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Where enterprise leads, finance follows. In-sample and out-of-sample evidence on the causal relation between finance and growth

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Abstract

Evidence on the causality between finance and growth is largely inconclusive. In this study, a large cross-sectional data set of 74 economies for the period between 1975 and 2005 is examined. We summarise evidence from in-sample and out-of-sample causality tests based on rolling estimation steps. It is found that growth impacts on financial development in a stronger way than vice versa. These findings are consistent if economies are categorised into distinct income groups.

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1 Introduction

Whether financial development causes economic growth or vice versa is highly debated. On the one hand, several economists and institutions such as Schumpeter (1911), McKinnon (1973), Shaw (1973), the World Bank (1989) or Levine (2005) emphasise the importance of a developed financial system as a prerequisite for economic growth. They argue that a developed financial system enhances the mobilisation of savings, identifies high return projects, diversifies risks and facilitates transactions. These functions might promote both the overall level and the efficiency of investment. On the other hand, arguments in favour of the reverse causal direction have been put forth by Robinson (1952), who asserts that '*where enterprize leads finance follows*'. According to this view, the financial system develops in response to the demand generated by a growing real economy. Thirdly, a bidirectional causality between finance and growth has been explicitly asserted by Patrick (1966) and Greenwood and Jovanovic (1990). Patrick (1966) refers to the view that the financial system develops as a result of the demand emanating from growth in the real sector as "demand-following phenomenon". Likewise, he calls the claim that the development of the financial sector ahead of demand induces economic growth as "supply-leading phenomenon".

The literature also suggests that the direction of the finance-growth causality may depend on the level of economic development. In this respect, Patrick (1966) conjectures that "supply-leading" might be more dominant at earlier stages of economic development while "demand-following" plays a significant role at later stages. In the growth model by Greenwood and Jovanovic (1990), however, financial development occurs endogenously at a later stage of economic development, since the creation and deployment of financial institutions is costly.

Intensive empirical research on the causality between finance and growth has provided conflicting evidence. Cross-country studies repeatedly show that financial development impacts positively on economic growth (see King and Levine, 1993; Levine et al., 2000; Beck et al., 2000b; Hassan et al., 2011). However, most of these studies do not explicitly test the possibility that growth might also affect finance. On the other hand, time series based studies arrive at ambiguous conclusions (see Demetriades and Hussein, 1996; Xu, 2000; Christopoulos and Tsionas, 2004; Apergis et al., 2007; Ang and McKibbin, 2007; Ang, 2008; Hassan et al., 2011). Similarly, the evidence with regard to the dependence of the causal directions on the level of economic development is inconclusive. Contrary to Patrick's conjecture, Xu (2000) reports weaker and, for some countries negative, causality from finance to growth in low-income economies and a strong causal impact of finance on growth in high-income economies. However, Hassan et al. (2011) find evidence for bidirectional causality between finance and growth for most geographic regions and evidence of causality from growth to finance in two of the poorest regions. These findings apparently support the predictions of the model by Greenwood and Jovanovic (1990).

We contribute to the empirical literature in three aspects. Firstly, we investigate the causal impact of financial development on growth (abbreviated FG henceforth) and the reverse direction (GF) by means of both in-sample (IS) tests and out-of-sample (OS) forecast comparisons. To this end, we rely on summarising economy-specific evidence from bivariate SUR models. We focus on causal relations regarding the short- to medium term, hence we examine growth rates of the observed time series. This means that causality tests

refer to short and medium term periods of less than one decade, corresponding to typical planning horizons of institutional decision takers. Furthermore, impulse response functions are employed to investigate the direction and dynamic behaviour of the causal relations. Secondly, a large cross section dimension allows us to examine whether causal effects depend on an economies' individual stage of development (Patrick, 1966). Hence, we examine causality test results for subgroups of economies, which are distinguished according to their level of income. Thirdly, the potentially time-dependent nature of causal relationships might be a reason for conflicting empirical evidence. Therefore, we test for causality in an iterative way, relying on a short subperiod of the entire time dimension at each estimation step. The remainder of this study begins with an introduction of the data in Section 2. The IS and OS approaches to causality testing are described in Section 3, followed by a discussion of results. Section 4 concludes.

