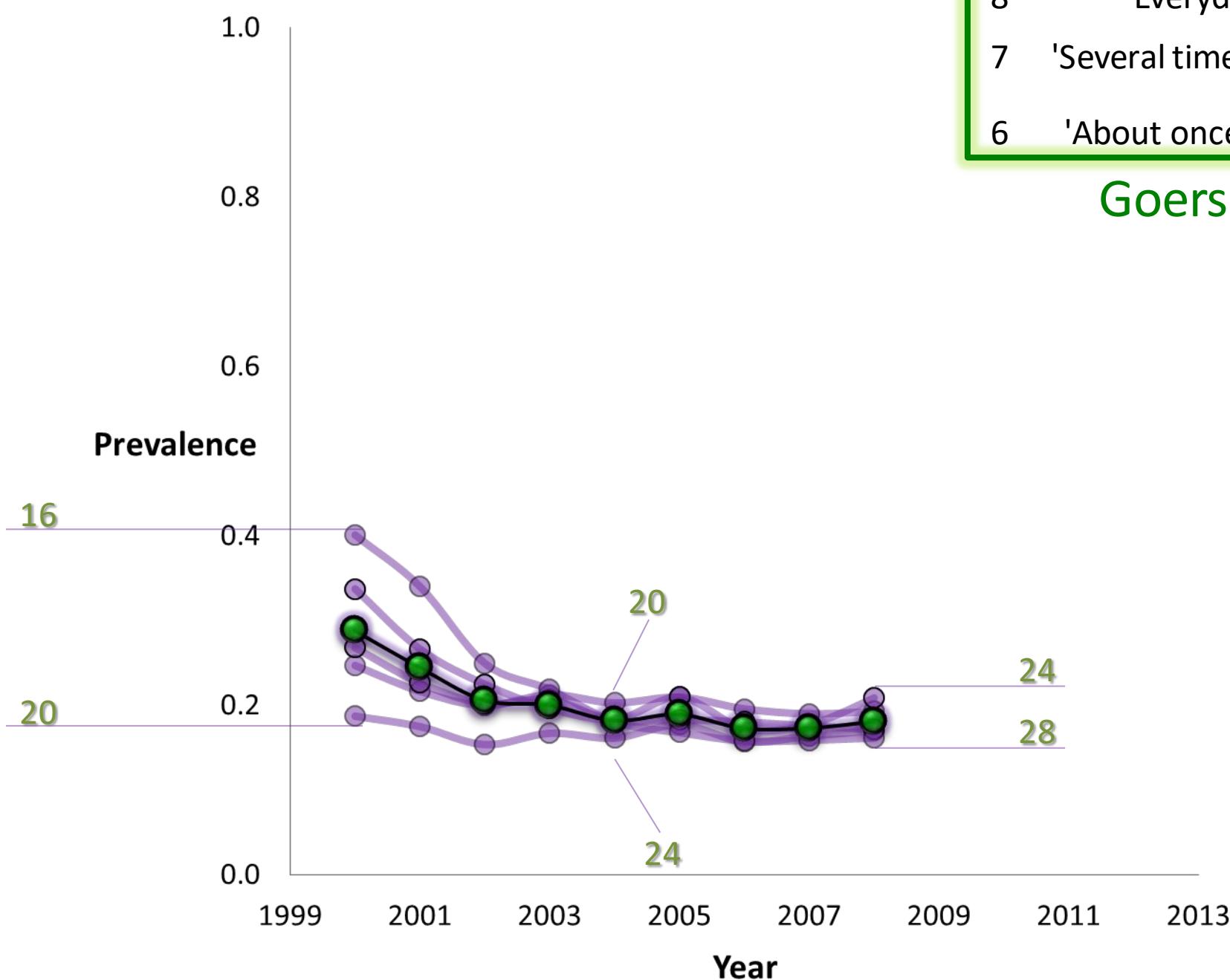
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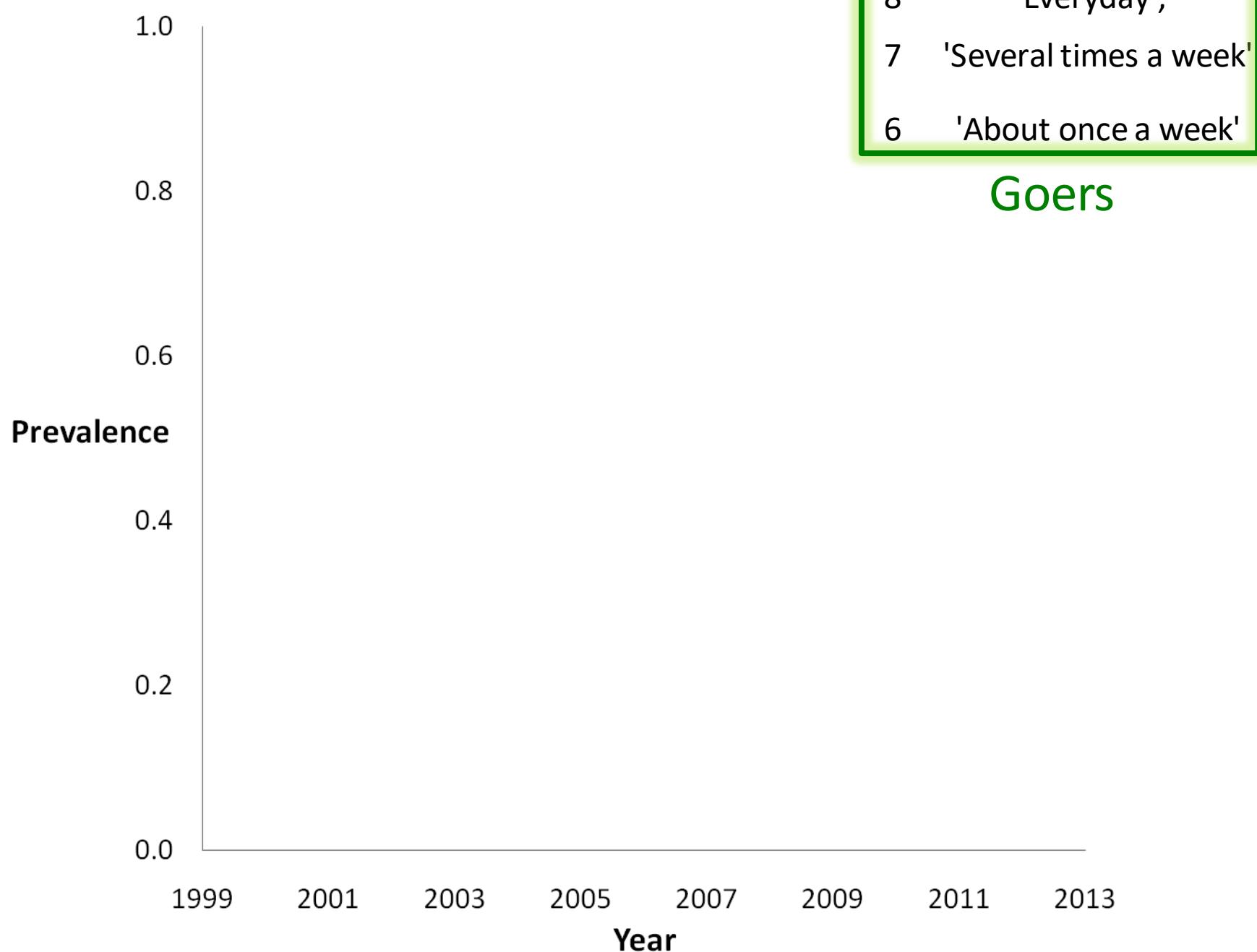
Goers

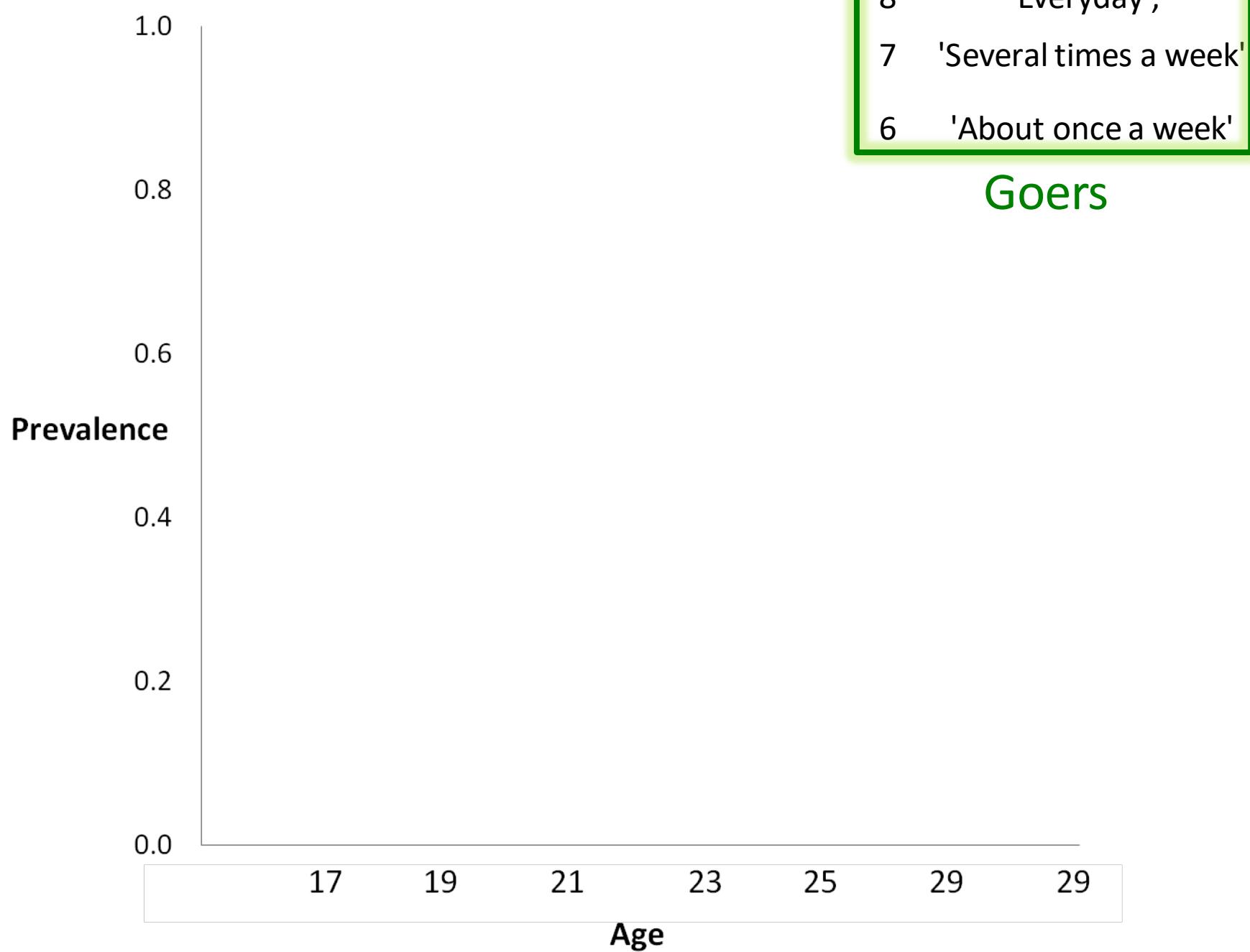


- 8 'Everyday';
- 7 'Several times a week'
- 6 'About once a week'

