

This is "Monetary Policy Transmission Mechanisms", chapter 24 from the book <u>Finance</u>, <u>Banking</u>, and <u>Money (index.html)</u> (v. 1.1).

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Chapter 24

Monetary Policy Transmission Mechanisms

CHAPTER OBJECTIVES

By the end of this chapter, students should be able to:

- 1. Explain why structural models are generally superior to reduced-form models.
- 2. Describe the types of evidence that can strengthen researchers' conviction that a reduced-form model has the direction of causation right, say, from money (M) to output (Y).
- 3. Describe the evidence that money matters.
- 4. List and explain several important monetary policy transmission mechanisms.

24.1 Modeling Reality

LEARNING OBJECTIVE

1. Why are structural models generally superior to reduced-form models?

We've learned in the last few chapters that monetary policy is not the end-all and be-all of the economy or even of policymakers' attempts to manipulate it. But we knew that before. The question before us is, *Given what we know of IS-LM and AS-AD*, just how important is monetary policy? And how do we know? We've got theories galore—notions about how changes in sundry variables, like interest rates, create certain outcomes, like changes in prices and aggregate output. But how well do those theories describe reality? To answer those questions, we need empirical evidence, good hard numbers. We also need to know how scientists and social scientists evaluate such evidence.

Structural models explicitly link variables from initial cause all the way to final effect via every intermediate step along the causal chain. Reduced-form evidence makes assertions only about initial causes and ultimate effects, treating the links in between as an impenetrable black box. The quantity theory makes just such a reduced-form claim when it asserts that, as the money supply increases, so too does output. In other words, the quantity theory is not explicit about the **transmission mechanisms**¹ of monetary policy. On the other hand, the assertion that increasing the money supply decreases interest rates, which spurs investment, which leads to higher output, ceteris paribus, is a structural model. Such a model can be assessed at every link in the chain: MS up, *i* down, I up, Y up. If the relationship between MS and Y begins to break down, economists with a structural model can try to figure out specifically why. Those touting only a reduced-form model will be flummoxed. Structural models also strengthen our confidence that changes in MS cause changes in Y.

Because they leave so much out, reduced-form models may point only to variables that are correlated, that rise and fall in tandem over time. *Correlation, alas, is not causation; the link between variables that are only correlated can be easily broken*. All sorts of superstitions are based on mere correlation, as their practitioners eventually discover to their chagrin and loss, http://www.dallasobserver.com/2005-09-08/dining/tryst-of-fate/ like those who wear goofy-looking rally caps to win baseball games. http://en.wikipedia.org/wiki/Rally_cap Reverse causation is also rampant. People who see a high correlation between X and Y often think that X causes Y when in fact Y causes X. For example, there is a high correlation between fan

^{1.} How A leads to B in a causal chain; how A is transmitted, so to speak, to B.

attendance levels and home team victories. Some superfans http://en.wikipedia.org/wiki/Bill_Swerski's_Superfans take this to "prove" that high attendance causes the home team to win by acting as a sixth, tenth, or twelfth player, depending on the sport. Fans have swayed the outcome of a few games, usually by touching baseballs still in play, http://www.usatoday.com/sports/columnist/lopresti/2003-10-15-lopresti_x.htm but the causation mostly runs in the other direction—teams that win many games tend to attract more fans.

Omitted variables can also cloud the connections made by reduced-form models. "Caffeine drinkers have higher rates of coronary heart disease (CHD) than people who don't consume caffeine" is a reduced-form model that probably suffers from omitted variables in the form of selection biases. In other words, caffeine drinkers drink caffeine because they don't get enough sleep; have hectic, stressful lives; and so forth. It may be that those other factors give them heart attacks, not the caffeine per se. Or the caffeine interacts with those other variables in complex ways that are difficult to unravel without growing human beings in test tubes (even more alarming!).

24.1 Modeling Reality 462

Stop and Think Box

A recent reduced-form study shows a high degree of correlation between smoking marijuana and bad life outcomes: long stints of unemployment, criminal arrests, higher chance of disability, lower lifetime income, and early death. Does that study effectively condemn pot smoking?