2 Data

We employ annual data from 74 economies covering the period 1975-2005. Economies are classified into four income groups[1] based on their latest (2005) real GDP per capita and the World Bank's classification criteria in 2006.[2] We employ a widely used measure of financial development, namely credit by deposit money banks and other financial institutions to the non financial private sector as a percentage of GDP ($PRIV$, in growth rates). This data is taken from the 2008 update of the *Financial Development and Structure Database* of Beck et al. (2000)[3]. The merits of $PRIV$ are that it singles out credit to the private sector and, moreover, excludes credit issued by the central bank. Consequently, it is argued to be more suitable to examine the impact of financial development on economic growth than other measures, as, for instance, the ratio of monetary aggregates M2 or M3 to GDP (De Gregorio and Guidotti, 1995; Levine et al., 2000). Economic growth is expressed by the growth rate of real GDP per capita ($GROW$). We control for inflation and an economies' openness to trade as two widely used determinants of economic growth and financial development (Levine et al., 2000; Baltagi et al., 2009; Bittencourt, 2011; Badinger and Nindl, 2012). Inflation

[1] *Low income*: Burkina Faso, Burundi, Cameroon, Cote d'Ivoire, Gambia, Ghana, India, Kenya, Lesotho, Madagascar, Nepal, Niger, Nigeria, Pakistan, Papua New Guinea, Rwanda, Senegal, Sierra Leone, Togo.
Lower middle income: Algeria, Dominican Republic, Ecuador, Egypt, El Salvador, Fiji, Guatemala, Honduras, Paraguay, Philippines, South Africa, Srilanka, Suriname, Swaziland, Syria, Thailand.
Upper middle income: Botswana, Chile, Costa Rica, Gabon, Malaysia, Malta, Mauritius, Mexico, Saudi Arabia, Seychelles, St. Vincent and the Grenadines, Trinidad & Tobago, Uruguay, Venezuela.
High income: Australia, Austria, Bahamas, Belgium, Canada, Cyprus, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Israel, Italy, Japan, Korea, Netherlands, New Zealand, Norway, Portugal, Spain, Sweden, UK, US.

[2] `http://data.worldbank.org/about/country-classifications/a-short-history`. Note, however, that we group Algeria, Cameroon, Malta, Saudi Arabia, and Trinidad and Tobago differently from the World Bank's classification in 2006. This is because our classification is based on GDP, which is typically employed as the measure of economic development in the literature on causality between finance and growth. The World Bank's categorisation, in contrast, is based on the Gross National Income (GNI). However, the GNI-based classification yields qualitatively unaffected findings, which may be obtained from the authors upon request.

[3] `http://go.worldbank.org/X23UD9QUX0`

obtains as the growth rate of the GDP deflator ($INFL$). Trade openness is the growth rate of the ratio of imports plus exports to GDP ($OPEN$). All data series except $PRIV$ are drawn from the 2009 edition of the World Bank's *World Development Indicators* database.

3 Causality testing

Subsequently, we describe the IS and OS approach to testing for causality and discuss the results.

3.1 *In-sample schemes*

For $GROW$ and $PRIV$ in economy i at time t, we estimate bivariate SUR regressions

$$\begin{pmatrix} PRIV_{it} \\ GROW_{it} \end{pmatrix} = \begin{pmatrix} \mu_{i1} \\ \mu_{i2} \end{pmatrix} + \underbrace{\begin{bmatrix} a_{11,i} & a_{12,i} \\ a_{21,i} & a_{22,i} \end{bmatrix}}_{A_i} \begin{pmatrix} PRIV_{i,t-1} \\ GROW_{i,t-1} \end{pmatrix} + B_i \begin{pmatrix} x^{\bullet}_{i,t-1} \\ x^{\bullet}_{i,t-1} \end{pmatrix} + \begin{pmatrix} v_{i1t} \\ v_{i2t} \end{pmatrix}, \qquad \begin{matrix} i = 1, ..., 74, \\ t = \tau - E + 1, ..., \tau, \end{matrix} \tag{1}$$