Goers







Prevalence

1.0
0.8
0.6
0.4
0.2
0.0

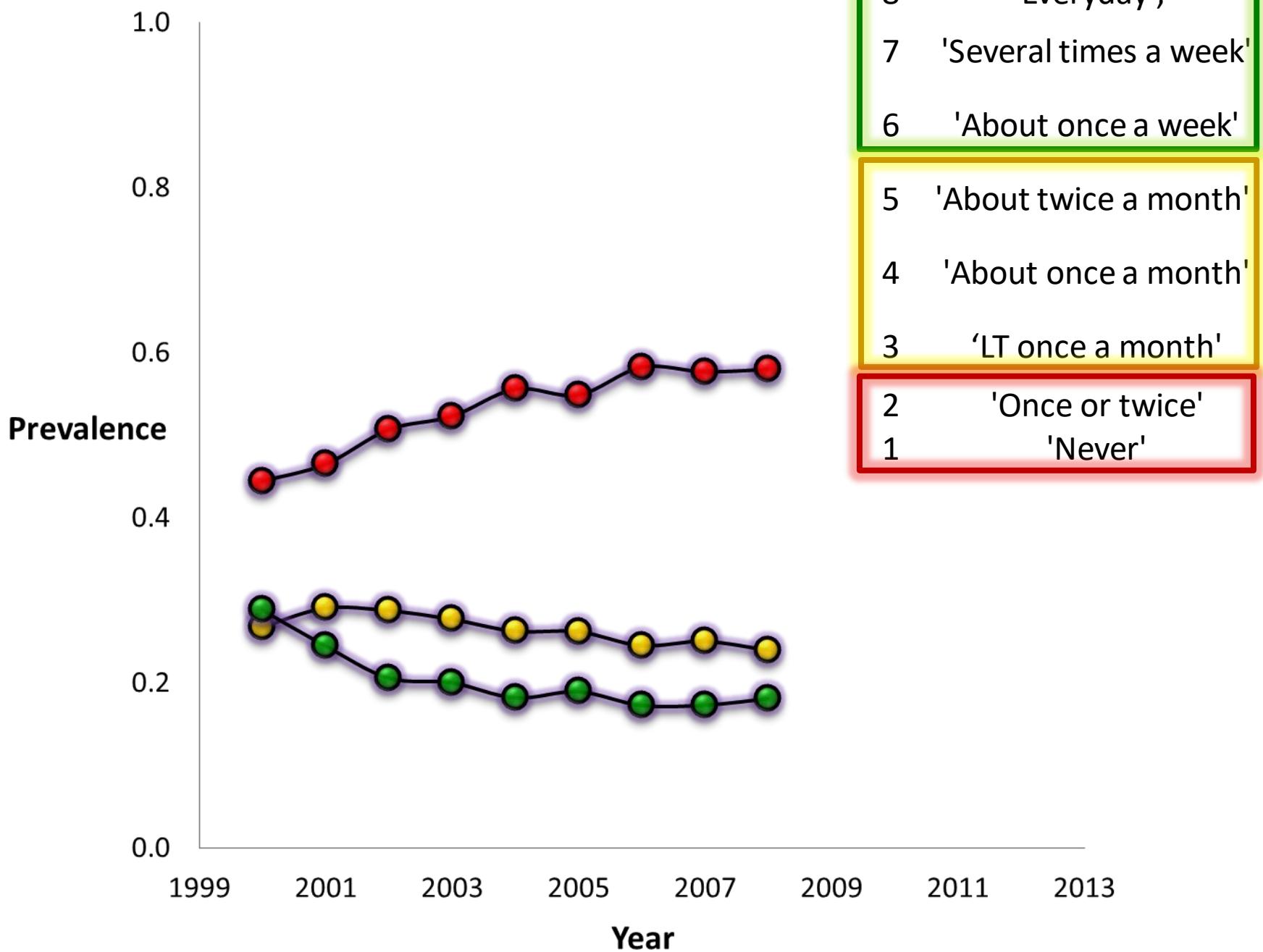
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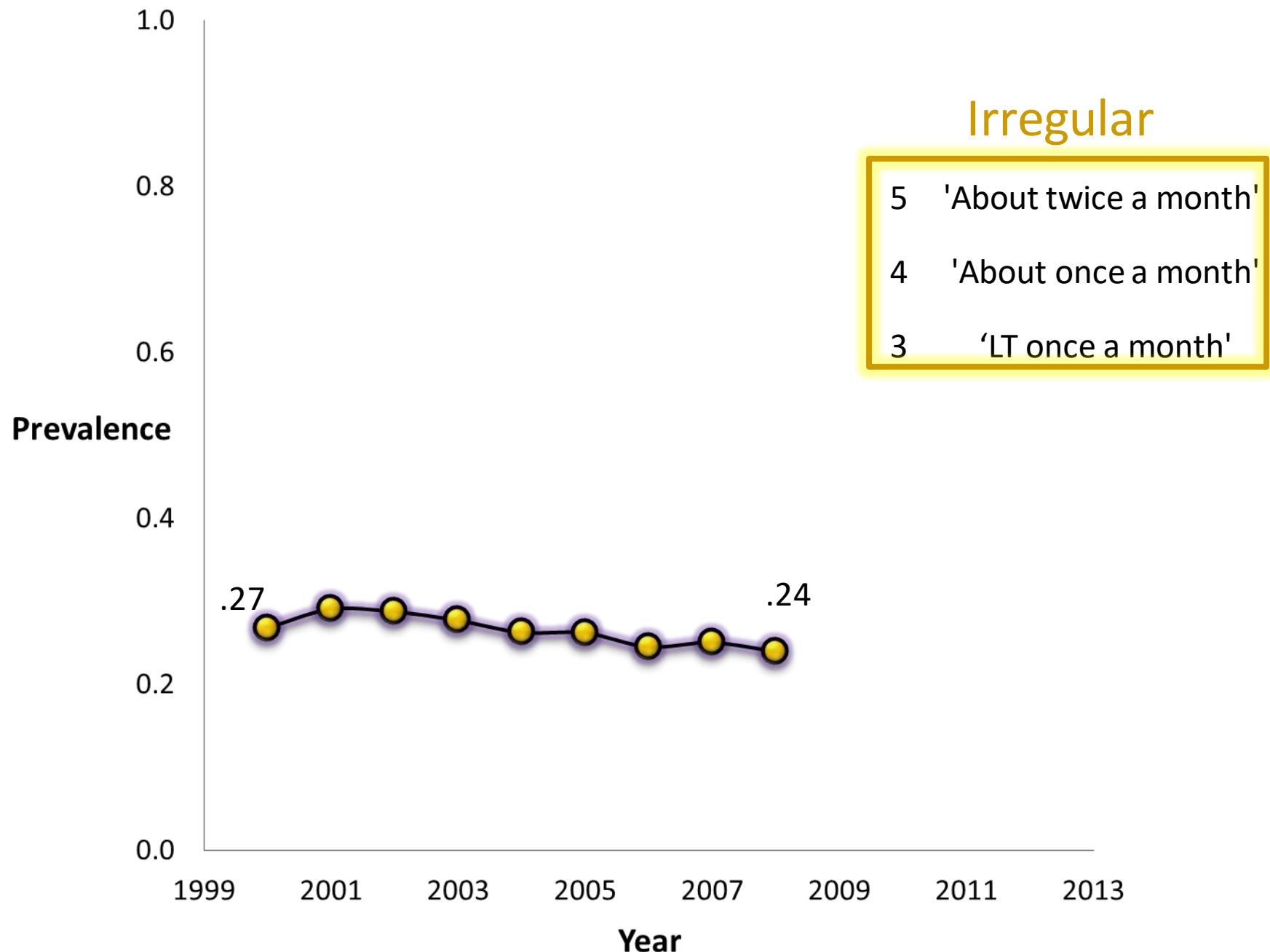
Age

- 8 'Everyday';
- 7 'Several times a week'
- 6 'About once a week'

Goers

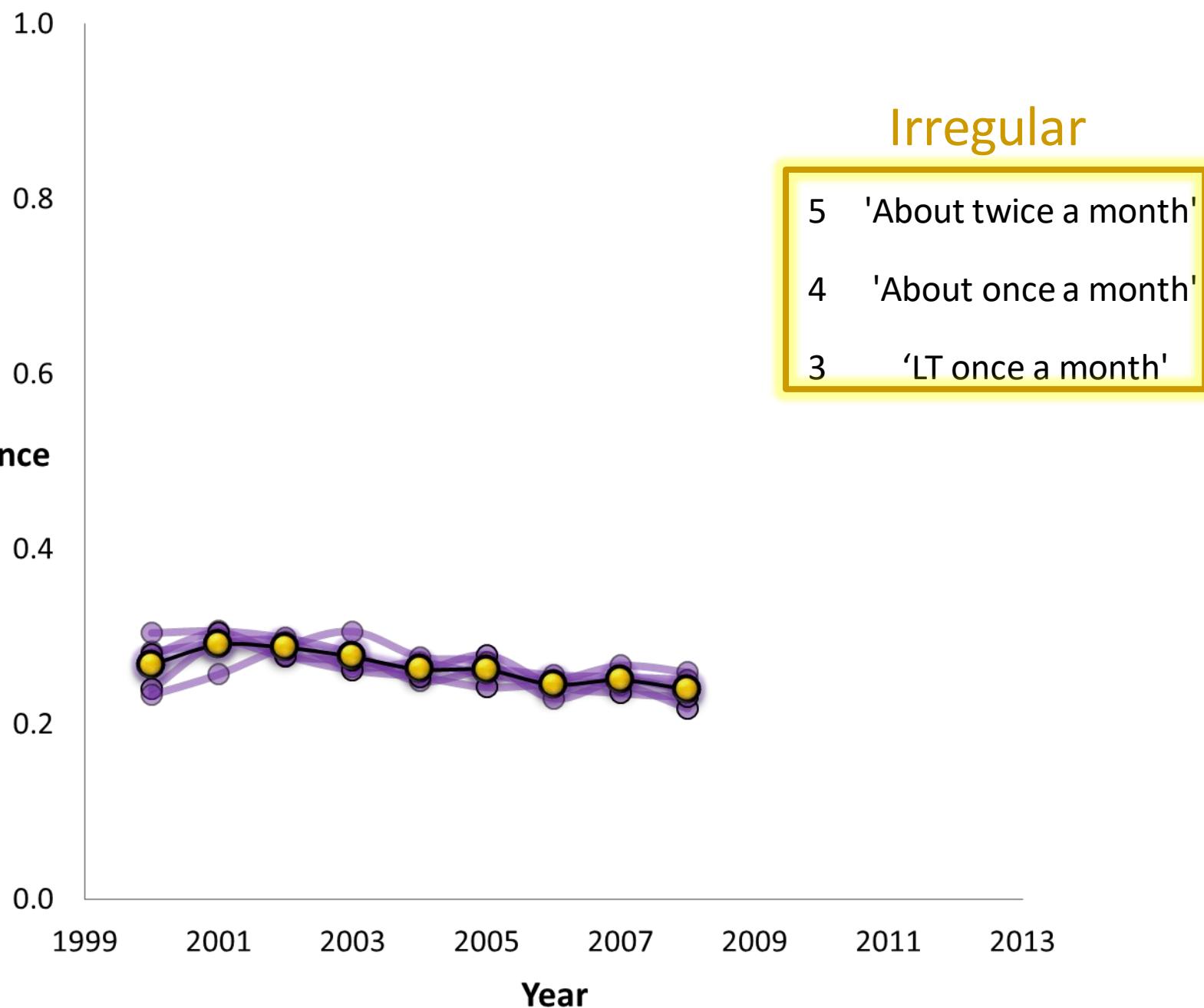
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Prevalence