Not nearly as much as it would if it presented a structural model that carefully laid out and tested the precise chain by which marijuana smoking causes those bad outcomes. Omitted variables and even reverse causation can be at play in the reduced-form version. For example, some people smoke pot because they have cancer. Some cancer treatments require nasty doses of chemotherapy, the effect of which is to cause pain and reduce appetite. Taking a toke reduces the pain and restores appetite. Needless to say, such people have lower life expectancies than people without cancer. Therefore, they have lower lifetime income and a higher chance of disability and unemployment. Because not all states have medical marijuana exceptions, they are also more liable to criminal arrest. Similarly, unemployed people might be more likely to take a little Mary Jane after lunch or perhaps down a couple of cannabis brownies for dessert, again reversing the direction of causation. A possible omitted variable is selection bias: people who smoke pot might be less educated than those who abstain from the weed, and it is the dearth of education that leads to high unemployment, more arrests, and so forth. Unfortunately, bad science like this study pervades public discourse. Of course, this does not mean that you should go get yourself a blunt. Study instead. Correlation studies show that studying . .

24.1 Modeling Reality 463

KEY TAKEAWAYS

- Structural models trace the entire causal chain, step by step, allowing researchers to be pretty confident about the direction of causation and to trace any breakdowns in the model to specific relationships.
- Reduced-form models link initial variables to supposed outcomes via an impenetrable black box.
- The problem is that correlation does not always indicate causation. X may increase and decrease with Y, although X does not cause Y because Y may cause X (reverse causation), or Z (an omitted variable) may cause X and Y.
- Reduced-form models can and have led to all sorts of goofy conclusions, like doctors kill people (they seem to be ubiquitous during plagues, accidents, and the like) and police officers cause crime (the number on the streets goes up during crime waves, and they are always at crime scenes—very suspicious). In case you can't tell, I'm being sarcastic.
- On the other hand, reduced-form models are inexpensive compared to structural ones.

24.1 Modeling Reality 464

24.2 How Important Is Monetary Policy?

LEARNING OBJECTIVES

- 1. What types of evidence can strengthen researchers' conviction that a reduced-form model has the direction of causation right, say, from M to Y? How?
- 2. What evidence is there that money matters?

Early Keynesians believed that monetary policy did not matter at all because they could not find any evidence that interest rates affected planned business investment. Milton Friedman and Anna Schwartz, another monetarist, countered with a huge tome called A Monetary History of the United States, 1867–1960 which purported to show that the Keynesians had it all wrong, especially their kooky claim that monetary policy during the Great Depression had been easy (low real interest rates and MS growth). Nominal rates on risky securities had in fact soared in 1930–1933, the depths of the depression. Because the price level was falling, real interest rates, via the Fisher Equation, were much higher than nominal rates. If you borrowed \$100, you'd have to repay only \$102 in a year, but those 102 smackers could buy a heck of a lot more goods and services a year hence. So real rates were more on the order of 8 to 10 percent, which is pretty darn high. The link between interest rates and investment, the monetarists showed, was between investment and real interest rates, not nominal interest rates.

As noted above, the early monetarists relied on MV = PY, a reduced-form model. To strengthen their conviction that causation indeed ran from M to Y instead of Y to M or some unknown variables A...Z to M and Y, the monetarists relied on three types of empirical evidence: timing, statistical, and historical. Timing evidence tries to show that increases in M happen before increases in Y, and not vice versa, relying on the commonplace assumption that causes occur before their effects. Friedman and Schwartz showed that money growth slowed before recessions, but the timing was highly variable. Sometimes slowing money growth occurred sixteen months before output turned south; other times, only a few months passed. That is great stuff, but it is hardly foolproof because, as Steve Miller points out, time keeps on slipping, slipping, slipping, into the future.http://www.lyricsfreak.com/s/steve+miller/fly+like+an+eagle_20130994.html Maybe a decline in output caused the decline in the money supply. Changes in M and Y, in other words, could be causing each other in a sort of virtuous or pernicious cycle or chicken-egg problem. Or again maybe there is a mysterious variable Z running the whole show behind the scenes.

Statistical evidence is subject to the same criticisms plus the old adage that there are three types of untruths (besides Stephen Colbert's

truthiness, http://en.wikipedia.org/wiki/Truthiness of course): lies, damn lies, and statistics. By changing starting and ending dates, conflating the difference between statistical significance and economic

significance, http://www.deirdremccloskey.com/articles/stats/preface_ziliak.php manipulating the dates of structural breaks, and introducing who knows how many other subtle little fibs, researchers can make mountains out of molehills, and vice versa. It's kinda funny that when monetarists used statistical tests, the quantity theory won and money mattered, but when the early Keynesians conducted the tests, the quantity theory looked, if not insane, at least inane.