where $(v_{i1t}, v_{i2t})' \sim (0, \Omega_i)$ and τ denotes the end of the estimation window E.
To address potential structural changes in causal relations, we estimate (1) in a stepwise manner for $\tau = T - T_0, ..., T - 1$. Overall evidence on causality is obtained by subsuming time-local evidence across economies. Predetermined influences are represented as $x^{\bullet}_{i,t-1} \in \{OPEN_{i,t-1}, INFL_{i,t-1}\}$. Distinct control variates $x^{\bullet}_{i,t-1}$ are included in (1) interchangeably to retain a parsimonious model structure for economy-specific estimation. Analysing annual observations, the choice of a single lagged term seems sufficient to model the dynamics in $PRIV$ and $GROW$[4]. The parameters in A_i and B_i express the impact of finance, growth and further predetermined variables, respectively. We distinguish five related null hypotheses of noncausality. In all cases, the alternative hypothesis is that both causal effects hold jointly, i.e. $H_1 : \ a_{12} \neq 0 \ \wedge \ a_{21} \neq 0$ in (1). Conversely, the most restrictive assertion is that both causal effects are absent, i.e. $H_0 : \ a_{12} = a_{21} = 0$. Rejections of $H_{01} : \ a_{12} = 0$ or $H_{02} : \ a_{21} = 0$ indicate that $GROW$ influences $PRIV$ in the former, or the reverse causal effect in the latter case. Furthermore, by consideration of the conditional hypotheses $H_{03} : \ a_{12} = 0 \mid a_{21} = 0$ and $H_{04} : \ a_{21} = 0 \mid a_{12} = 0$, we focus on those instances where only a single causal effect is present, meaning that only one of the unconditional hypothesis H_{01} and H_{02} can be rejected. Rejections of H_{03} or H_{04} provide more clear-cut evidence on the respective importance of the two alternative causal directions. More pronounced evidence for H_{03} than for H_{04} means that where growth increases, the financial development of an economy is likely to follow. For

[4]Inferential results are qualitatively unaffected by consideration of higher lag orders or the joint incorporation of $OPEN_{i,t-1}$ and $INFL_{i,t-1}$ and are available from the authors upon request. Furthermore, we employ several diagnostic tests regarding disturbances from (1) to assess the admissibility of the model specification.

hypotheses testing, we consider F-tests at the 5% significance level[5].

3.2 *Out-of-sample schemes*

Causality may also be detected with reference to forecasting ability. Within each subperiod, one-step predictions obtain as

$$\begin{pmatrix} \widehat{PRIV}^{(\circ)}_{i,\tau+1|t} \\ \widehat{GROW}^{(\circ)}_{i,\tau+1|t} \end{pmatrix} = \begin{pmatrix} \hat{\mu}_{i1} \\ \hat{\mu}_{i2} \end{pmatrix} + \hat{A}^{(\circ)}_i \begin{pmatrix} PRIV_{i,\tau} \\ GROW_{i,\tau} \end{pmatrix} + \hat{B}_i \begin{pmatrix} x^{\bullet}_{i,\tau} \\ x^{\bullet}_{i,\tau} \end{pmatrix}, \tag{2}$$

where $\tau = T - T_0, ..., T-1$ and '$\circ$' refers to estimates under distinct hypotheses $\circ \in \{H_{01}, H_{02}, H_1\}$[6]. At the end of each estimation window τ, forecasts are obtained from estimates $\hat{\mu}_{i1}, \hat{\mu}_{i2}, \hat{A}^{(\circ)}_i, \hat{B}_i$. Forecasting accuracy is evaluated by means of absolute forecast errors

$$\mathrm{AE}^{(\circ)}_{\tau+1|\tau}(y_i) \;=\; |\hat{y}^{(\circ)}_{i,\tau+1|t} - y_{i,\tau+1}|, \tag{3}$$