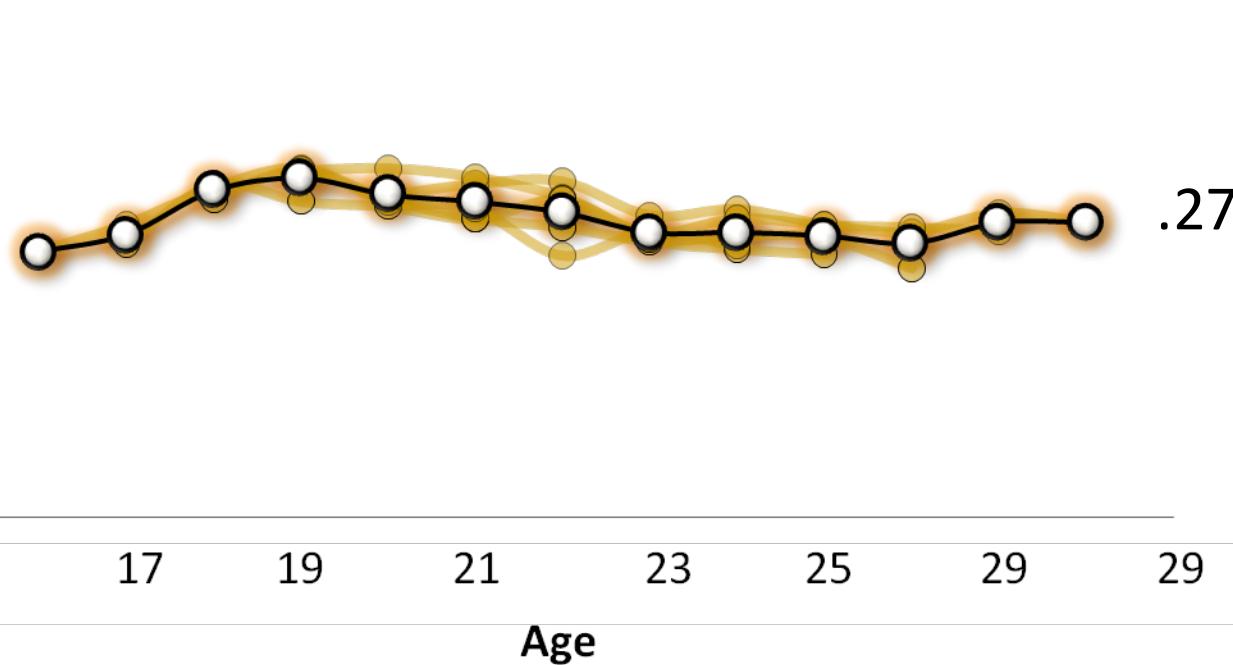


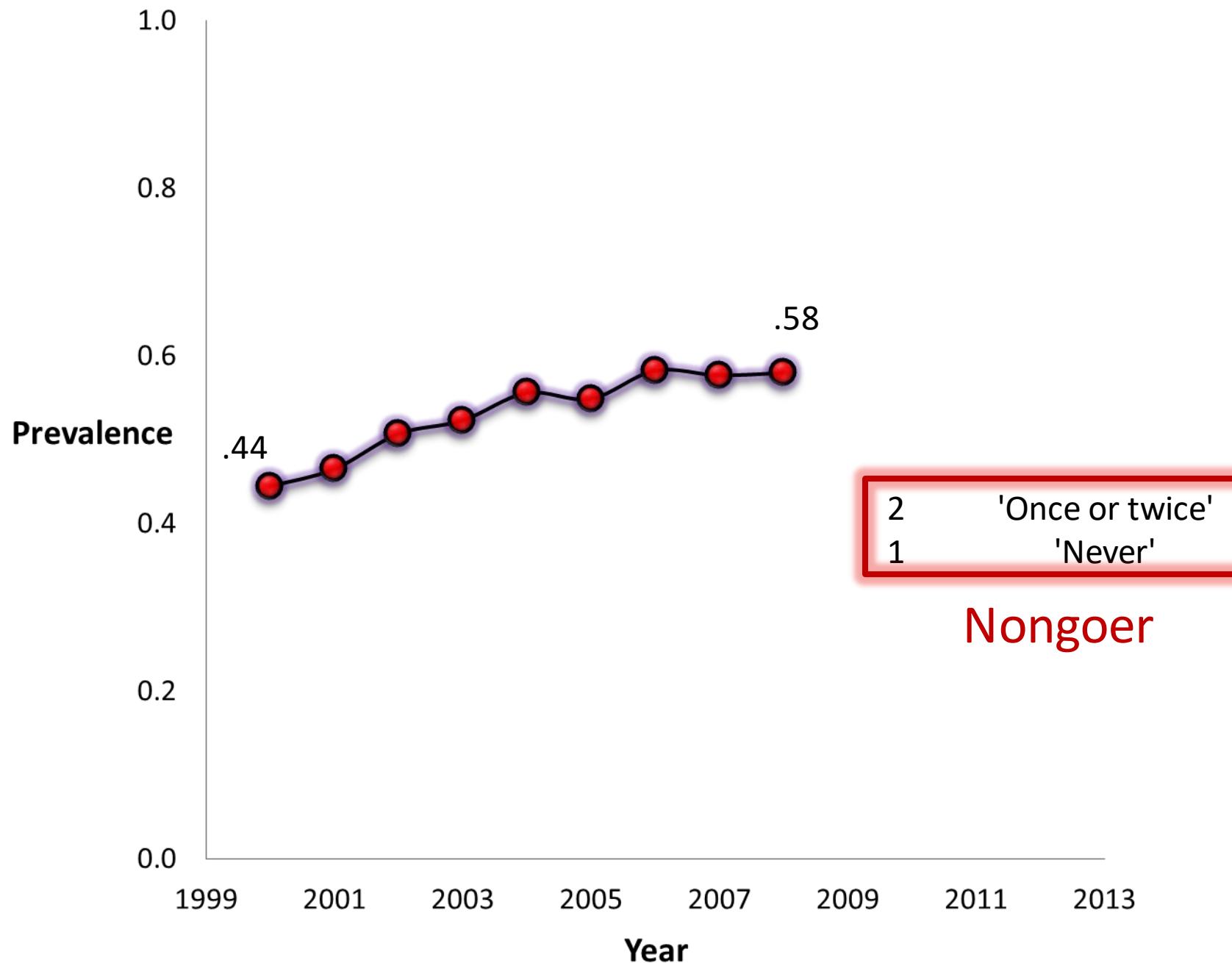
Prevalence

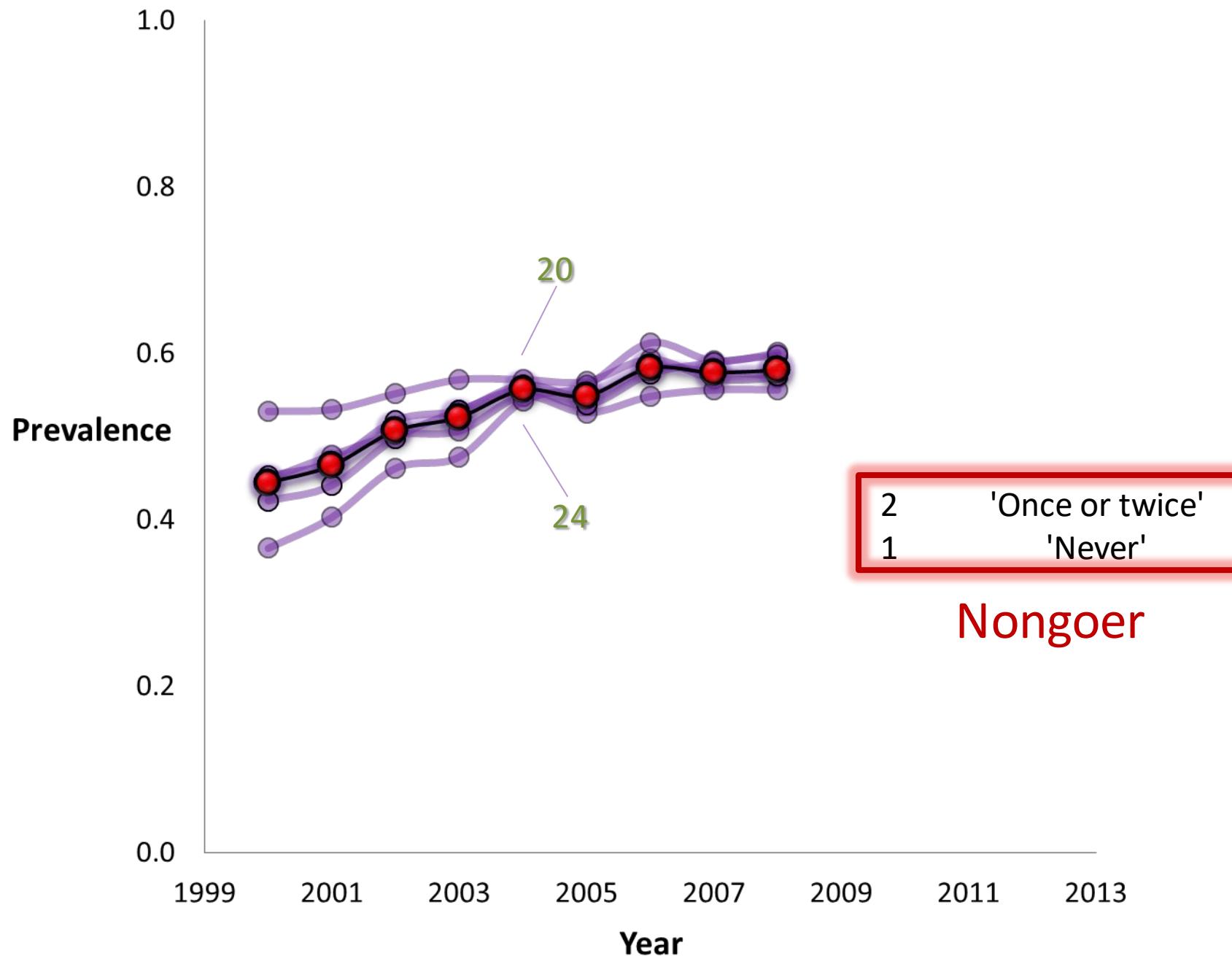
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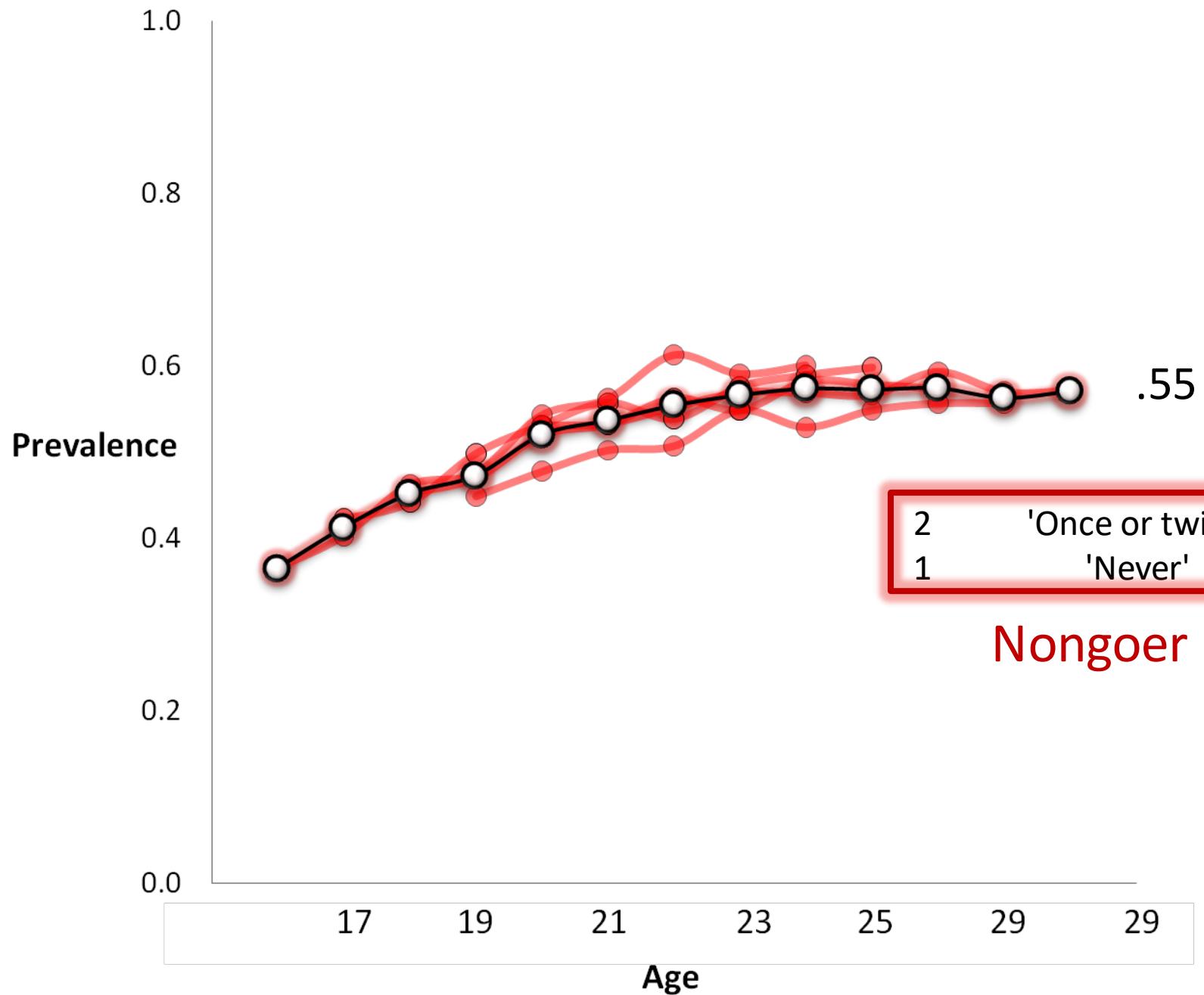
Irregular

- 5 'About twice a month'
- 4 'About once a month'
- 3 'LT once a month'



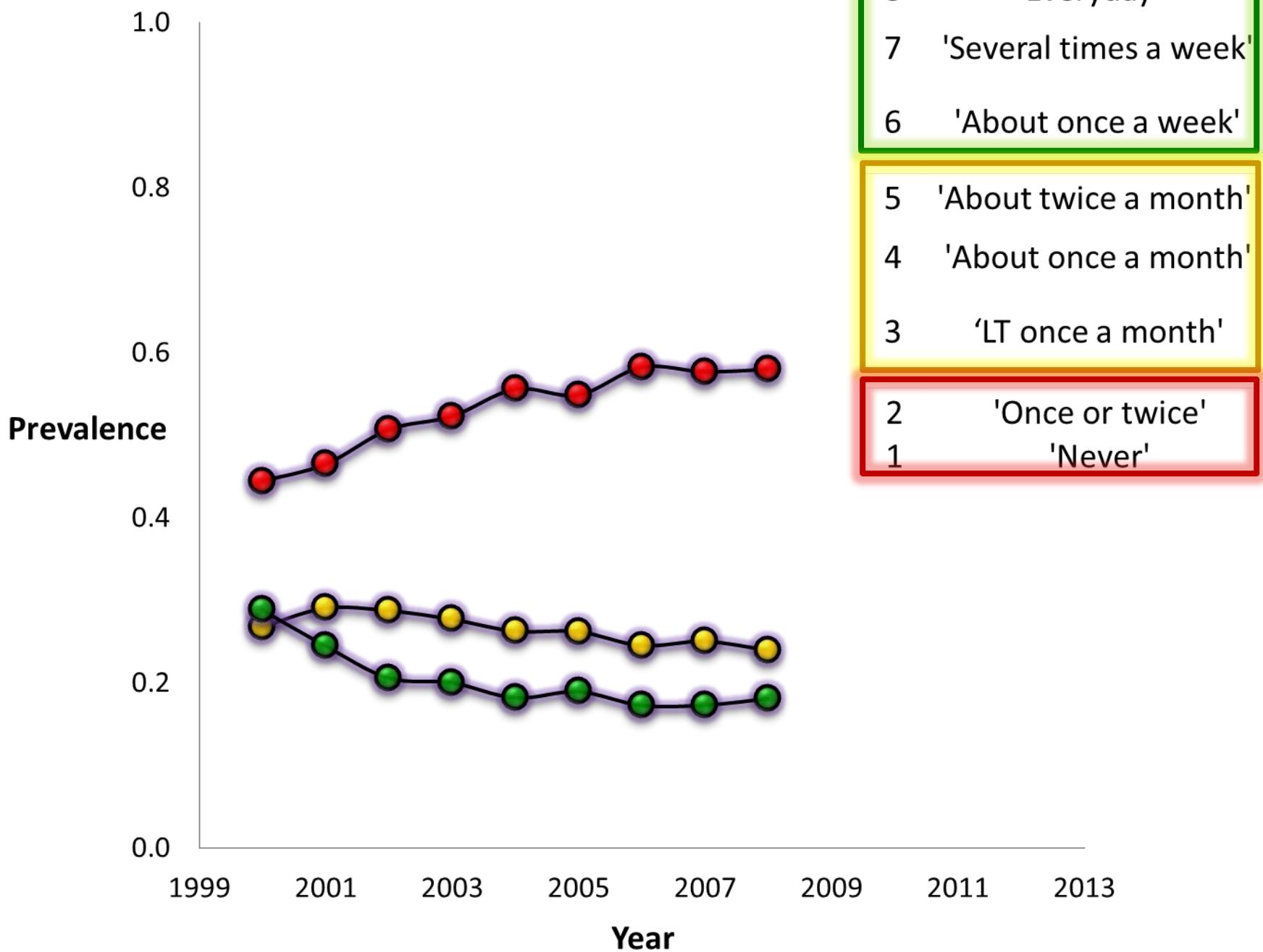






Summary so far

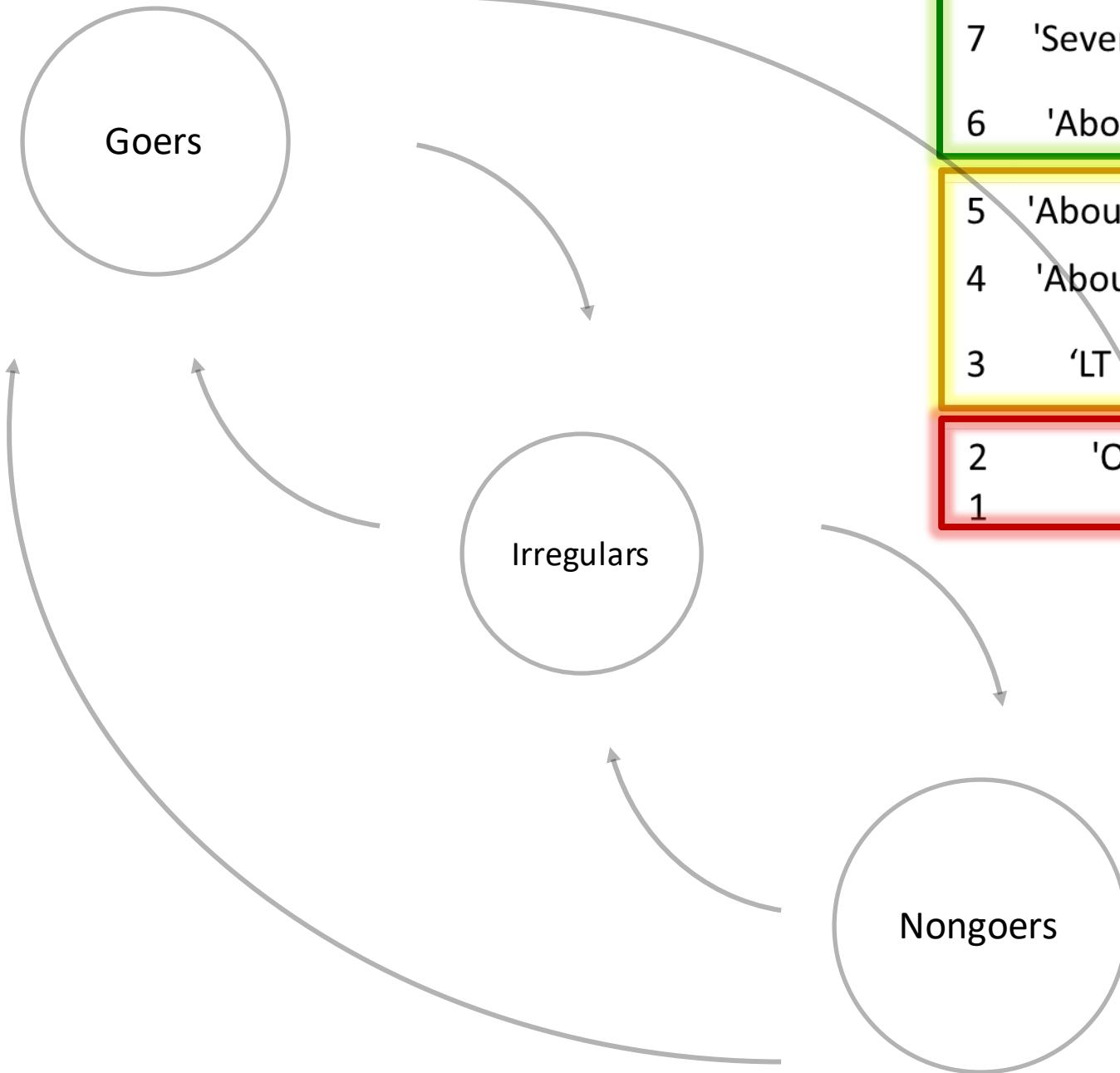
- Religious Involvement changes rapidly between 16 and 21 years of age
- But does not change much after that
- Goers are most AGE diverse / predictive
- Irregulars are not AGE diverse at all
- Nongoers continue increase in numbers slowly after 21 – secularization effect?