But Friedman and Schwartz had an empirical ace up their sleeves: historical evidence from periods in which declines in the money supply appear to be exogenous, by which economists mean "caused by something outside the model," thus eliminating doubts about omitted variables and reverse causation. White-labcoat scientists (you know, physicists, chemists, and so forth—"real" scientists) know that variables change exogenously because they are the ones making the changes. They can do this systematically in dozens, hundreds, even thousands of test tubes, Petri dishes, atomic acceleration experiments, and what not, carefully controlling for each variable (making sure that everything is ceteris paribus), then measuring and comparing the results. As social scientists, economists cannot run such experiments. They can and do turn to history, however, for so-called natural experiments. That's what the monetarists did, and what they found was that exogenous declines in MS led to recessions (lower Y*) every time. Economic and financial history wins! (Disclaimer: One of the authors of this textbook [Wright] is a financial historian.) While they did not abandon the view that C, G, I, NX, and T also affect output, Keynesians now accept money's role in helping to determine Y. (A new group, the realbusiness-cycle theorists associated with the Minneapolis Fed, has recently challenged the notion that money matters, but those folks haven't made it into the land of undergraduate textbooks quite yet.)

KEY TAKEAWAYS

- Timing, statistical, and historical evidence strengthen researchers' belief in causation.
- Timing evidence attempts to show that changes in M occur before changes in Y.
- Statistical evidence attempts to show that one model's predictions are closer to reality than another's.
- The problem with stats, though, is that those running the tests appear to rig them (consciously or not), so the stats often tell us more about the researcher than they do about reality.
- Historical evidence, particularly so-called natural experiments in which variables change exogenously and hence are analogous to controlled scientific experiments, provide the best sort of evidence on the direction of causation.
- The monetarists showed that there is a strong correlation between changes in the MS and changes in Y and also proffered timing, statistical, and historical evidence of a causal link.
- Historical evidence is the most convincing because it shows that the MS sometimes changed exogenously, that is, for reasons clearly unrelated to Y or other plausible causal variables, and that when it did, Y changed with the expected sign (+ if MS increased, if it decreased).

24.3 Transmission Mechanisms

LEARNING OBJECTIVE

1. What are monetary policy transmission mechanisms and why are they important?

Most economists accept the proposition that money matters and have been searching for structural models that delineate the specific transmission mechanisms between MS and Y. The most basic model says the following:

Expansionary monetary policy (EMP), real interest rates down, investment up, aggregate output up

The importance of interest rates for consumer expenditures (especially on durables like autos, refrigerators, and homes) and net exports has also been recognized, leading to the following:

EMP,
$$i_r \downarrow$$
, $I + C + NX \uparrow$, $Y \uparrow$

Tobin's q, the market value of companies divided by the replacement cost of physical capital, is clearly analogous to i and related to I. When q is high, firms sell their highly valued stock to raise cash and buy new physical plant and build inventories. When q is low, by contrast, firms don't get much for their stock compared to the cost of physical capital, so they don't sell stock to fund increases in I. By increasing stock prices, the MS may be positively related to q. Thus, another monetary policy transmission mechanism may be the following:

EMP, Ps
$$\uparrow$$
, q \uparrow , I \uparrow , Y \uparrow

The wealth effect is a transmission mechanism whereby expansionary monetary policy leads to increases in the prices of stocks, homes, collectibles, and other assets, in other words, an increase in individual wealth. That increase, in turn, induces people to consume more:

EMP,
$$P_a \uparrow$$
, wealth \uparrow , $C \uparrow$, $Y \uparrow$

Chapter 24 Monetary Policy Transmission Mechanisms

The credit view posits several straightforward transmission mechanisms, including bank loans, asymmetric information, and balance sheets:

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EMP, bank deposits \uparrow, bank loans \uparrow, I \uparrow, Y\uparrow
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EMP, net worth \downarrow , asymmetric information \downarrow , lending \uparrow , I + C \uparrow , Y \uparrow

EMP, $i \downarrow$, cash flow \uparrow , asymmetric information \downarrow , lending \uparrow , I + C \uparrow , Y \uparrow

EMP, unanticipated $P^* \uparrow$, real net worth \uparrow , asymmetric information \downarrow , lending \uparrow , I \uparrow , Y \uparrow

Asymmetric information (that horrible three-headed hound from Hades) is a powerful and important theory, so scholars' confidence in these transmission mechanisms is high.

Stop and Think Box

The Fed thought that it would quickly squelch the recession that began in March 2001, yet the downturn lasted until November of that year. The terrorist attacks that September worsened matters, but the Fed had hoped to reverse the drop in Y* well before then. Why was the Fed's forecast overly optimistic? (*Hint*: Corporate accounting scandals at Enron, Arthur Andersen, and other firms were part of the mix.)