with $y_{i,\tau+1} \in \{PRIV_{i,\tau+1}, GROW_{i,\tau+1}\}$. Cases where $\mathrm{AE}^{(\circ)}_{\tau+1|\tau}(y_i)$ are lower for predictions from (2) under H_1 than under H_{01} or H_{02} are regarded as evidence for the GF or FG hypothesis, respectively. Rejections of H_0 obtain if predictions under H_{01} and H_{02} are both outperformed by those under H_1. In addition, we consider the binary directional accuracy (DA)

$$\mathrm{DA}^{(\circ)}_{\tau+1|\tau}(y_i) \;=\; \mathrm{I}(\hat{y}^{(\circ)}_{i,\tau+1|\tau} \times y_{i,\tau+1|\tau} \geq 0),$$

where $\mathrm{I}(\cdot)$ is an indicator function. Thus, if the sign of a prediction $\hat{y}^{(\circ)}_{i,\tau+1|\tau}$ matches the one of $y_{i,\tau+1|\tau}$, positivity of $\hat{y}^{(\circ)}_{i,\tau+1|\tau} \times y_{i,\tau+1|\tau}$ indicates a directionally accurate forecast. Since, in contrast to $\mathrm{AE}^{(\circ)}_{\tau+1|\tau}(y_i)$, $\mathrm{DA}^{(\circ)}_{\tau+1|\tau}(y_i)$ increases with predictive accuracy, higher DA under H_1 indicates evidence for the GF or FG hypothesis in this case. The most recent $T_0 \in \{10, 15\}$ years are considered as alternative evaluation samples. The cross section is divided into income-subgroups of sizes $N_g = 19, 16, 14, 25$.

3.3 *Results*

Firstly, the specification of (1) is evaluated by means of residual diagnostics. The total number of tests conducted for each income group of economies is $\mathcal{T}_g = T_0$ (time instances) $\times\, N_g$ (economies). To test for serial correlation, LM tests as introduced by Breusch (1978) and Godfrey (1978) are employed, whereas ARCH-LM tests (Engle, 1982) serve as a means to assess heteroscedastic features in the estimated residuals. Additionally, we test for

[5]Test outcomes are qualitatively similar for alternative significance levels of 1% or 10% and are available from the authors on request.

[6]The testing procedure could equivalently depart from imposing an a priori constraint on A_i according to H_0 and, consequently, regarding $H_{01}, ..., H_{04}$ and H_1 as alternative hypotheses. However, since this setting might give rise to omitted variables bias, we regard H_1 as the superior reference.

nonnormality of the residuals from (1) by means of the Lilliefors (1967) test. Since residual characteristics might differ across economies and given the relatively large number of model evaluations $\mathcal{T}_g$, the application of a nonparametric test might be preferable to more restrictive testing procedures. Test results are summarised in table 1. On average over economies and time instances, we find only little evidence for serial correlation. Rejection frequencies hardly exceed the significance level of 5%. The ARCH-LM tests and nonnormality tests additionally indicate that SUR disturbances may be characterised as white noise processes in the majority of cases.

Table 1: Residual diagnostic tests results

$T_0 = 15$ (1991-2005)			$T_0 = 10$ (1996-2005)	
Dep. var.:	*PRIV*	*GROW*	*PRIV*	*GROW*
Serial correlation LM test				
low	0.70	3.16	0.53	3.16
lower middle	8.75	7.50	6.88	6.88
upper middle	3.81	1.90	0.71	2.14
high	8.27	5.33	7.60	6.80
Heteroscedasticity (ARCH-LM) test				
low	10.53	10.18	13.16	12.11
lower middle	13.75	12.08	14.37	15.63
upper middle	10.48	6.67	10.71	9.29
high	15.20	9.87	17.60	10.00
Nonnormality test (Lilliefors test)				
low	8.07	12.98	6.32	12.11
lower middle	12.92	12.08	15.00	15.63
upper middle	12.38	15.71	14.29	14.29
high	14.13	8.80	18.40	9.60

Note: Reported numbers represent percentages of $\mathcal{T}_g$ instances where test statistics indicate rejections of the null hypotheses of 1.) no first order serial correlation, 2.) no conditional heteroscedasticity or 3.) no deviations from normality in estimation disturbances from (1) at the 5% level. Results for alternative significance levels of 1% or 10% are qualitatively similar and available from the authors upon request.