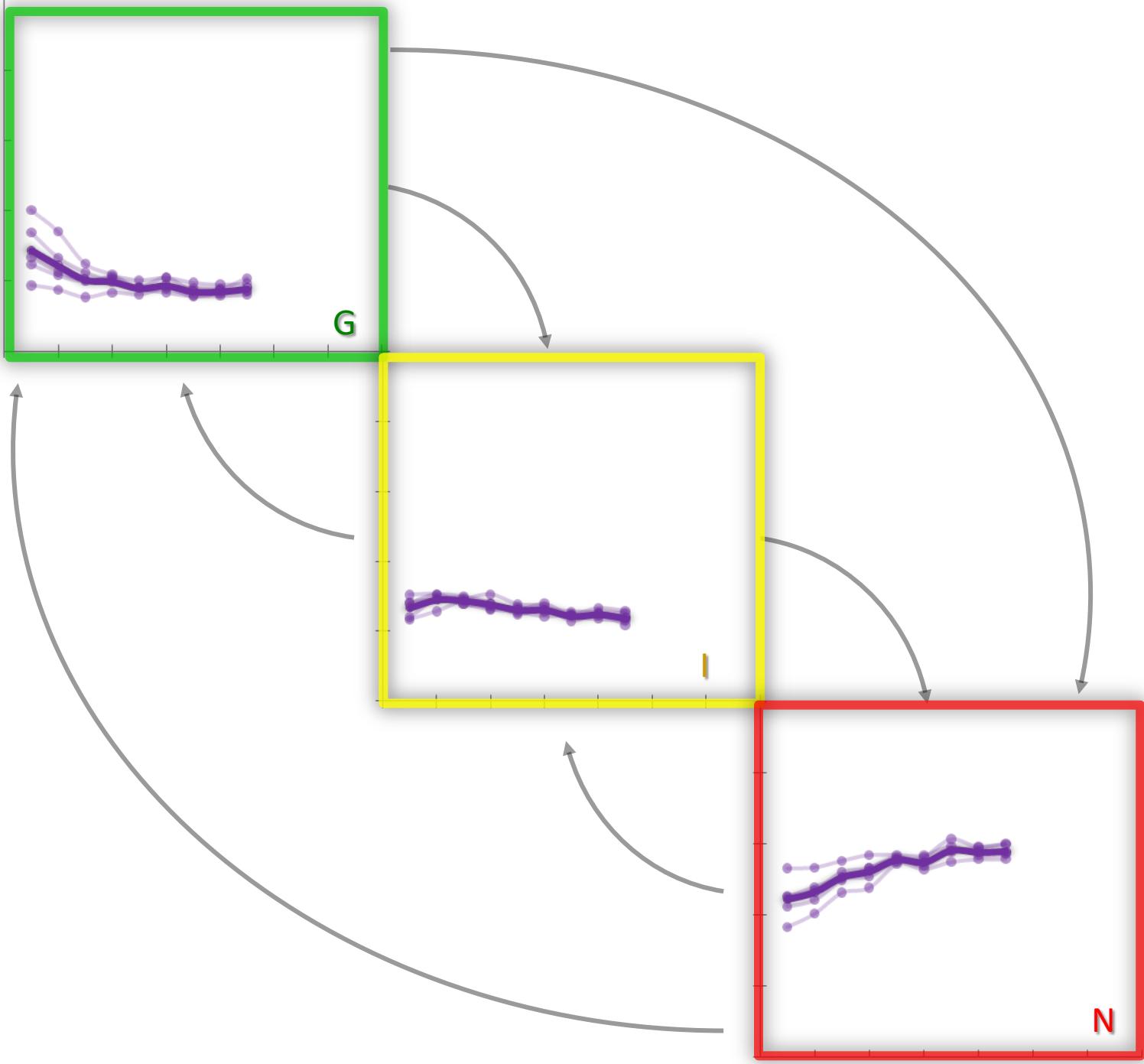
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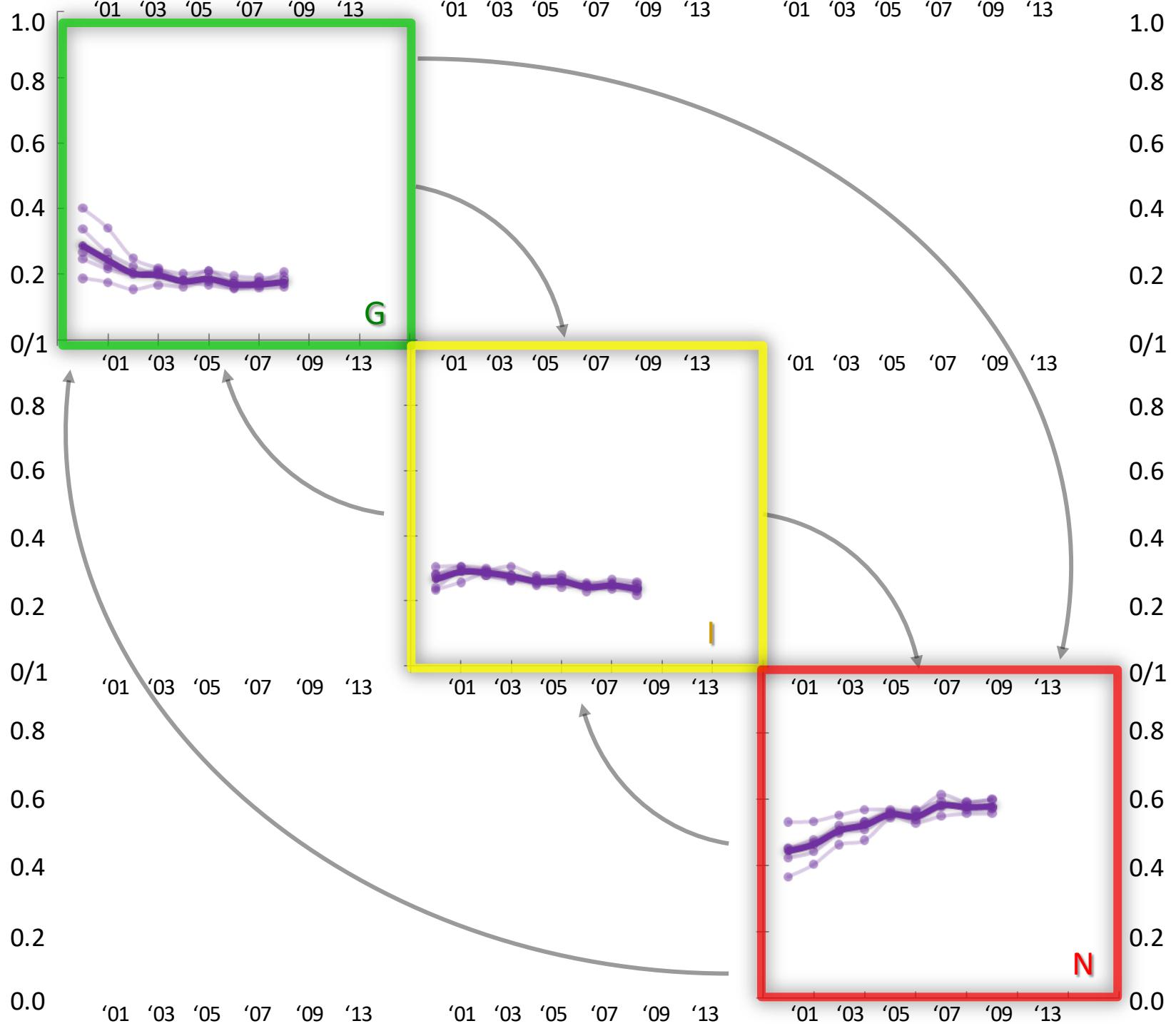
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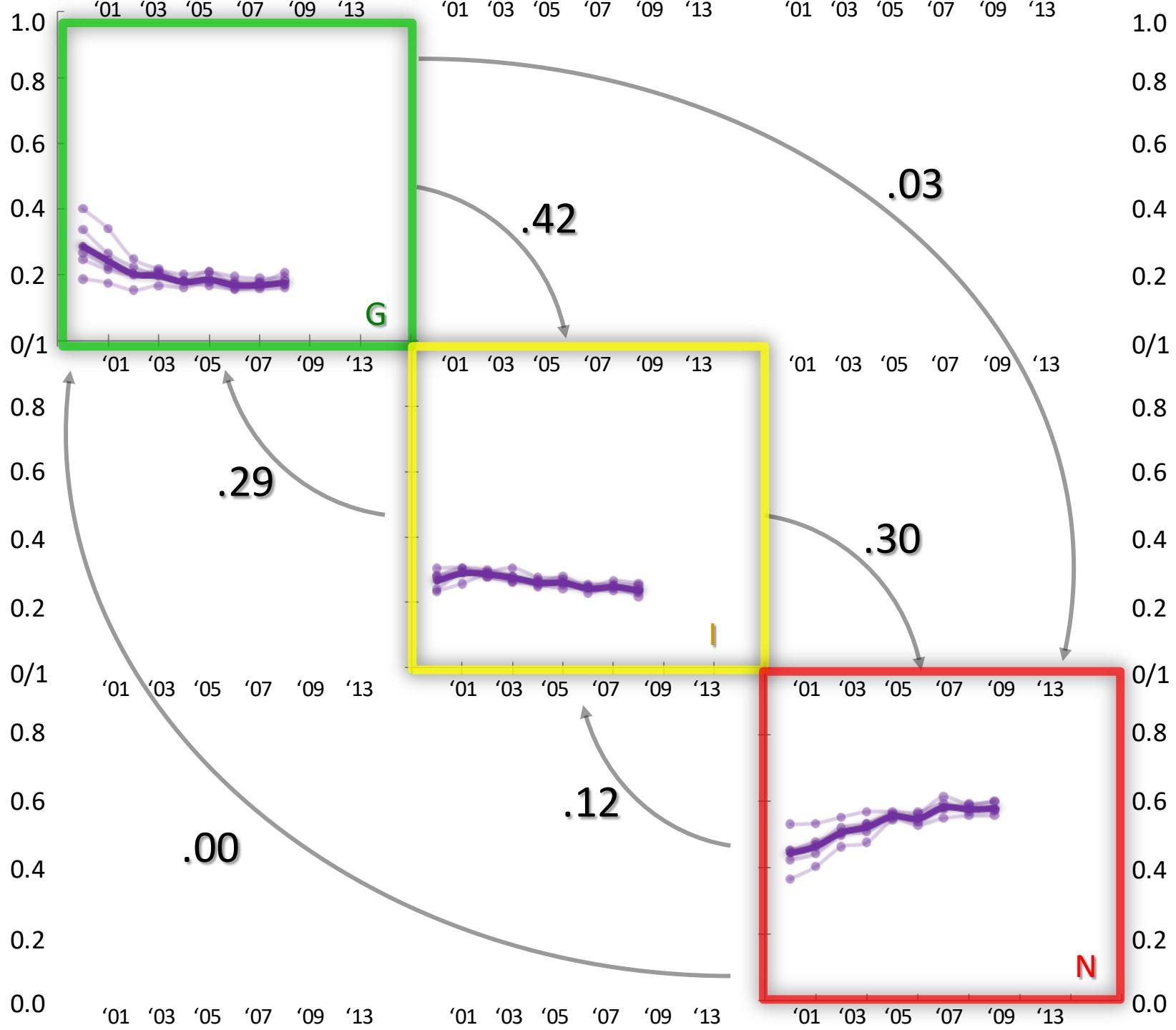
- 2 'Once or twice'
- 1 'Never'