The Fed might not have counted on some major monetary policy transmission mechanisms, including reductions in asymmetric information, being muted by the accounting scandals. In other words,

EMP, net worth \uparrow , asymmetric information \downarrow , lending \uparrow , I + C \uparrow , Y \uparrow

EMP, $i \downarrow$, cash flow \uparrow , asymmetric information \downarrow , lending \uparrow , I + C \uparrow , Y \uparrow

EMP, unanticipated P \uparrow , real net worth \uparrow , asymmetric information \downarrow , lending \uparrow , I \uparrow , Y \uparrow

became something more akin to the following:

EMP, net worth \uparrow , asymmetric information — (flat or no change), lending —, I + C —, Y —

EMP, $i \uparrow$, cash flow \uparrow , asymmetric information —, lending —, I + C —, Y —

EMP, unanticipated P \uparrow , real net worth \uparrow , asymmetric information—, lending —, I —, Y — because asymmetric information remained high due to the fact that economic agents felt as though they could no longer count on the truthfulness of corporate financial statements.

The takeaway of all this for monetary policymakers, and those interested in their policies (including you, as you know from <u>Chapter 1 "Money, Banking, and Your World"</u>), is that monetary policy needs to take more into account than just short-term interest rates. Policymakers need to worry about real interest rates, including

long-term rates; unexpected changes in the price level; the interest rates on risky bonds; the prices of other assets, including corporate equities, homes, and the like; the quantity of bank loans; and the bite of adverse selection, moral hazard, and the principal-agent problem.

Stop and Think Box

Japan's economy was going gangbusters until about 1990 or so, when it entered a fifteen-year economic funk. To try to get the Japanese economy moving again, the Bank of Japan lowered short-term interest rates all the way to zero for many years on end, to no avail. Why didn't the Japanese economy revive due to the monetary stimulus? What should the Japanese have done instead?

As it turns out, i_r stayed quite high because the Japanese expected, and received, price deflation. Through the Fisher Equation, we know that $i_r = i - \pi^e$, or real interest rates equal nominal interest rates minus inflation expectations. If π^e is negative, which it is when prices are expected to fall, ir will be > i. So i can be 0 but ir can be 1, 2, 3 10 percent per year if prices are expected to decline by that much. So instead of EMP, $i_r -$, I + C + NX -, Y- the Japanese experienced $i_r -$, I + C + NX -, Y-. Not good. They should have pumped up the MS much faster, driving π^e from negative whatever to zero or even positive, and thus making real interest rates low or negative, and hence a stimulant. The Japanese made other mistakes as well, allowing land and equities prices to plummet, thereby nixing the Tobin's q and wealth effect transmission mechanisms. They also kept some big shaky banks from failing, which kept levels of asymmetric information high and bank loan levels low, squelching the credit channels.

KEY TAKEAWAYS

- Monetary policy transmission mechanisms are essentially structural models that predict the precise chains of causation between expansionary monetary policy (EMP) or tight monetary policy (TMP) and Y.
- They are important because they provide central bankers and other monetary policymakers with a detailed view of how changes in the MS affect Y, allowing them to see why some policies don't work as much or as quickly as anticipated.
- That, in turn, allows them to become better policymakers, to the extent that is possible in a world of rational expectations. (See <u>Chapter 26</u> "Rational Expectations Redux: Monetary Policy Implications".)
- Transmission mechanisms include:
- EMP, $i_r \downarrow$, I + C + NX \(\gamma\), Y\(\gamma\), EMP, $q \uparrow$, I\(\gamma\), Y\(\gamma\)
- EMP, $P_a \uparrow$, wealth \uparrow , $C \uparrow$, $Y \uparrow$
- EMP, bank deposits \(\gamma\), bank loans \(\gamma\), I \(\gamma\), Y \(\gamma\)
- EMP, net worth \uparrow , asymmetric information \downarrow , lending \uparrow , I + C \uparrow , Y \uparrow
- EMP, $i \downarrow$, cash flow \uparrow , asymmetric information \downarrow , lending \uparrow , I + C \uparrow , Y \uparrow
- EMP, unanticipated P* \(\gamma\), real net worth \(\gamma\), asymmetric information \(\psi\), lending \(\gamma\), I \(\gamma\), Y\(\gamma\)

24.4 Suggested Reading

Angeloni, Ignazio, Anil Kashyap, and Benoit Mojon. *Monetary Policy Transmission in the Euro Area*. New York: Cambridge University Press, 2004.

Friedman, Milton, and Anna Schwartz. *A Monetary History of the United States*, 1867–1960. Princeton, NJ: Princeton University Press, 1971.

Mahadeva, Lavan, and Peter Sinclair. *Monetary Transmission in Diverse Economies*. New York: Cambridge University Press, 2002.