The outcomes of the IS tests and OS results are reported in tables 2 and 3, respectively. Summary statistics for IS tests refer to fractions of all $\mathcal{T}_g$ cases where F tests indicate rejections of $H_0, ..., H_{04}$ with 5% significance. The results of the OS study in table 3 are summarised analogously as percentages of all $\mathcal{T}_g$ cases where the prediction scheme in (2) obtains higher AE losses or lower (DA) gains than under H_0, H_{01} or H_{02} than under H_1. Given a significant amount of evidence for bidirectional causality, the results in table 2 show that evidence in favour of the GF effect is stronger than for the reverse causal impact. Rejection frequencies for H_{01} are in almost all cases higher than for H_{02}, irrespectively of further control variables. The outcomes are also robust across income groups. Rejections of H_{03} and H_{04} reinstate these findings, i.e. for cases where causality points in only one direction, the impact of growth on finance is more pronounced than vice versa. Similarly, the higher rejection frequencies in table 3 suggest that there is stronger OS evidence against H_{01} than against

H_{02}. Hence, the incorporation of $GROW$ as a predictor variable for $PRIV$ is more likely to decrease the AE (and increase DA) of forecasts than the reverse way[7]. Apart from the direction of causality, the sign and the dynamics of the relation between $PRIV$ and $GROW$ might be of interest for economic policy. To investigate these issues, we report generalised impulse response functions (IRF) as introduced by Pesaran and Shin (1998). This sort of IRF addresses the potential emergence of instantaneously correlated shocks without being affected by the ordering of the variables in (1), in contrast to orthogonal IRFs based on the Cholesky decomposition[8]. In figure 1, these IRFs display the dynamic responses as implied by estimation of (1) to shocks in $PRIV$ and $GROW$, on average across $\mathcal{T}_g$ instances. The graphs show that the impact of shocks in $GROW$ on $PRIV$ and the reverse effect are positive for lower and intermediate income groups. However, the instantaneous effect of $PRIV$ on $GROW$ and vice versa are significantly negative for high income economies, though the impact of $GROW$ on $PRIV$ is relatively small in magnitude. The most pronounced negative impact points from $PRIV$ to $GROW$ for high income economies. These findings are largely in line with those obtained, e.g., by Hassan et al. (2011). In sum, evidence from both IS and OS schemes more strongly supports the view that '*where enterprise leads finance follows*' (Robinson, 1952) than the 'finance leads growth' hypothesis.

4 Conclusions

We find stronger evidence for the hypothesis that economic growth influences financial development than for the reverse causal effect. Our findings are consistent across income groups and confirmed by in-sample and out-of-sample causality testing. By means of impulse response functions we document that the positive association between finance and growth might turn negative in the short run for high-income economies.

[7]The DA statistics are throughout above 50%, which implies that all model specifications deliver economically meaningful predictions. To economise on space, we do not report DA statistics for predictions under each hypothesis. However, corresponding results are available from the authors upon request.

[8]The results obtained with orthogonalised IRFs as implied by the Cholesky decomposition are, however, qualitatively equivalent to the ones reported in figure 1 and may be obtained from the authors upon request.