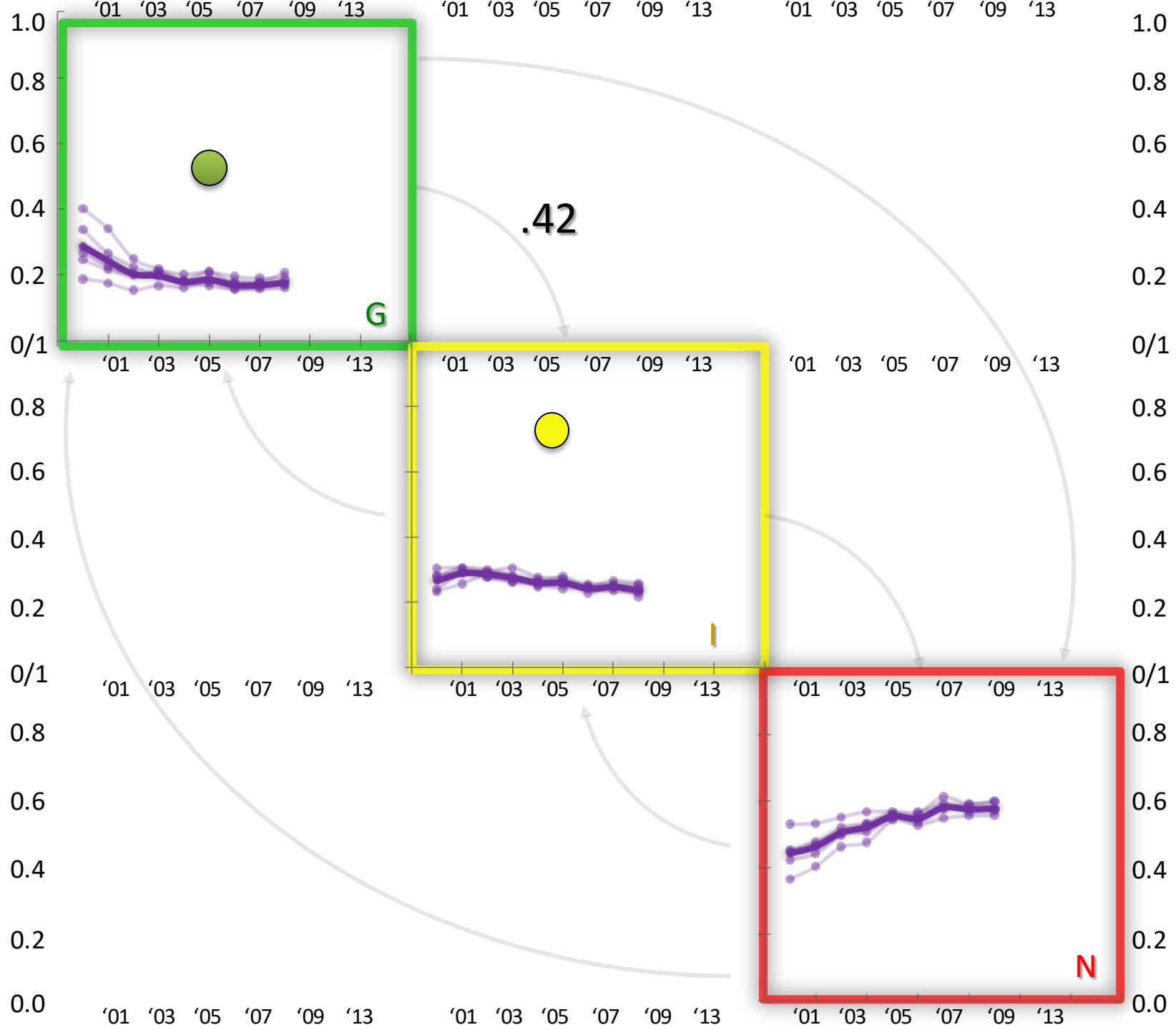


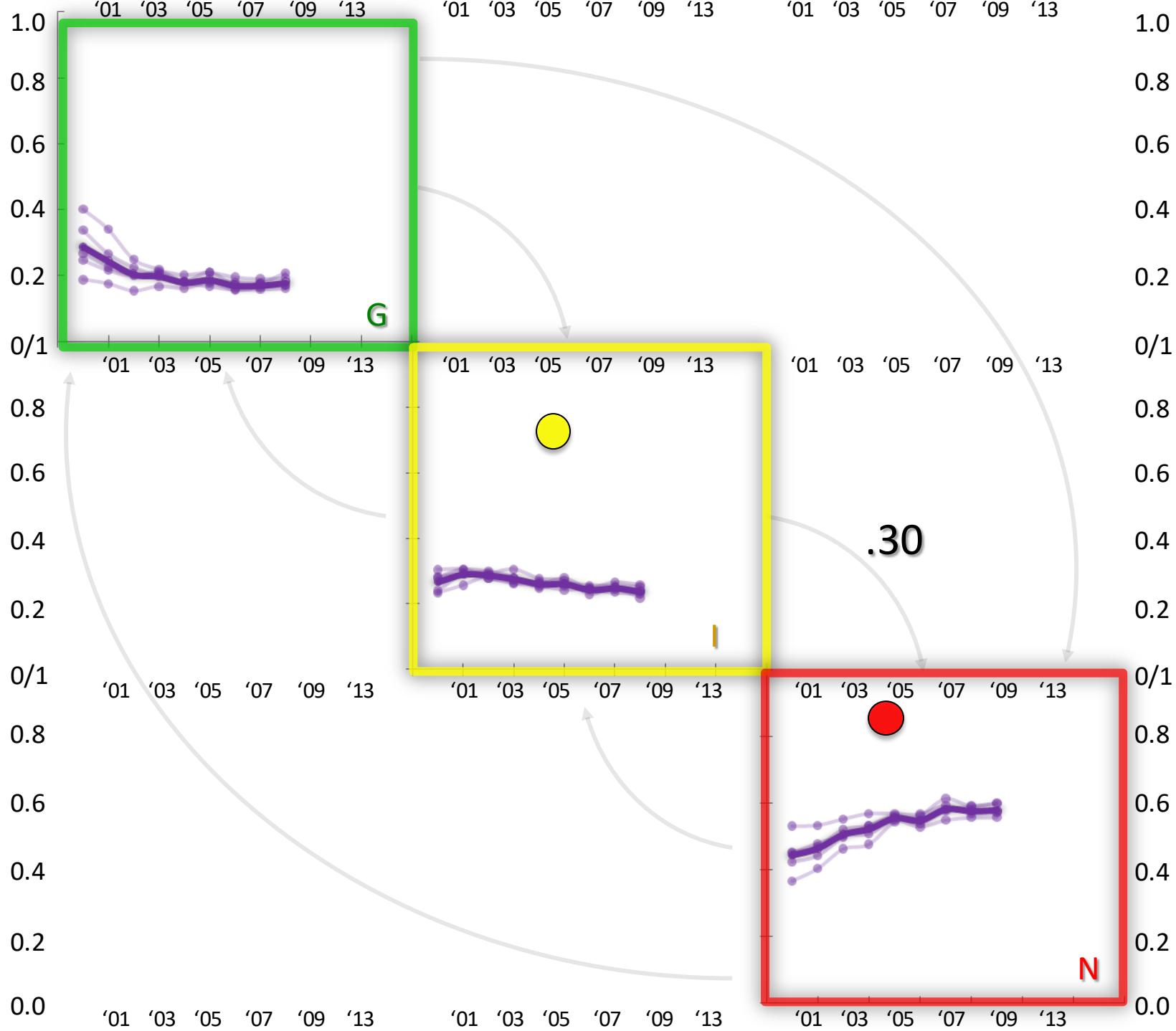
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| 1 | 'Never' |