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Table 2: IS results

$T_0 = 15$ (1991-2005)															
Control var.:	none					$OPEN_{i,\tau-1}$					$INFL_{i,\tau-1}$				
	H_0	H_{01}	H_{02}	H_{03}	H_{04}	H_0	H_{01}	H_{02}	H_{03}	H_{04}	H_0	H_{01}	H_{02}	H_{03}	H_{04}
low	16.49	13.33	11.23	12.28	10.18	9.82	12.28	10.88	11.58	10.18	11.93	10.18	12.28	9.12	11.23
lower middle	24.58	22.08	12.92	19.17	10.00	30.42	25.83	13.33	22.08	9.58	22.92	23.33	8.33	20.83	5.83
upper middle	29.05	23.81	21.43	14.29	11.90	25.71	20.00	21.90	10.48	12.38	20.95	18.10	16.67	10.00	8.57
high	30.40	32.53	14.13	25.87	7.47	29.33	30.40	13.07	25.33	8.00	30.40	29.33	14.93	24.27	9.87
$T_0 = 10$ (1996-2005)															
low	17.89	12.63	11.58	12.11	11.05	8.95	15.79	6.84	15.79	6.84	10.53	12.63	9.47	12.11	8.95
lower middle	27.50	25.00	13.75	21.88	10.63	31.87	29.38	11.88	25.62	8.13	25.62	25.00	7.50	21.88	4.38
upper middle	30.71	22.86	20.00	15.71	12.86	25.00	18.57	19.29	10.00	10.71	20.00	16.43	15.00	9.29	7.86
high	35.20	36.80	16.00	28.00	7.20	32.40	33.60	14.80	27.20	8.40	34.00	33.20	16.80	25.60	9.20

Note: Cell entries report rejection frequencies of distinct null hypotheses of noncausality at the 5% significance level. Evidence for GF obtains as rejections of H_{01} and H_{03}, respectively, whereas evidence for FG is measured by rejection frequencies of H_{02} and H_{04}. Rejecting H_0 indicates bidirectional influence. Columns $OPEN_{\tau-1}$ and $INFL_{\tau-1}$ refer to cases where (1) includes additional control variables.

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Table 3: OS results

$T_0 = 15$ (1991-2005)	AE criterion								
Control var.:	none			$OPEN_{i,\tau-1}$			$INFL_{i,\tau-1}$		
	H_0	H_{01}	H_{02}	H_0	H_{01}	H_{02}	H_0	H_{01}	H_{02}
low	22.63	50.53	42.63	20.53	45.26	45.26	24.74	46.84	50.00
lower middle	27.50	51.88	49.38	24.38	47.50	49.38	23.13	51.25	46.88
upper middle	23.57	51.43	45.00	19.29	50.00	44.29	21.43	47.14	49.29
high	24.40	56.80	44.00	22.00	53.20	41.60	21.20	53.20*	40.80
$T_0 = 10$ (1996-2005)									
low	23.16	45.79	43.68	22.63	43.68	47.37	23.16	47.89	46.32
lower middle	23.75	53.75	43.13	21.25	56.25	42.50	24.38	53.75	43.13
upper middle	25.00	57.14	44.29	17.86	52.14	37.86	21.43	56.43	38.57
high	20.80	52.00	42.80	20.80	50.40	42.00	22.40	52.80	46.40
$T_0 = 15$ (1991-2005)	DA criterion								
low	1.05	7.37	6.84	0.53	10.00	3.68	0.00	8.95	5.26
lower middle	0.63	6.88	2.50	0.00	6.88	3.13	0.63	8.13	4.38
upper middle	0.00	6.43	5.00	0.00	8.57	2.86	0.00	5.71	5.00
high	0.00	4.40	4.40	0.00	7.60	2.40	0.00	7.20	3.20
$T_0 = 10$ (1996-2005)									
low	1.05	10.00	4.21	0.53	9.47	4.21	0.00	10.00	4.74
lower middle	0.00	8.25	3.63	0.00	6.25	3.75	0.00	10.00	3.13
upper middle	0.00	9.00	3.86	0.00	9.29	2.14	0.00	8.57	5.00
high	0.00	6.80	4.00	0.00	8.00	2.00	0.80	9.60	4.00

Note: Cell entries in columns H_{01} and H_{02} denote fractions of $\mathcal{T}_g$ cases where $\text{AE}^{(H01)} > \text{AE}^{(H1)}$ or $\text{AE}^{(H02)} > \text{AE}^{(H1)}$, respectively. Instances of H_0 obtain as the number of cases where evidence for both $\text{AE}^{(H01)} > \text{AE}^{(H1)}$ and $\text{AE}^{(H02)} > \text{AE}^{(H1)}$ is found. Conversely, in the lower panel, $\text{DA}^{(H01)} < \text{DA}^{(H1)}$ means rejection of H_{01}.