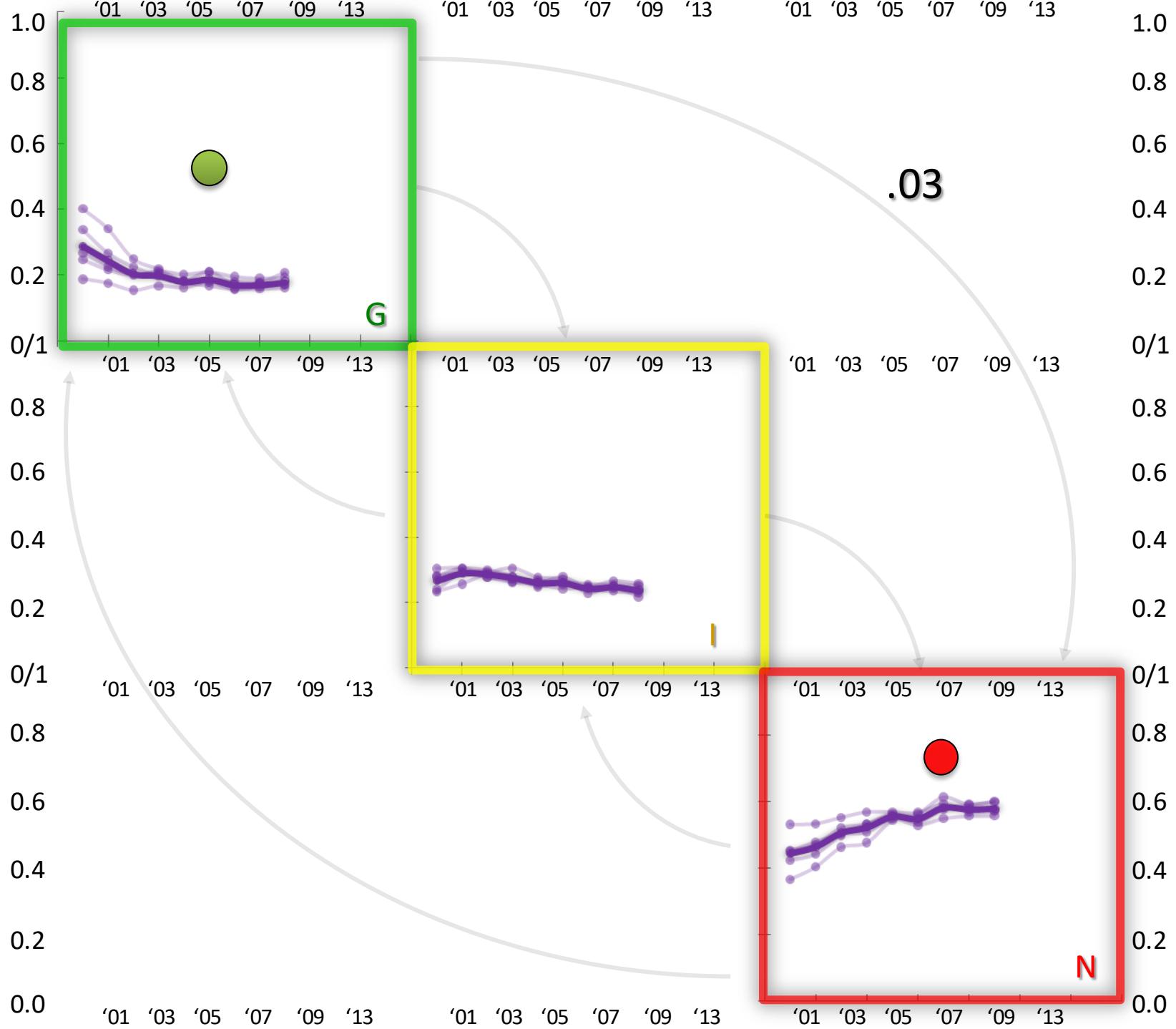


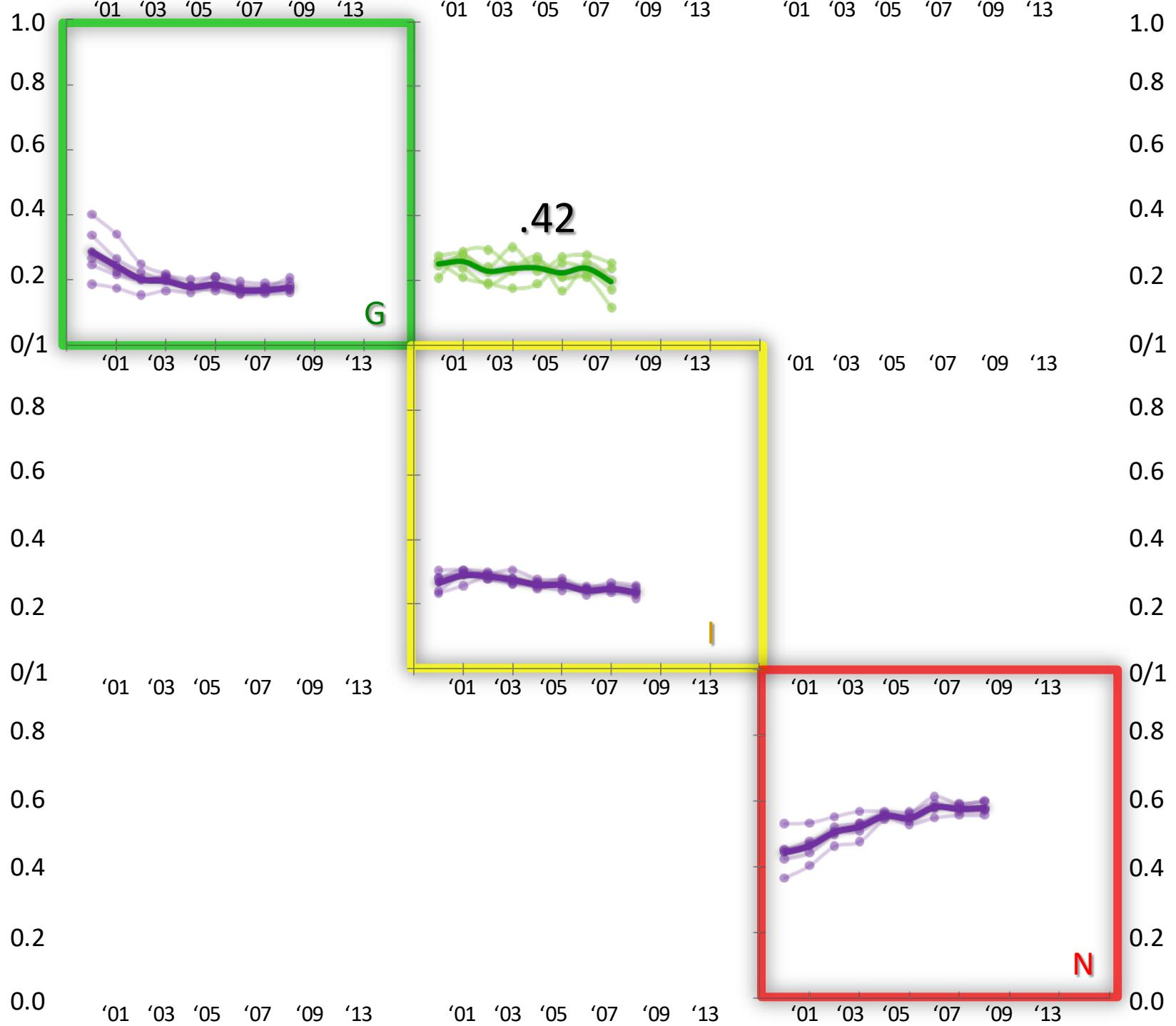


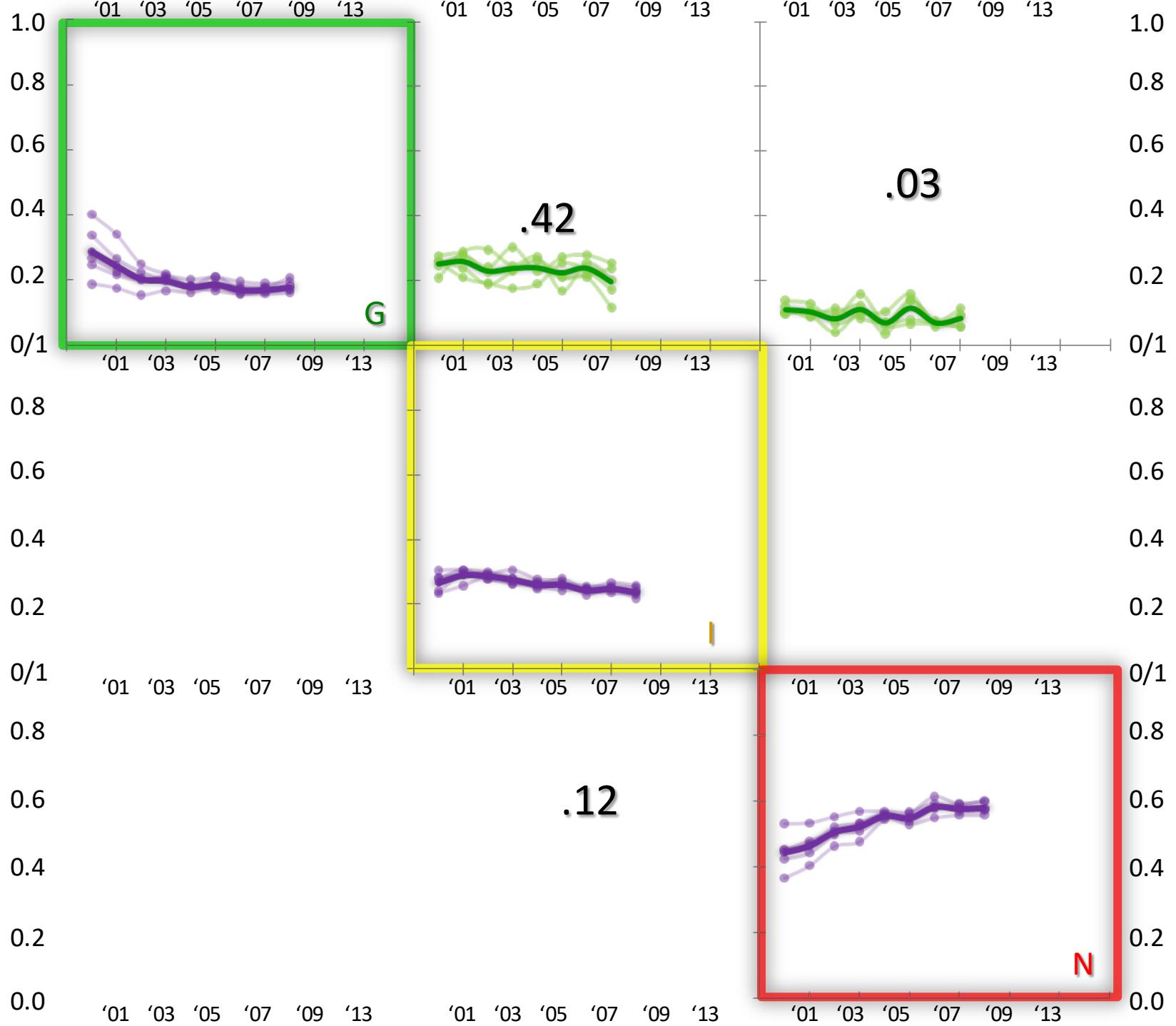


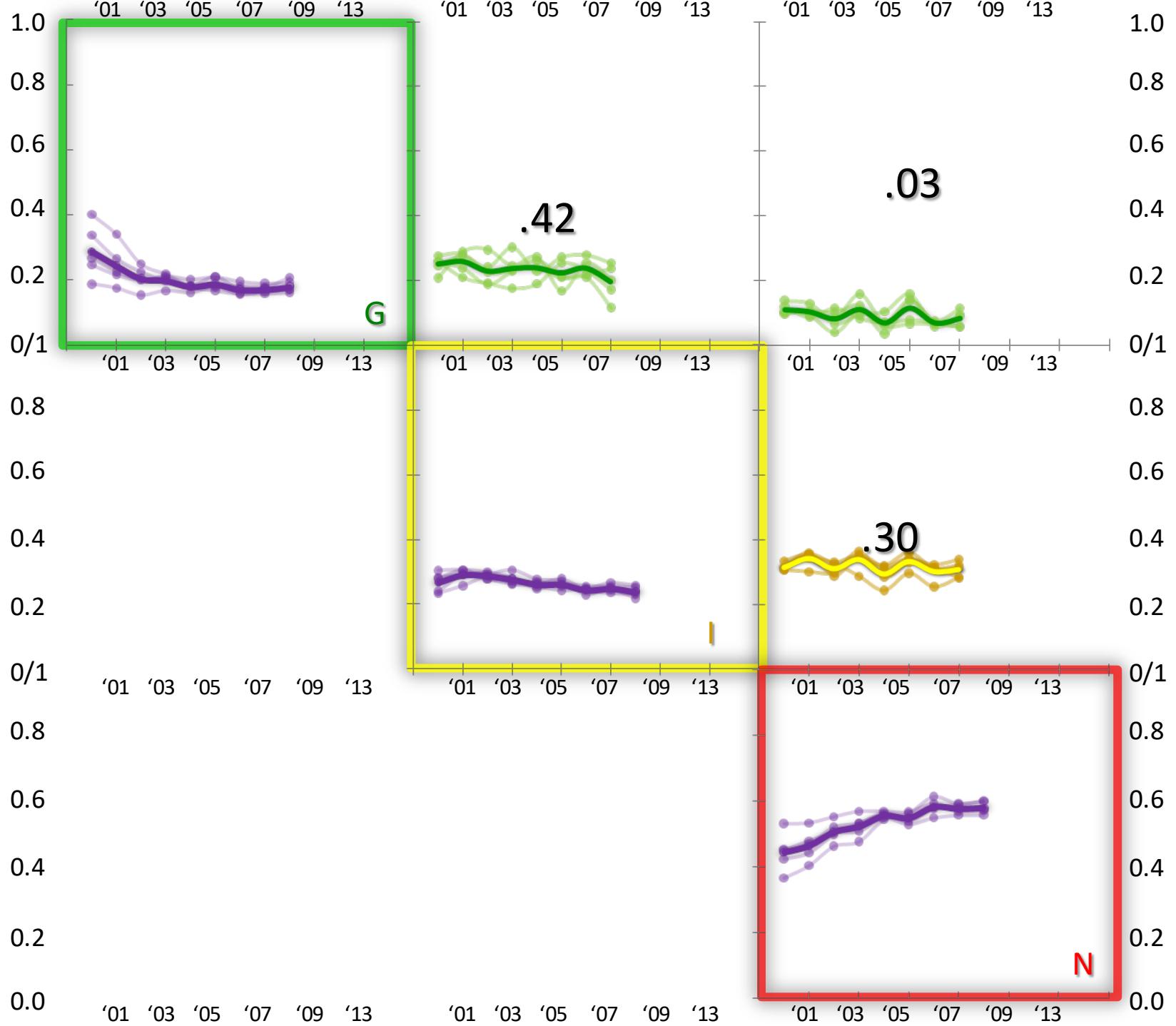


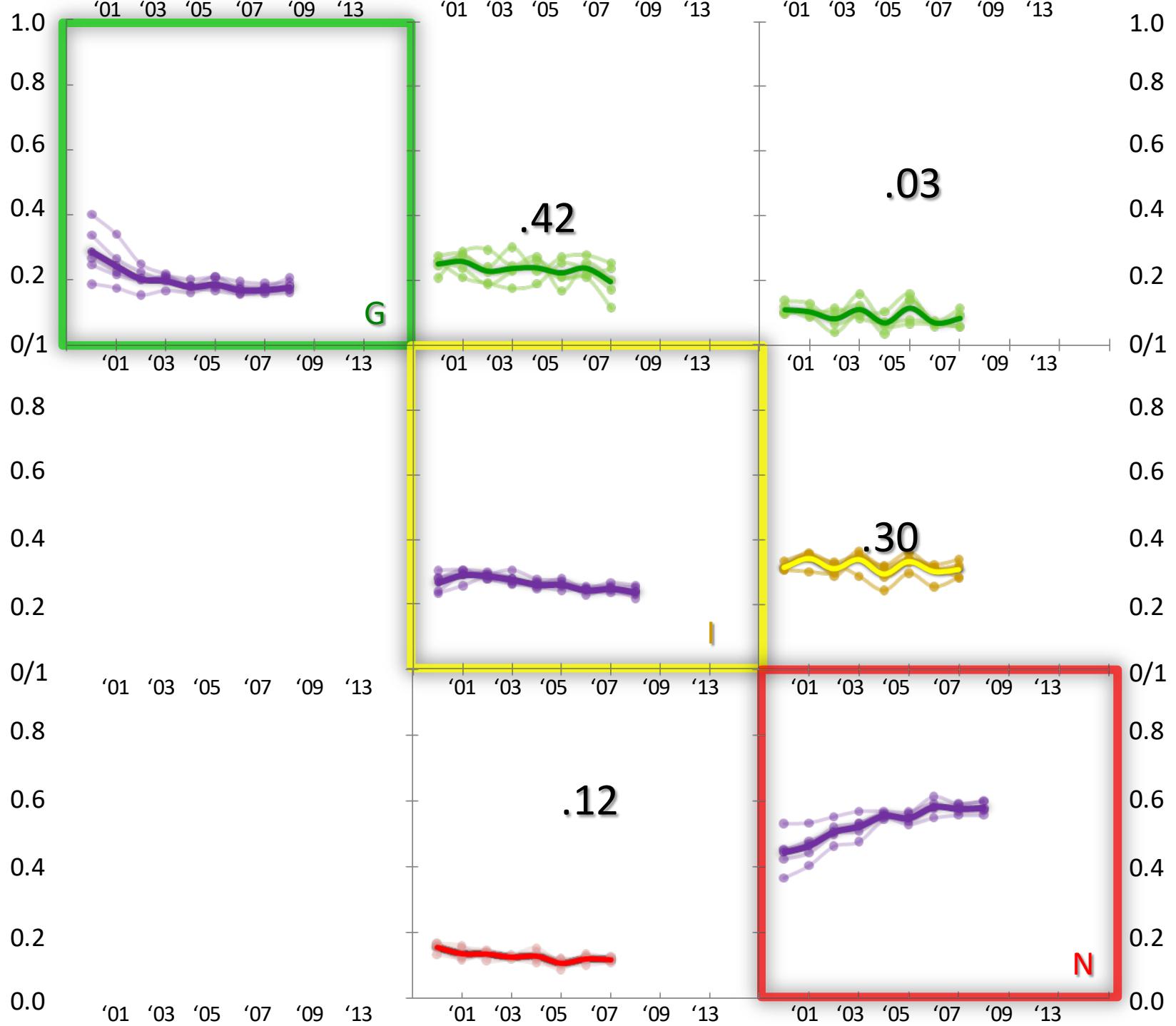


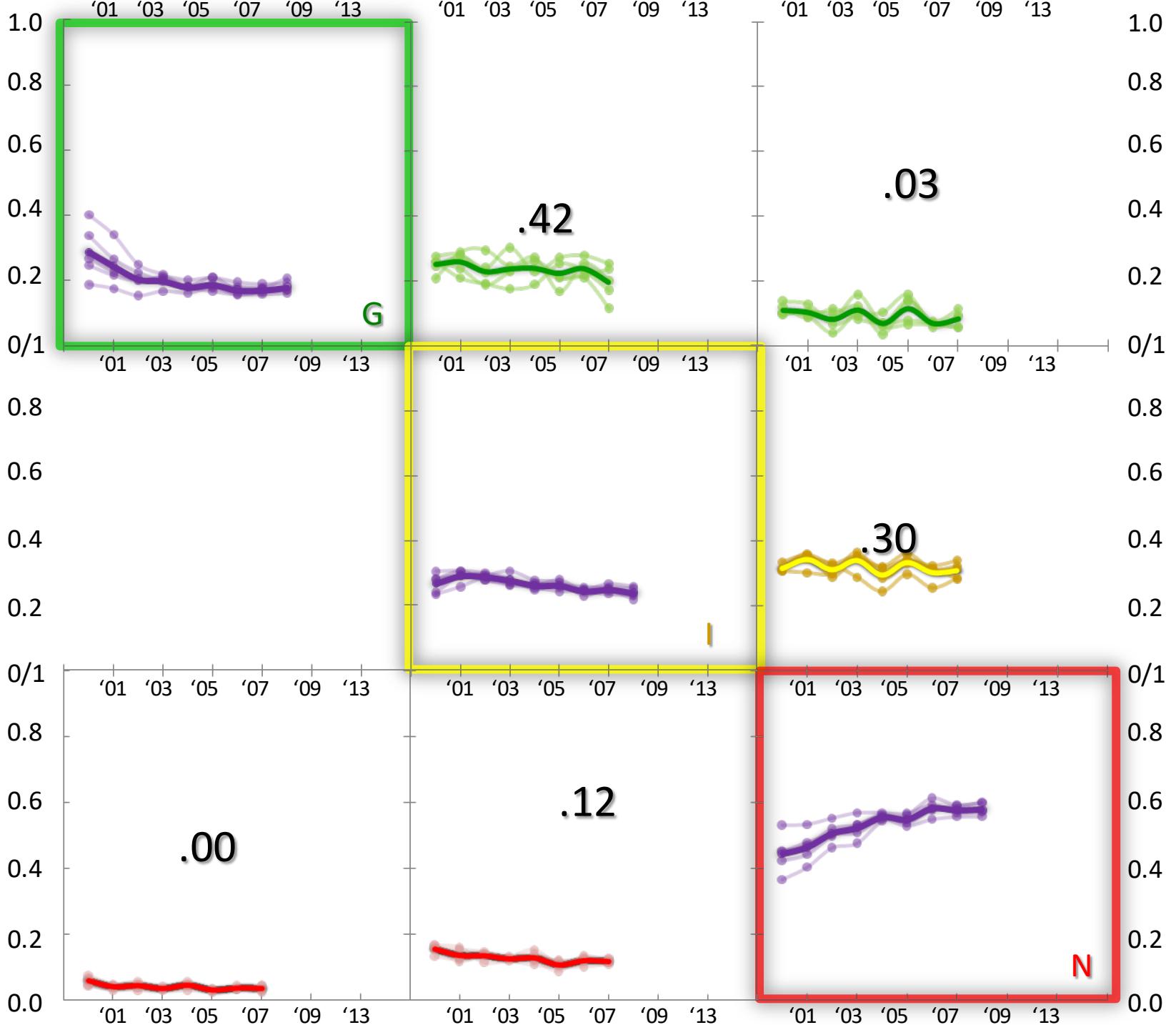


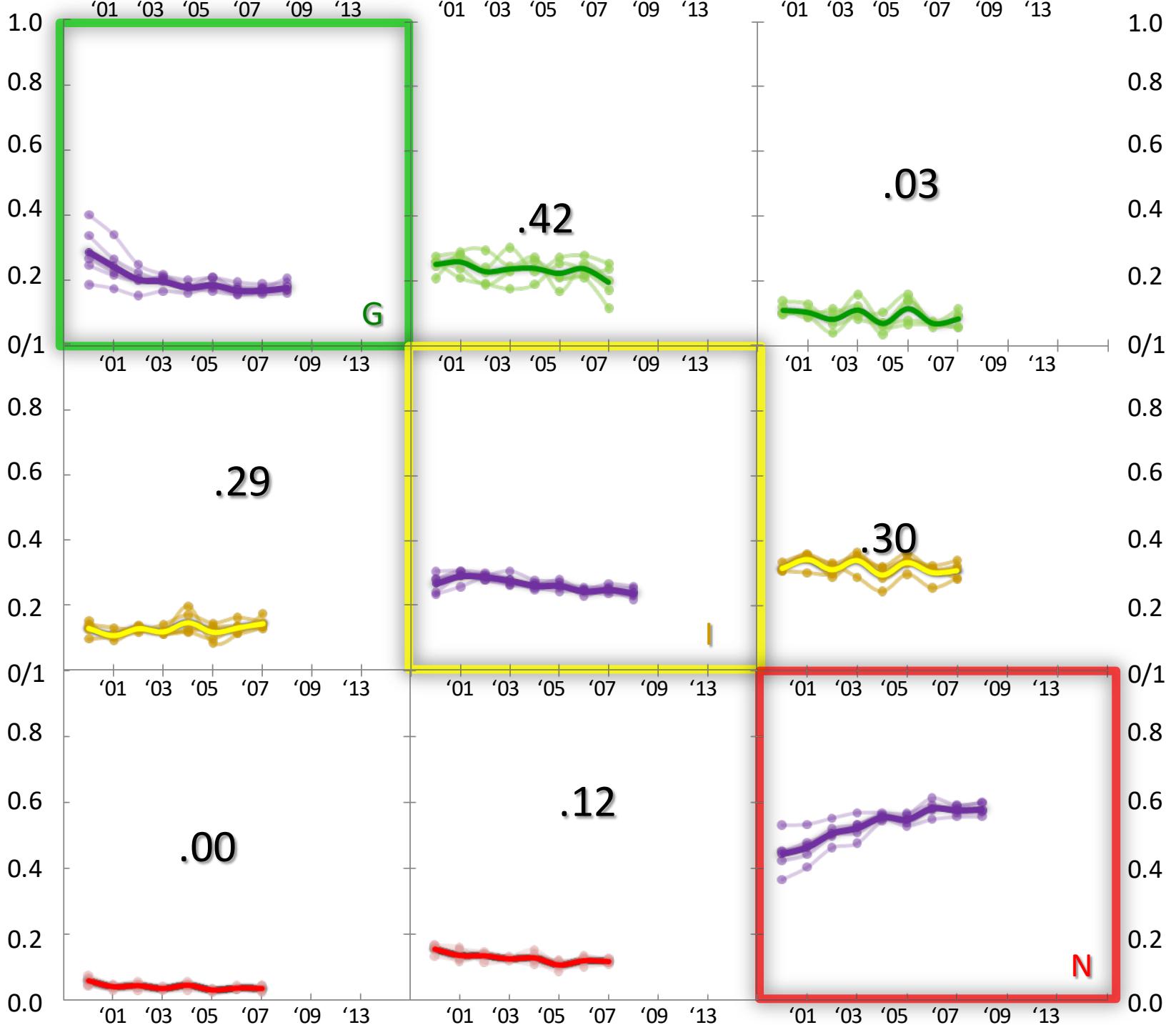


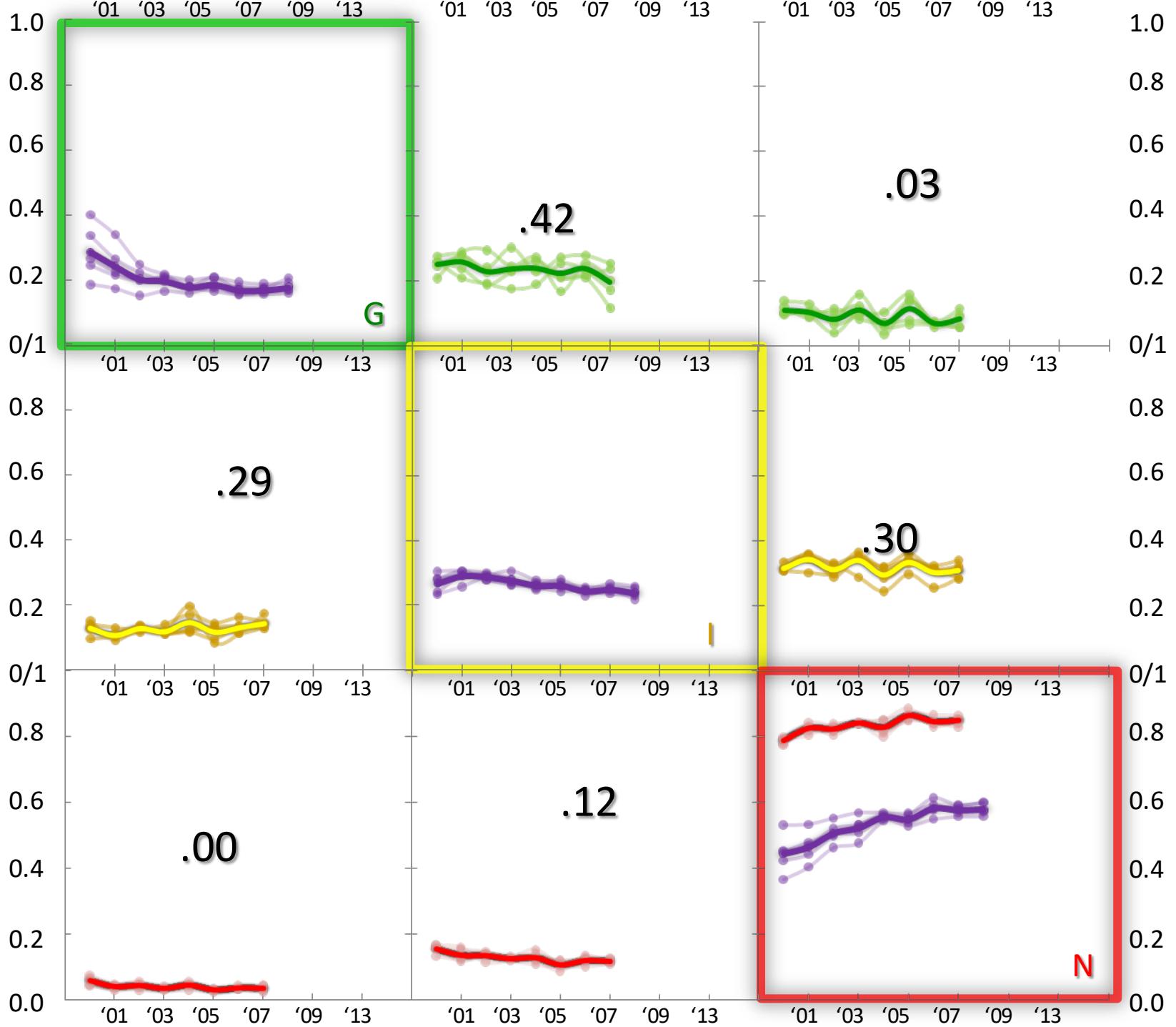


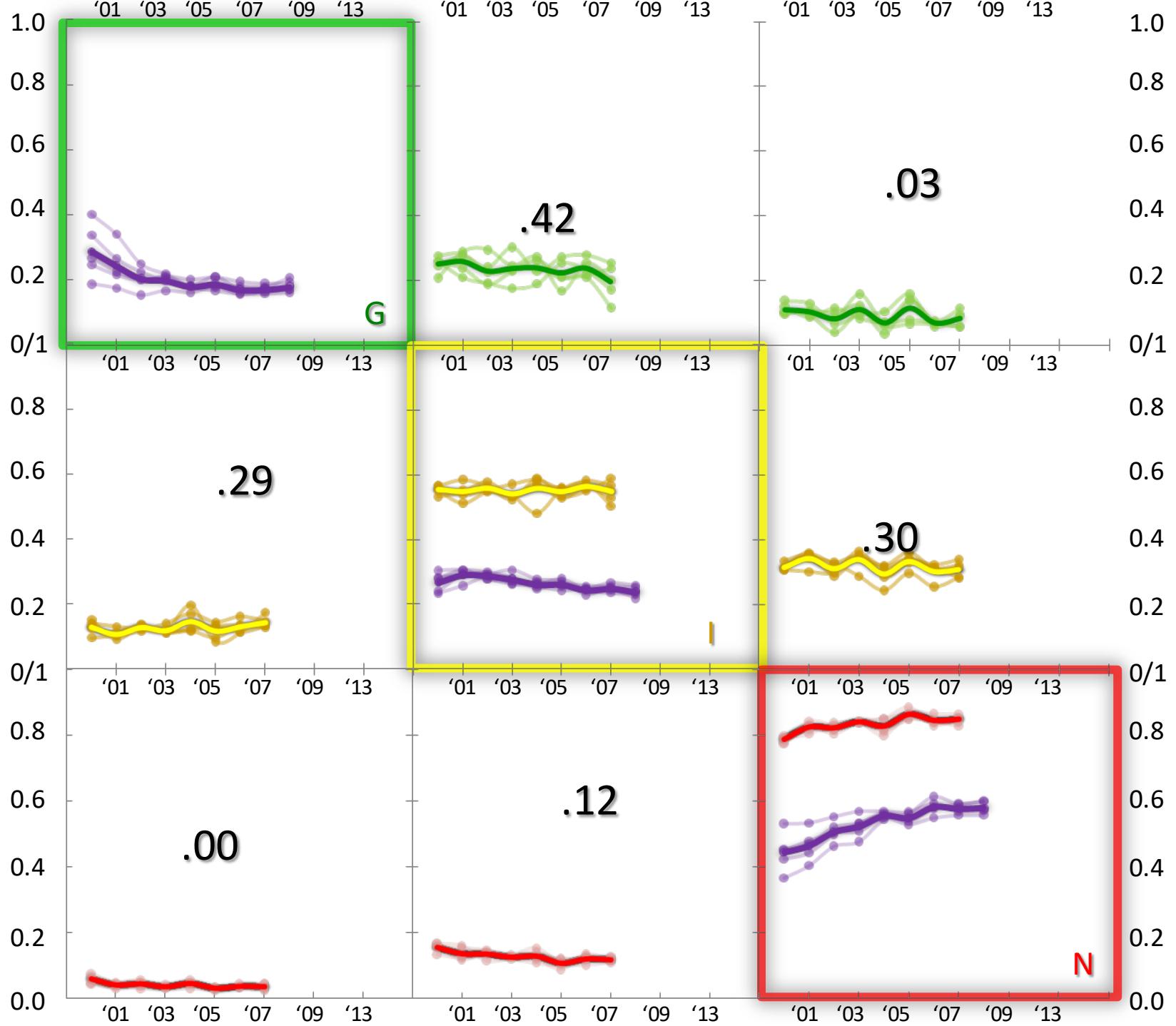


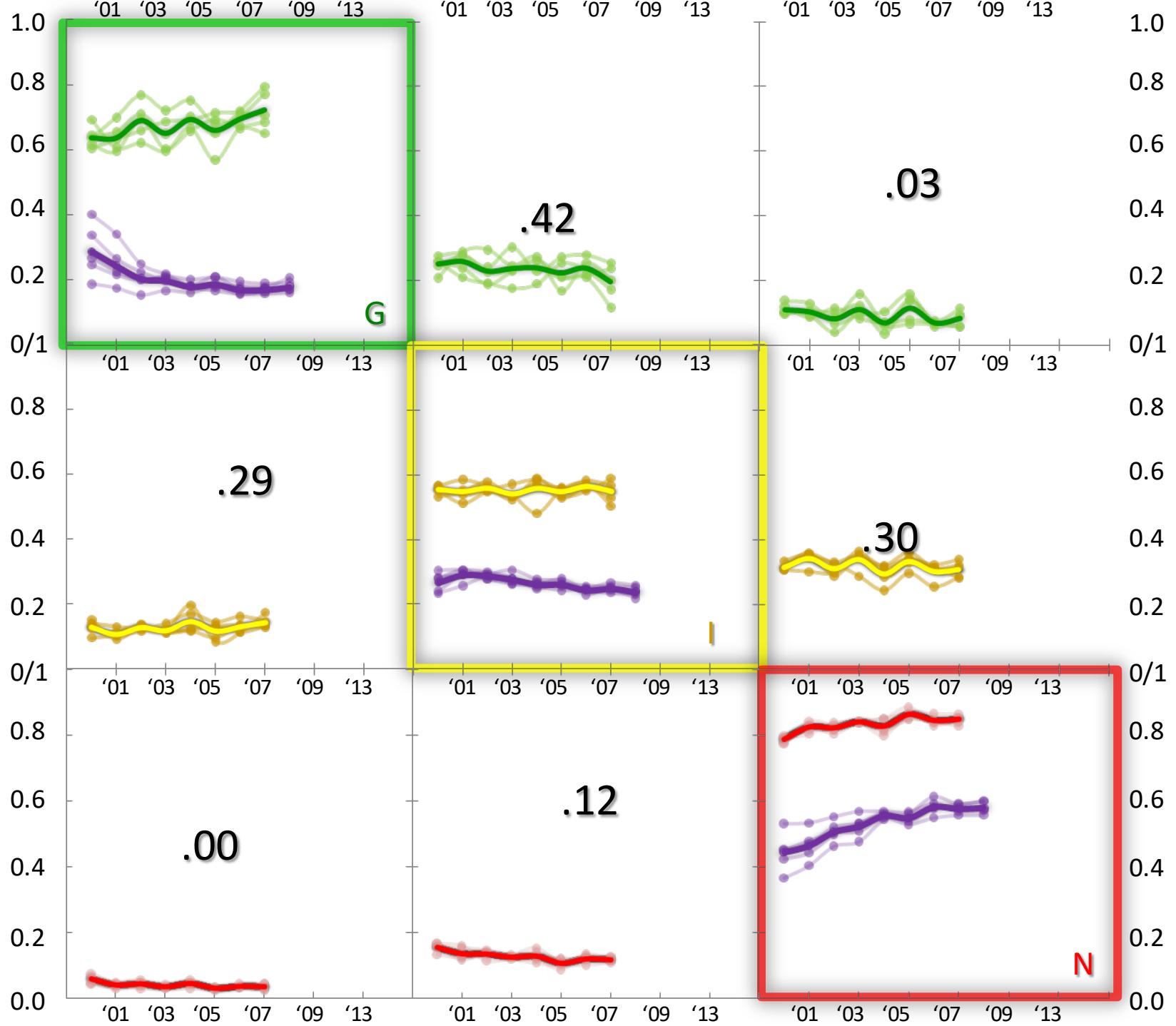






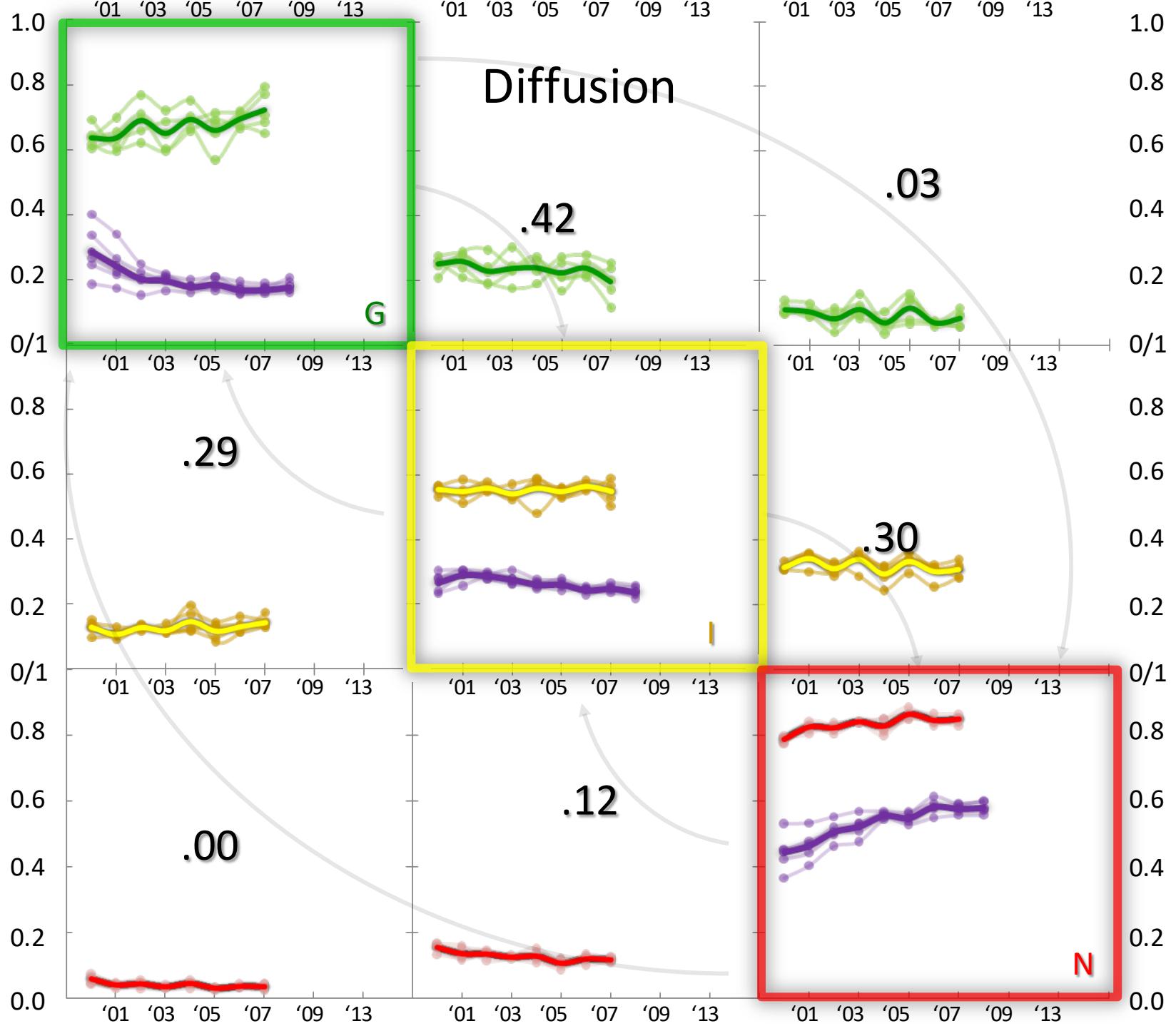


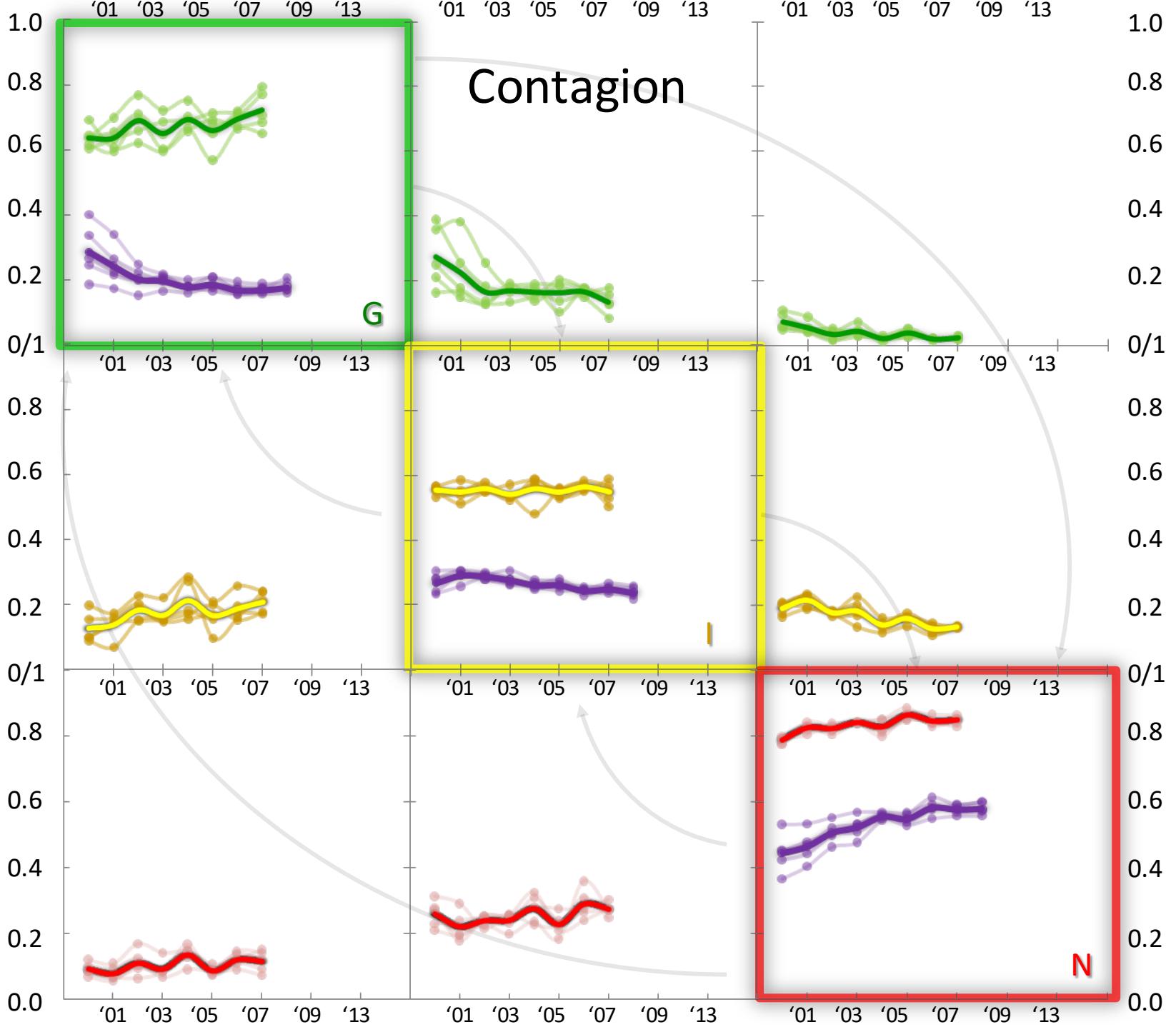


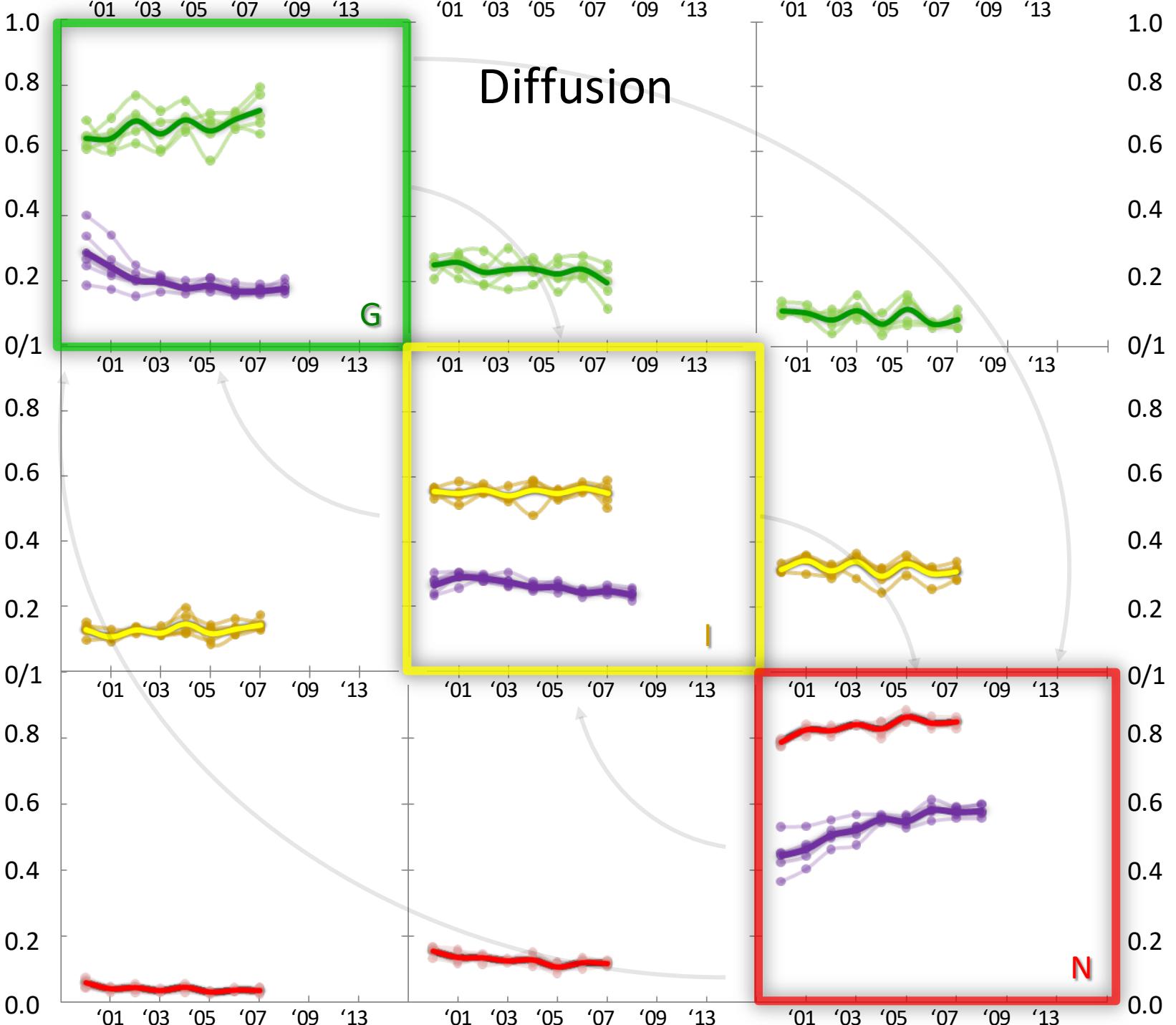




$$\chi^2 = 7.95, df = 6$$







Religious Behavior Resume

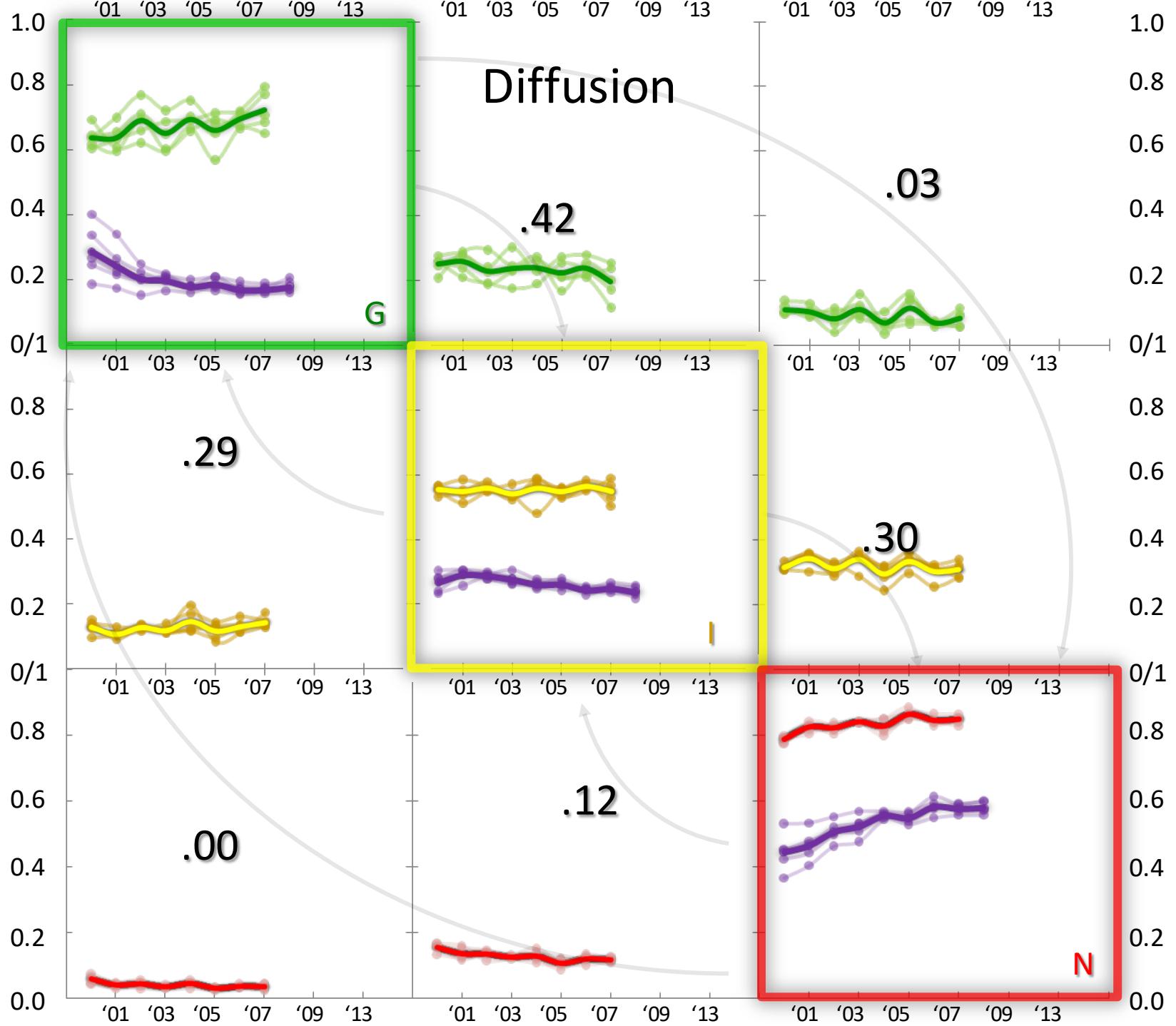
- Nongoers are likely to stay goers. Highest resilience
- Goers rarely become Nongoers, they first become irregulars.
- Nongoers never move back to goers, they become irregulars, for at least awhile
- Goers are the most resilient, but once out, lots happens

Lifecycle/Generation Resume

- Five year difference among cohorts is noticeable only until 21 years of age – evidence for lifecycle effect.
- Categories stabilize at .18(Goers), .27(Irregulars), and .55(Nongoers) – psychological profile of this generation
- System self-regulates: steady ratios and steady transition rates.

EMOSA Resume

- Diffusion explains data better
- Constant rate of transitions as a function of originating category
- Originating vs. Originating + Destination
- Decrease in religious involvement has evidence for contagion, while increase is purely diffusive.
- EMOSA creates a digital fossil of the behavior



Questions?