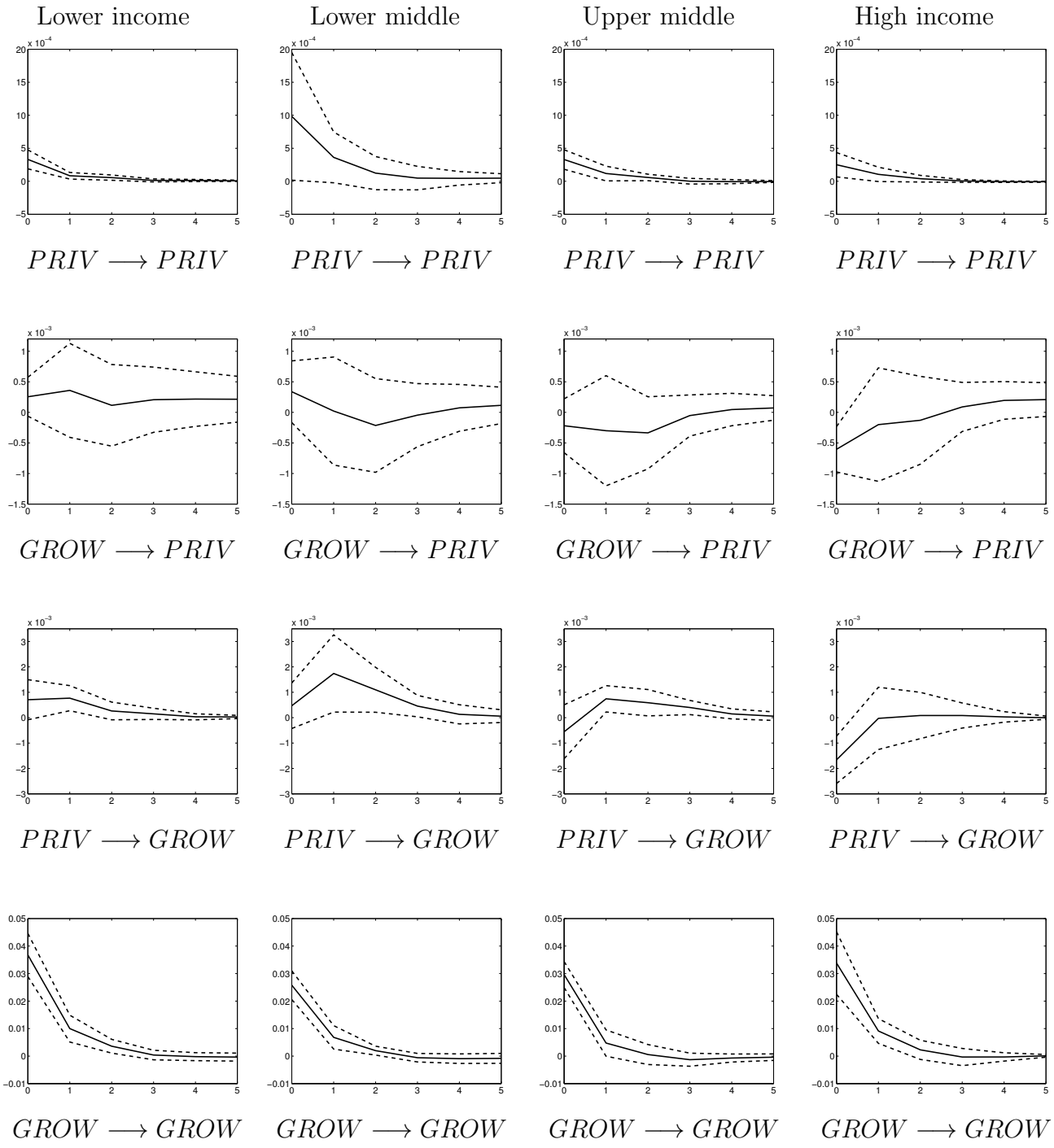

Figure 1: Generalised impulse response functions (IRFs). Income-group specific IRFs obtain as averages over $\mathcal{T}_g$ instances. Dashed lines indicate approximate ± 2 standard error confidence bands.